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Australasian Medical Congress

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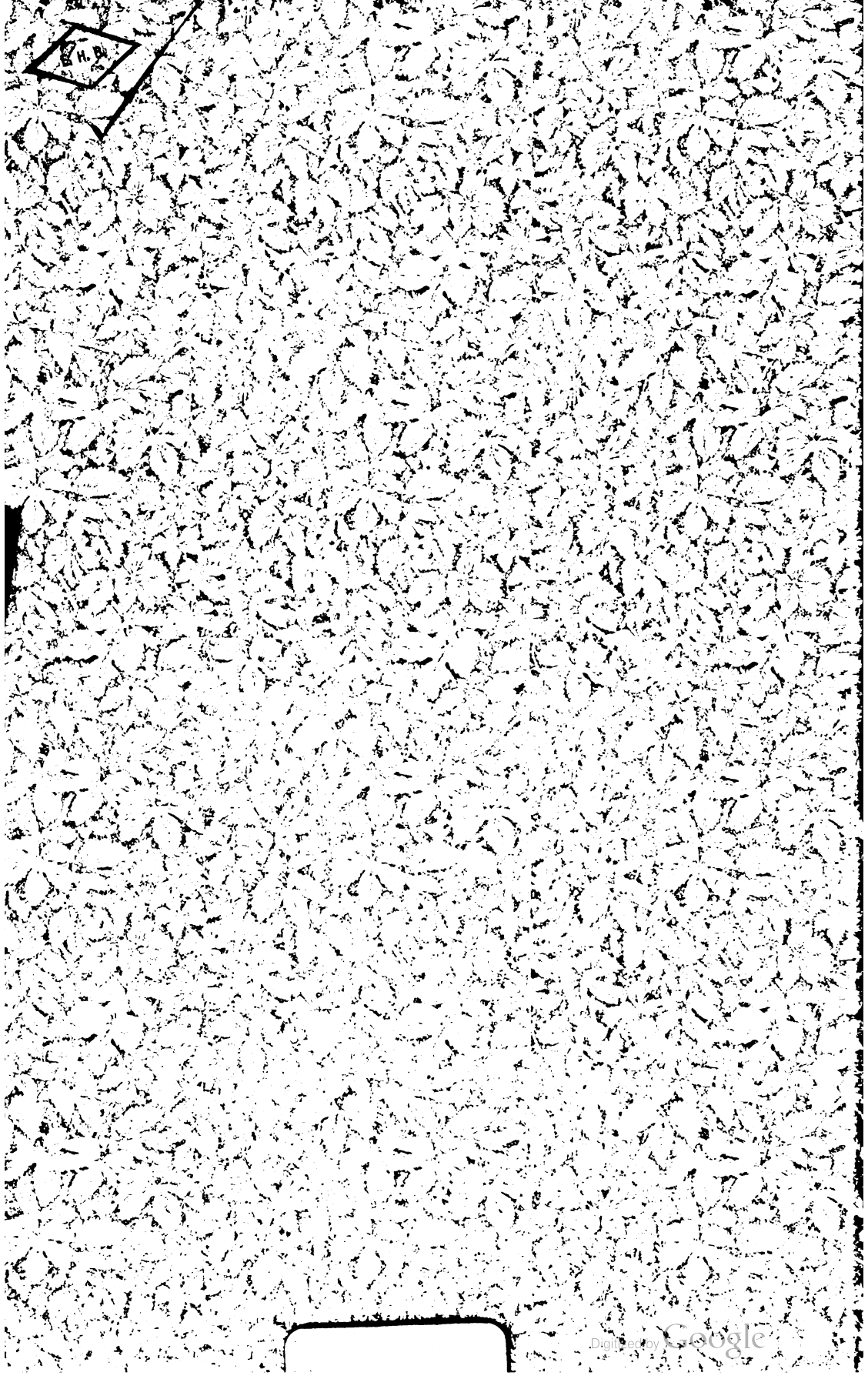
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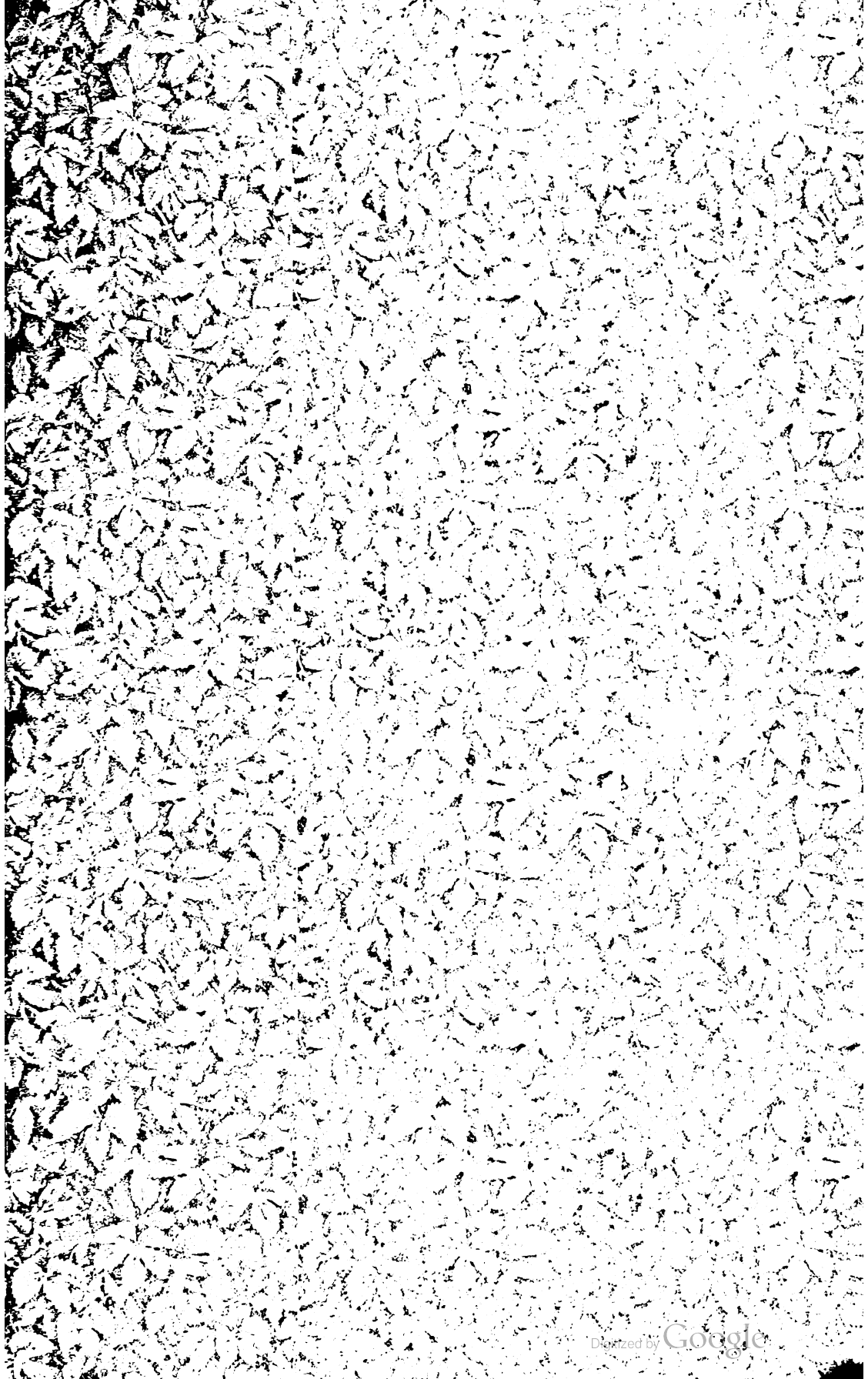
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AUSTRALASIAN

MEDICAL CONGRESS

(Formerly the Intercolonial Medical Congress of Australasia).

TRANSACTIONS

OF

THE SEVENTH SESSION,

HELD AT

ADELAIDE, SEPTEMBER, 1905.

PUBLISHED UNDER THE DIRECTION OF THE EDITORIAL COMMITTEE.

South Australia:
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PREFACE.

In presenting to members of the Australasian Medical Congress the Transactions of the Adelaide Session of 1905, the Editorial Committee regret the long delay that has attended its publication.

For this delay unusual pressure of work in the Government Printing Office and the prior claims of official work are chiefly responsible; but it must be confessed that the Committee have also found themselves hindered by the neglect of some authors to forward their papers at a sufficiently early date.

The Transactions embodied in this volume form a continuance of the series issued in previous years as the "Transactions of the Intercolonial Medical Congress of Australasia," and it is hoped that members will find it, like its predecessors, a faithful record of their principal proceedings.

The Committee desire to express their appreciation of the liberality of the Government of South Australia in defraying the cost of publication of the volume, and of the care taken in its production by the official printer (Mr. Bristow) and his staff.

E. C. STIRLING, *President.*

Adelaide, November, 1906.

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J. R. Baker, B.A., LL.B. Cantab., Chairman of Local Board of Health, Adelaide.
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C. A. Bayer, Esq., Hydraulic Engineer, South Australia.
The Hon. G. Brookman, M.L.C., South Australia.
Theodore Bruce, Esq., Mayor of Adelaide.
Richard Caton, M.D., &c., Liverpool, England.
T. G. Ellery, Esq., Town Clerk, Adelaide.
A. G. Fryett, Esq., Melbourne, Victoria.
C. R. Hodge, Esq., Registrar, University of Adelaide.
T. Roberts, Esq., Chief Mechanical Engineer to the Railway Department of South Australia.

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Australasian Medical Congress.

SEVENTH SESSION.

PROGRAMME OF CONGRESS WORK.

Monday, September 4th.

10 a.m. Registration of Members at University.

11.30 a.m. General Business Meeting of Congress at University.

8.30 p.m. Inaugural Meeting and President's Address at the Elder Hall, University.

Tuesday, September 5th.

10 a.m. Address in Medicine by Dr. Daniel Colquhoun, M.D. (Lond.), M.R.C.P., M.R.C.S., of Dunedin, President of the Section, in the Prince of Wales' Theatre, University.

11 a.m. to 1 p.m. } Primary Meetings of all Sections.

12 noon. Section of Pathology: Address on Cancer, by Professor Welsh, M.A., M.D., B.Sc. (Edin.), of Sydney, President of the Section.

Wednesday, September 6th.

10 a.m. Address in Surgery by Mr. Fred. Bird, M.B., M.S. (Melb.), M.R.C.S, of Melbourne, President of the Section.

11 a.m. to 1 p.m. } Sectional Work.

12 noon. Section of State Medicine: Address by Mr. Syme, M.B., M.S. (Melb.), F.R.C.S., President of the Section.

Thursday, September 7th.

10 a.m. to 1 p.m. } Sectional Work.

Friday, September 8th

10 a.m. to 1 p.m. } Sectional Work.

8 p.m. Popular Health Lecture by Dr. W. G. Armstrong, B.A., M.B., Ch.M. (Syd.), D.P.H. (Cantab.), President of the Section of Public Health, in the Town Hall, under the auspices of the Right Worshipful the Mayor of Adelaide.

Saturday, September 9th.

9.30 a.m. Sectional Meetings.

10 a.m. Final General Meeting of Congress at University.

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Section I.

MEDICINE.

President .. .. Daniel Colquhoun, M.D., Lond., M.R.C.P., M.R.C.S. ; Dunedin.

Vice-Presidents .. G. E. Rennie, M.D., Lond., M.R.C.P., M.R.C.S. ; Sydney.
David Grant, M.A., M.D., Edin. ; Melbourne.
A. J. Turner, M.D., Lond., M.R.C.S. ; Brisbane.
H. Astles, M.D., St. And., F.R.C.P. Edin. ; Perth.
R. R. Whishaw, M.B., B.C., Cantab. ; Hobart.
W. Fell, M.D., Oxon., M.R.C.S., L.R.C.P. ; Wellington, N.Z.

Secretary .. .. H. Swift, M.D., Cantab., M.R.C.S. ; Adelaide.

LIST OF PAPERS.

The Address in Medicine—Dr. Colquhoun.

1. Diet in Relation to Kidney Disease.
   Dr. Rennie.
2. Epilepsy : Is it incurable ?
   Dr. Rennie.
3. The Mineral Waters of New Zealand.
   Dr. Wohlmann.
4. Is there such a Disease as Croup ?
   Dr. H. E. Astles.
6. Preliminary Communication on the No-Food Treatment of Typhoid Fever.
   Dr. Abramowski.
7. Pulmonary Tuberculosis.
   Dr. McIntyre Sinclair.
8. Sanatorium Treatment of Phthisis.
   Dr. Stoney.
9. Relation of Bovine to Human Tuberculosis.
   Dr. W. C. Wilkinson.
10. The Results of Sanatorium Treatment of Pulmonary Tuberculosis.
    Dr. W. C. Wilkinson.
11. Sanatorium Treatment of Phthisis.
    Dr. Gault.
12. Anchylostomiasis.
    Dr. MacDonald.
13. The Nasal Treatment of Asthma.
    Dr. W. V. Robertson
14. Anaemias of Children and Infancy.
    Dr. C. Baxter Tyrie.
15. The Blood Count of Appendicitis.
    Dr. Wilton Love.
16. The Medical Treatment of Appendicitis.
    Dr. F. L. Benham.
17. A Fatal Case of Acute Pancreatitis.
    Dr. F. L. Benham.
18. Radium and Electro Therapeutics in the Treatment of Skin Diseases.
    Dr. H. Lawrence.

    Exhibits—"An X Ray Bath."
    Dr. H. Lawrence.

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19. Present Position of the Rontgen Rays in Medicine and Surgery.
Dr. L. H. Harris.

20. A Case of Spleno-Medullary Leucocythaemia treated with X Rays.
Dr. F. Clendinnen.

21. A Case of Lymphadenoma (internal) treated with X Rays.
Dr. F. Clendinnen.

22. A Case of Bazin's Malady Treated with Ultra-Violet Light.
Dr. F. Clindennen.

23. The Principles of Chemistry and the Study of Medicine.
Dr. Reissmann.

24. On Leukaemia and allied conditions in Children.
Dr. J. Macdonald Gill.

25. Two Cases of Lead-Poisoning.
Dr. J. Macdonald Gill.

26. The External Application of Guaiacol.
Dr. T. C. Moore.

27. The Complications of Pneumonia other than Pulmonary.
Dr. Scott Skirving.

28. Use and Abuse of Mental Therapeutics.
Dr. Springthorpe.

29. Transposition of the Organs verified by *Post-mortem* Examination.
Dr. Jarvie Hood.

CLINICAL EXHIBITS: 9 A.M., WEDNESDAY, SEPTEMBER 6TH.

Clinical Session—

Exhibits: Cases of Skin Disease, &c.
Dr. H. Swift.

Section II.

SURGERY.

President .. .. F. D. Bird, M.B., M.S., Melb., M.R.C.S.; Melbourne.

Vice-Presidents .. H. V. C. Hinder, M.B., Ch.M., Syd.; Sydney.
R. Hamilton Russell, F.R.C.S.; Melbourne.
E. S. Jackson, M.B., Ch.B., Melb.; Brisbane.
F. Tratman, M.D., Lond., M.R.C.S., L.R.C.P., D.P.H., Lond.; Perth.
R. G. Scott, M.B., Ch.M., Edin.; Hobart.
W. E. Collins, M.B., Lond., M.R.C.S.; Wellington, N.Z.

Secretaries .. .. W. Anstey Giles, M.B., Ch.M., Edin.; Adelaide.
C. E. Todd, M.D., Brux., M.R.C.S.: Adelaide.

LIST OF PAPERS.

1. Address in Surgery.
Mr. F. Bird.

2. Prostatectomy.
Dr. A. MacCormick.

3. Prostatectomy.
Dr. H. O'Hara.

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4. Enlarged Prostate.
   Dr. H. L. Maitland.
5. Prostatectomy.
   Dr. Poulton.
6. Urinary Surgery.
   Dr. C. Hinder.
7. Papilloma of Kidney.
   Dr. A. A. London.
8. Cerebral Hydatids.
   Dr. McCormick.
9. Congenital Hypertrophic Stenosis of Pylorus.
   Dr. A. McKay.
10. Intestinal Anastomosis.
    Dr. MacCormick.
11. Stereoscope in Surgery.
    Dr. C. H. Souter.
12. Radical Cure of Hernia.
    Dr. H. Russell.
13. Hernia.
    Mr. F. Bird.
14. Appendicitis.
    Dr. C. Hinder.
15. Appendicitis.
    Dr. Long.
16. Appendicitis.
    Dr. McCallum.
17. Appendicitis.
    Dr. Kelvington,
18. Appendicitis.
    Dr. Scott.
19. Appendicitis,
    Dr. Newman.
20. Appendicitis.
    Dr. Steer Bowker.
21. Observations on Appendicitis.
    Dr. Fiaschi.
22. Treatment of Movable Kidney.
    Dr. Collins.
23. Some Subject in Paediatrics.
    Dr. Turner.
24. Nerve Crossing and Suturing.
    Dr. Kelvington.
25. Some Strange Coincidences in Surgical Cases.
    Dr. Bickle.
26. Notes on Modern Cerebral Surgery.
    Dr. Marten.
27. Brophy's Method of Treating Cleft Palate.
    Dr. M. Morton.
28. Transplantation of Ureters into Rectum.
    Dr. Newland.

CLINICAL EXHIBITS: 9 A.M., THURSDAY, SEPTEMBER 7TH.

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Section III.

GYNÆCOLOGY AND OBSTETRICS.

President .. .. W. S. Byrne, M.D., Ch.M., Dub., M.R.C.P.; Brisbane.

Vice-Presidents .. E. T. Thring, F.R.C.S., L.R.C.P.; Sydney.
G. Rothwell Adam, M.D., Ch.M., Edin., M.D., Melb.; Melbourne
F. G. Connolly, M.R.C.S., M.R.C.P.; Brisbane.
H. Horrocks, M.D., Lond., M.R.C.S., L.R.C.P.; Perth.
C. J. Pike, M.B., Ch.B., Lond., M.R.C.S.; Launceston.
B. Moorehouse, M.B., Ch.M., Edin., M.R.C.S.; Christchurch, N.Z.

Secretary .. .. J. A. G. Hamilton, B.A., M.B., Dub., L.R.C.S., Edin.; Adelaide.

LIST OF PAPERS.

The Opening Address by the President.

1. Some Causes of Pelvic Suppuration.
   Dr. Rothwell Adam.

2. Treatment of Pelvic Suppuration.
   Dr. R. Worrall.

3. Some Practical Points in Relation to frequently performed Operations in Gynæcology.
   Dr. F. W. Thring.

4. The Evolution of the Abdominal Operation and Incision.
   Dr. Foreman.

5. Chorion Epithelioma.
   Dr. Batchelor.

6. Differential Diagnosis between Appendicitis and Pelvic Disease in Young Women.
   Dr. D. Hooper.

7. Puerperal Sepsis.
   Dr. Cowen.

8. The Toxæmias of Pregnancy.
   Dr. Batchelor.

9. Paper.
   Dr. Felix Meyer.

10. Thrombosis of Jugular Vein after Parturition.
    Dr. F. Clendinnen.

11. Retroflexion of Uterus.
    Dr. T. G. Wilson.

12. Acute Dilatation of Stomach following Abdominal Section.
    Dr. J. A. G. Hamilton.

13. Modern Treatment of Puerperal Sepsis following Labor and Abortion.
    Dr. Connolly.

14. A Rare Form of Pelvic Tumor.
    Dr. Nyulasy.

15. Ovarian Dermoid.
    Dr. Setzke.

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Section IV.

EYE, EAR, AND THROAT.

Presidents .. .. A. J. Brady, L.R.C.S.I., L.K.Q.C.P.I. ; Sydney.
W. Odillo Maher, M.D., Ch.M., Dub., M.D., Syd., M.R.C.S. ; Sydney

Vice-Presidents .. G. T. Hankins, M.R.C.S. ; Sydney.
P. S. Webster, M.D., Durh., M.R.C.S., L.R.C.P. ; Melbourne.
J. Lockhart Gibson, M.D., Edin., M.R.C.S. ; Brisbane.
H. T. Kelsall, M.D., B.S., Lond., M.R.C.S., L.R.C.P. ; Perth.
W. W. Giblin, M.R.C.S., L.R.C.P. ; Hobart.
H. Lindo Ferguson, M.A., M.D., Dub., F.R.C.S.I. ; Dunedin, N.Z.

Secretaries .. .. Charles W. Hamilton, B.A., M.D., Dub. ; Adelaide.
M. J. Symons, M.D., Ch.M., Edin. ; Adelaide.

(A) EYE DIVISION.—PAPERS, &c.

President .. .. Dr. W. Odillo Maher.

Secretary .. .. Dr. M. J. Symons.

Tuesday, 11 a.m.—Opening Address by the President.

Discussion Paper on Trachoma by Dr. Antill Pockley.

1. Keratitis during and after Dengue.
2. Pseudo-Myopia and its Treatment.
   Dr. Lockhart Gibson.
3. Cases Illustrating the Influence of Increased Intracranial Pressure as a Cause of Optic Neuritis.
4. Exophthalmos in relation to Disease in the adjacent Bony Cavities.
   Dr. Wallace Mackenzie.

Wednesday, 11 a.m.—Discussion Paper on Plumbic Neuritis—Dr. Lockhart Gibson.

1. Treatment of Trachoma with Cuprol.
2. Cases of Extraction of Foreign Bodies by Magnet.
3. The Difficulties in arriving at a Decision as to when an Injured Eye must be removed.
   Dr. G. M. Scott.
4. Accommodation in Aphakic Eyes.
   Dr. R. H. Jones.
5. On the suturing of gaping wounds of the Sclerotic, and of those made by the Surgeon in Cataract Operations.
   Dr. J. P. Ryan.
6. Paper undescribed.
   Dr. Andrew Orr.
7. Paper undescribed.
   Dr. Andrew Orr.
8. Lantern Slide Illustrations.
   Dr. Andrew Orr.
9. Melanotic Sarcoma of the Conjunctiva.
   Dr. Pockley.
10. Myasthenia Gravis in special relation to Eye and Throat Conditions.
    Dr. G. H. Hogg.
11. Exhibit.—A Cast of Paraphimosis Conjunctivæ.
    Dr. L. Herschell Harris.

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(B) NOSE, EAR, AND THROAT DIVISION.—PAPERS, &c.

President .. .. Dr. A. J. Brady.

Secretary .. .. Dr. C. W. Hamilton.

The following subjects have been selected for special discussion:—

A. The Indications for the Radical Mastoid Operation, the Methods of its performance, and the value or otherwise of Epithelial Grafting.

B. The Diagnosis and Treatment of Suppuration of the Accessory Sinuses of the Nose.

Discussion *A* will be introduced by Dr. Webster (Melbourne), Dr. Lockhart Gibson (Brisbane), and Dr. Arthur (Sydney).

Discussion *B* will be introduced by Dr. Kirkland (Sydney), and Dr. Hogg (Launceston). The following Papers will be read:—

1. Nasal and Nasopharyngeal Factors in Chronic Catarrhal Disease of the Middle Ear.
   Dr. Ewing.
2. The Therapeutics of the Commoner Affections of the Nose.
   Dr. Kirkland.
3. Certain Considerations regarding Operations of the Mastoid.
   Dr. Arthur.

Section V.

PATHOLOGY, ANATOMY, PHYSIOLOGY, & THERAPEUTICS.

President .. .. Professor D. A. Welsh, M.A., M.D., B.Sc., Edin.; Sydney.

Vice-Presidents .. Professor J. T. Wilson, M.B., Ch.M., Edin.; Sydney.
C. H. Mollison, M.B., Ch.B., Melb., M.R.C.S.; Melbourne.
J. A. Wheeler, M.B., Lond.; Brisbane.
W. Trethowan, M.B., Ch.M., Aberd.; Perth.
A. H. Clarke, M.R.C., L.R.C.P.; Hobart.
W. S. Roberts, M.R.C.S.; Dunedin, N.Z.

Secretary .. .. W. R. Cavenagh-Mainwaring, M.B., Ad., F.R.C.S.; Adelaide.

LIST OF PAPERS.

1. Address by the President.
   Professor Welsh.
2. Paper on Deciduoma Malignum.
   Professor Welsh.
3. Leucocythaemia.
   Dr. Reissmann.
4. Human Parasites.
   Dr. Angas Johnson.
5. Note on a New Human Parasite.
   Dr. MacCormick and Professor Welsh.
6. Note on a Larval Tapeworm from the Human Subject.
   Dr. MacCormick and Dr. J. P. Hill.
7. Note on the frequency of Streptothrix Infections in Man.
   Dr. MacCormick, Dr. Barling, and Professor Welsh.
8. Exhibits with Lantern.
   Dr. J. Thomson.

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Section VI.

PUBLIC HEALTH, &c.

President .. .. W. G. Armstrong, B.A., M.B., Ch.M., Syd., D.P.H., Cantab.; Sydney.

Vice-Presidents .. Frank Tidswell, M.D., Syd., D.P.H., Cantab.; Sydney.
W. Beattie Smith, F.R.C.S. *et* L.R.C.P., Edin.; Melbourne.
B. B. Ham, M.B., Brux., M.R.C.S., D.P.H., Cantab.; Brisbane.
Ernest Black, L.R.C.P. *et* L.R.C.S., Edin.; Perth.
George F. Read, L.R.C.P. *et* L.R.C.S., Edin.; New Norfolk, Tasmania.
James Mason, M.D., Brux., L.R.C.P. *et* L.R.C.S., Edin., D.P.H., Cantab.; Wellington, N.Z.

Secretary .. .. T. Borthwick, M.D., Ch.M., Edin.; Adelaide.

LIST OF PAPERS.

1. Presidential Address.—Some Lessons from the Statistics of Infantile Mortality in Sydney.
2. Sewage Treatment—A Review, by Dr. E. S. Stokes, Sydney.
3. Typhoid Fever in New South Wales, 1898-1904, by Dr. R. J. Millard, Sydney.
4. Summer Diarrhœa in Infants, from a public health point of view, by Dr. W. F. Litchfield, Sydney.
5. A Short Record of Twenty Years' Sanitary Progress in Melbourne, by Dr. J. Jamieson, Melbourne.
6. Sanitary Administration in Adelaide, by Mr. T. Geo. Ellery, Secretary Local Board of Health, Adelaide.
7. A Review of the Public Health Legislation of Australasia, by J. R. Baker, LL.B. (Cantab.), Chairman Health Committee, Adelaide.
8. School Hygiene, by Dr. J. S. C. Elkington, Hobart.
9. Attitude of the State towards Consumption, by Dr. J. M. Mason, Wellington.
10. Notification of Phthisis, by Dr. T. Borthwick, Adelaide.
11. Public Lecture.—Some Aspects of Municipal Sanitary Administration, by Dr. W. G. Armstrong, Sydney.
12. Exhibits, by Dr. F. Tidswell, Sydney:
    1. Collection of formalinised cultures of typical bacteria, yeasts, and moulds.
    2. Collection of photomicrographs of various pathogenic bacteria.
    3. A series of enlargements from photomicrographs illustrating the histopathology of Leprosy.
    4. Photographs of Board of Health Laboratories, Sydney.
13. Photomicrographs of Bacteria, &c. Dr. Andrew Orr, Brisbane.

RESOLUTIONS SUGGESTED.

1. "That in order to secure efficiency in the Sanitary Administration of Australian Cities, is it necessary to have Public Abattoirs, Refuse Destructors, and a Steam Disinfector, as part of their equipment."

2. "That the combination of City and Suburban Local Boards is essential to the effective sanitary administration of Metropolitan Areas, and should be made compulsory in future legislation."

3. "That the compulsory notification of Phthisis is advisable in the interests of Public Health."

4. "That it is desirable to exclude emigrants suffering from Phthisis who are likely to become a burden on the State in the near future."

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Section VII.

STATE MEDICINE AND MEDICAL ETHICS.

President .. .. G. A. Syme, M.B., M.S., Melb., F.R.C.S.; Melbourne.

Vice-Presidents .. Sir P. Sydney Jones, M.D., Lond., F.R.C.S.; Sydney.
Andrew Shields, M.D., Edin.; Melbourne.
Hon. C. F. Marks, M.D., Dub., M.R.C.S.; Brisbane.
T. G. Davy, M.A., M.D., Ch.B., Oxon.; Perth.
F. Ogston, M.D., Ch.M., Aberd.; Dunedin, N.Z.

Secretary .. .. A. A. Hamilton, B.A., M.D., Ch.M., Dub.; Adelaide.

LIST OF PAPERS.

1. Opening Address by the President.
   Mr. G. A. Syme.
2. Paper—On Hospital Abuse.
   Dr. Bryant.
3. Paying Patients in Public Hospitals.
   Dr. Moore.
4. Diminished Responsibility in relation to Criminal Law.
   Dr. A. Shields.
5. The Role of Provident Medical Associations.
   Dr. George Rennie.
6. The Ethical Relations between Town and Country Practitioners.
   Dr. F. Allwork.

SERVICE AT THE ANGLICAN CATHEDRAL.

SUNDAY, SEPTEMBER 3RD.

A special service in connection with the Australasian Medical Congress was held at St. Peter's Cathedral on Sunday, September 3rd, in the afternoon. There was a very large attendance, His Excellency the Governor and Lady Le Hunte, the Chief Justice and Lady Way, and the Mayor of Adelaide being amongst those present. The choir was augmented and the organ reinforced by an orchestra to render the music specially arranged for the occasion by the organist, Mr. J. M. Dunn, who conducted. Prayers were read by the Precentor, the Rev. Dr. Milne, and the Dean of Adelaide read the lessons. An effective sermon was preached by Canon Hopcraft from Revelations xix., 6, "The Lord God Omnipotent reigneth."

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PROGRAMME OF ENTERTAINMENTS.

—*—

Monday, September 4th.

MAYORAL RECEPTION.

The Mayor of Adelaide (Mr. Theodore Bruce) welcomed the Congress at an "At Home" in the Town Hall, on Monday afternoon. Several hundred guests, including members of the medical profession, their wives and daughters, as well as many other prominent citizens of Adelaide, were received by Mr. and Mrs. Bruce and the Misses Bruce. Shortly after the arrival of Lady Way (the wife of the Lieutenant-Governor) the National Anthem, played on the large organ, announced the arrival of the vice-regal party, which consisted of His Excellency and Lady Le Hunte, the Hon. Victor Hood (Private Secretary), and Miss Swete. Afternoon tea was served in the banqueting hall, and musical selections were given at intervals by Mr. Pybus (City Organist) and a large orchestra.

Tuesday, September 5th.

GOVERNMENT HOUSE GARDEN PARTY.

The perfect weather of the previous Sunday, which changed for the worse on Monday, was still more unsettled when the large number of invited guests were bidden to Government House on Tuesday afternoon. Fortunately the rain held off until the close of the entertainment, and intervals of sunshine gave favorable opportunities for exploring the vice-regal gardens and listening to the music of an excellent band. Refreshments were served upon the lawn, in large marquees.

Tuesday evening was set apart by the Reception Committee for private dinners and house parties. About half of the visitors and the honorary members of Congress were entertained at dinner at the Adelaide Club, on the invitation of its medical members, the club dining-room not being large enough to accommodate the whole number at one time. His Excellency the Governor, a patron of the Congress, was present. The remaining visitors were entertained at private houses.

Wednesday, September 6th.

VISIT TO THE CONSUMPTIVE SANATORIA IN THE HILLS AND TO THE NATIONAL PARK.

Motor cars belonging to local medical men and four five-horse drags conveyed visitors from the University to Belair, where, within a third of a mile from one another, near the National Park, are erected two sanatoria for consumptive patients. One, Nunyara, is a private hospital; the other, Kalyra, a public institution, built by the "James Brown Trust." The visit was so arranged that all could inspect both establishments. Dr. Gault showed visitors over the first-named institution, while Dr. Reissmann (medical superintendent) and some of the trustees conducted the party over the latter. Refreshments were provided at each place, and all were invited to afternoon tea at Dr. T. K. Hamilton's residence, at Belair. A drive through the National Park provided occupation for the ladies and others not interested in sanatorium treatment.

EXCURSION TO THE OUTER HARBOR.

For visitors not interested in sanatoria, an excursion on the Port River was provided. A special train conveyed a couple of hundred guests through

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Port Adelaide and, by the new branch line, to the Outer Harbor works now in progress at Light's Passage. After an inspection of this large undertaking the visitors were invited by Dr. and Mrs. Morris, of Port Adelaide, to return by steamer up the Port River. The Quarantine Station was briefly inspected *en route*, and throughout the afternoon refreshments and music were provided on the launch, which reached the wharf about dusk. Hence a special train conveyed the visitors to Adelaide.

THE PRESIDENT'S RECEPTION.

Nearly 600 guests accepted the invitation of the President and Mrs. Stirling to a reception and ball, at the Elder Hall of the University, on Wednesday evening. This was a brilliant success, the decorations, carried out in excellent taste in yellow and orange artificial flowers and natural foliage, by the Misses Stirling and lady friends, transforming the fine hall into a strikingly pretty ballroom. His Excellency the Governor and Lady Le Hunte, the Chief Justice and Lady Way, and some members of the Government were present. Songs and instrumental music were rendered during the reception, and dancing followed, supper being served in two large classrooms in the basement.

THURSDAY, SEPTEMBER 7TH.

VISIT TO THE SEWAGE FARM AND RAILWAY WORKSHOPS.

A special train conveyed about sixty members and their friends to Islington on Thursday afternoon. Under the guidance of the Hydraulic Engineer (Mr. C. A. Bayer) about twenty visitors alighted at the Sewage Farm, and were shown over the establishment, while the remainder proceeded by train about a mile farther to the Islington Workshops of the South Australian railways. There they were met by Mr. T. Roberts (Chief Mechanical Engineer), and conducted through the buildings by experts from the staff, who explained the various engineering processes. Mr. Roberts entertained the party, joined by those who had visited the Sewage Farm, at afternoon tea, served in the workmen's dining hall, and he was heartily thanked in a brief speech, as was also Mr. Bayer and the Railways Departments.

MRS. SYMONS'S "AT HOME."

Mrs. Symons gave an "At Home" at Pennington Terrace, North Adelaide, to all the lady visitors of the Congress. There was a crowded attendance during the afternoon.

GOVERNMENT HOUSE DINNER.

In the evening His Excellency the Governor entertained at dinner the medical representatives of the other States and the officers of Congress.

DINNER AT THE ADELAIDE CLUB.

Those visitors whom it had not been possible to invite on the previous Tuesday were entertained at dinner in the Adelaide Club, on the invitation of its medical members.

MRS. C. E. TODD'S BALL.

At the North Adelaide Institute, on Thursday evening, Dr. and Mrs. Todd gave a ball in honor of the members of the Congress. The hall was gracefully decorated, and a large number of guests were present.

FRIDAY, SEPTEMBER 8TH.

VISIT TO THE CREMATORIUM.

Vehicles were provided at an early hour to convey those interested to the Crematorium, at the West Terrace Cemetery, to witness the incineration of a sheep. Mr. Owen Smyth (Superintendent of Public Buildings), who had

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invited the visitors, met them at the entrance and showed them over the building. Everything having been previously prepared, the sheep's carcass was introduced into the furnace and soon reduced to ashes, the remains, contained in a small urn, being exhibited at the University on the following day.

MRS. SIMPSON NEWLAND'S GARDEN PARTY.

On Friday afternoon the majority of the visitors attended the garden party given in their honor by Mr. and Mrs. Simpson Newland, at their home, "Undelcarra," at Burnside. There was a large attendance.

SEPTIC TANKS AT GLENELG.

On the same afternoon about fifty visitors went by train to Glenelg. Vehicles were waiting at the station, which conveyed them about a mile and a half out of the town to the septic tanks recently constructed in connection with the new drainage system of Glenelg. Here the Hydraulic Engineer (Mr. C. A. Bayer) met the party and showed them over the establishment. On the return journey the visitors were driven along the esplanade through the seaside town to the residence of Mr. and Mrs. Richard Smith, who had asked a large party to meet them.

PUBLIC HEALTH LECTURE.

At the Town Hall, on Friday evening, a public health lecture was given by Dr. W. G. Armstrong, of Sydney, to a large and appreciative audience, which included many local municipal officers and officials. The subject was "Some aspects of municipal sanitary administration."

THE CHANCELLOR'S CONVERSAZIONE.

On Friday night a conversazione was given by the Chancellor of the University (Sir Samuel Way) to more than 600 guests. After the host and hostess had received their guests in the Elder Hall a musical programme was carried out, while at the same time lecturettes were given in an adjoining lecture theatre by Dr. John Thomson, of Brisbane, and Mr. T. G. Ellery (Town Clerk of Adelaide). On each occasion His Excellency the Governor and Lady Le Hunte were amongst the audience.

SATURDAY, SEPTEMBER 9TH.

EXCURSION TO HAPPY VALLEY.

At noon on Saturday about 150 guests, in ten large drags, journeyed to the Hon. George Brookman's farm, "Glenthorne," near Happy Valley. The drive of an hour and a half's duration was very pleasant, and on arrival at the farm Mr. and Mrs. Brookman had lunch awaiting in a large marquee. Afterwards many availed themselves of the opportunity to inspect the valuable stud on the estate. Rain then, unfortunately, came on, but this did not prevent about sixty guests from going about two miles farther to inspect the Happy Valley reservoir and waterworks.

EXCURSION TO MOUNT LOFTY.

Several drags conveyed the remainder of the visitors to Mount Lofty on Saturday afternoon. Afternoon tea was kindly provided at Mr. J. W. Bakewell's residence, near the summit, by his daughter, Mrs. O'Leary, of Glenelg. Unfortunately it became very wet during the afternoon.

DAILY LUNCHEON AT THE UNIVERSITY.

With the view of economising time, lunch was provided, daily, at the University for members of the Congress; the hosts on these occasions being Dr. Symons and some South Australian country members.

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Australasian Medical Congress.

SEVENTH SESSION,

HELD AT THE BUILDINGS OF THE UNIVERSITY OF ADELAIDE,

SEPTEMBER 4th to 9th, 1905.

FIRST BUSINESS MEETING, SEPTEMBER 4TH, AT NOON.

THE PRESIDENT (Professor E. C. Stirling) occupied the chair, and was supported by the Hon. G. H. Butler, M.L.C., Drs. John Thomson, F. C. Batchelor, J. C. Verco, the General Secretary (Dr. B. Poulton), the Treasurer (Dr. W. T. Hayward), and the Associate Secretary (Dr. J. B. Gunson).

THE PRESIDENT: Members of the Australasian Medical Congress—My first duty should be to thank you very sincerely for the distinguished honor you have conferred on me by electing me to this Presidential Chair; but, if you will permit me, I will postpone my thanks until this evening, when I hope that I may be able to make a more suitable acknowledgment than I do at the present time. Therefore, gentlemen, I propose that we should pass to the business of the day, and commence the work of the Congress. (Cheers.) My first duty is to declare this Congress duly constituted, and, in the second place, I will ask the Secretary, Dr. Poulton, to read the report of the Executive Committee. (Cheers.)

THE SECRETARY read the report of the Executive Committee, as follows:—

REPORT OF EXECUTIVE COMMITTEE TO CONGRESS.

At the last meeting of the Intercolonial Medical Congress of Australasia, held in Hobart during February, 1902, under the presidency of the Hon. G. H. Butler, M.L.C., it was decided—"That the name of Congress be altered from 'The Intercolonial Medical Congress of Australasia' to 'The Australasian Medical Congress,' and that the succeeding session be held in Adelaide in 1905."

On the motion of PROFESSOR ALLEN, M.D., Professor E. C. Stirling, C.M.G., F.R.S., M.D., Cantab., F.R.C.S., was unanimously elected President.

At a representative meeting of the South Australian Medical Profession held on May 29th, 1902, the following gentlemen were elected by ballot as the Executive Committee of the Australasian Medical Congress to be held in Adelaide in 1905:—

Dr. W. T. Corbin	Dr. M. J. Symons
" W. Anstey Giles	" C. E. Todd
" J. B. Gunson	" J. C. Verco
" J. A. G. Hamilton	Professor Watson
" R. H. Marten	

Dr. W. T. Hayward was elected Honorary Treasurer, and Dr. B. Poulton General Secretary.

The Executive Committee, so elected, added to their number Drs. T. Borthwick, R. Brummitt, Cavenagh-Mainwaring, W. L. Cleland, A. H. Gault,

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A. A. Hamilton, T. K. Hamilton, R. E. Harrold, Melville Jay, A. A. Lendon, A. M. Morgan, B. Smeaton, A. E. Wigg, W. B. Aitken, John Johnson, O. W. Smith, J. I. Sangster, H. A. Powell, C. H. Souter, M. P. O'Leary, T. James, E. L. Archer, R. W. Stewart, J. H. G. Drummond, J. H. Evans, R. St. Mark Dawes, E. W. Morris, F. S. Hone, and F. Goldsmith, and constituted themselves " The General Congress Committee."

This committee determined the division of Congress into seven sections, and appointed a secretary for each section. It also took preliminary steps for securing Presidents and Vice-Presidents of Sections, and Secretaries of Congress, in the several States, and appointed Dr. J. B. Gunson Associate Secretary; and subsequently, in August, 1902, nominated a standing Executive Committee for the conduct of all ordinary business of Congress consisting of:—

EXECUTIVE COMMITTEE.

The President	J. A. G. Hamilton
The Treasurer	T. K. Hamilton
The General Secretary	Melville Jay
The Associate Secretary	A. A. Lendon
T. Borthwick	R. H. Marten
W. R. Cavenagh-Mainwaring	H. Swift
W. L. Cleland	M. J. Symons
T. W. Corbin	C. E. Todd
W. Anstey Giles	J. C. Verco
A. A. Hamilton	Professor A. Watson

The Executive Committee has been cordially supported in its labors by many leaders of the profession in the various States, and has, in accordance with precedent, elected Presidents and Vice-Presidents of Sections from all the States except South Australia. Their Excellencies the Governor-General of Australasia, the late Governor-General, and the Governors of the several States of the Commonwealth, and of New Zealand have, with the Lieutenant-Governor of South Australia, graciously consented to become Patrons of the Congress which will be inaugurated by His Excellency Sir George R. Le Hunte, Governor of South Australia.

The Government of this State has very generously consented to print the transactions, and offers every facility to members for visiting Government institutions and establishments. Government has also, at the instance of the Executive Committee, secured the appointment by the other States of Government representatives in the persons of Dr. Ashburton Thompson (New South Wales), Dr. John Thomson (Queensland), Dr. T. H. Lovegrove (Western Australia), Dr. Elkington (Tasmania), and Dr. Mason (New Zealand). Victoria did not appoint a representative.

Dr. James Jamieson has been specially appointed to attend as representing the Melbourne City Council, and Dr. W. G. Armstrong by the city of Sydney.

Matters closely concerning Military Medicine and the Health of Railway and other Officials were brought under the notice of the Hobart Congress; and facilities will be offered during the present sitting for special meetings of Army Medical Officers and members of the profession connected with the Railway Departments.

The Committee have been most ably assisted by the advice and support of the State Secretaries, who were chosen so long ago as December, 1902, and who, since then, have continually worked to promote the success of this meeting. They are as follows:—Drs. A. A. Palmer, G. A. Syme, Wilton W. Love, A. J. H. Saw, Gregory Sprott, and J. O. Closs.

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The editors of the *Australasian Medical Gazette* and *The Australian Medical Journal* have constantly aided Congress in communicating with the profession. The committee wish gratefully to acknowledge the concessions given to members by the various State Governments with reference to special railway fares, and by the South Australian Railway Department in allowing them to travel in this State at single fares for double journeys. They desire also to record their appreciation of the generous allowances made by the shipping companies; and especially their sense of indebtedness to the Council of the University of Adelaide for the use of the greater part of these buildings, and the cordial sympathy extended by them to the work of Congress.

E. C. STIRLING, President.

September 4th, 1905. BEN. POULTON, General Secretary.

DR. POULTON: I move the adoption of this report.

DR. J. C. VERCO: I second that.

The motion was carried.

THE BALANCE-SHEET.

THE PRESIDENT: I ask for the Treasurer's report.

THE TREASURER: It is the duty of the Treasurer to report on the balance-sheet with which he has had nothing to do. I am really giving the report of the last Treasurer, Dr. Wolfhagen.

THE INTERCOLONIAL MEDICAL CONGRESS.—HOBART SESSION, FEBRUARY, 1902.

In Account with the Hon. Treasurer.

Cr.	£	s.	d.	Dr.	£	s	d.
By Subscriptions—				To Expenses Local Branches—			
318 members at £1 1s. ..	333	18	0	New South Wales £8 7 1			
" Remittance from Treasurer Brisbane Congress	109	19	4	Victoria 7 8 6			
" Proceeds Sale of Typewriter (Brisbane)	2	14	0	South Australia.. 3 7 0			
" Exhibitors.....	17	5	0	New Zealand.... 2 9 0			
" Savings Bank Interest.....	5	8	8	Queensland 2 1 0			
					23	12	7
				" Expenses, Hobart..	306	16	0
				" Remittance to Treasurer Adelaide Congress	100	0	0
				" Balance Remitted to Treasurer Adelaide Congress ..	38	16	5
	£469	5	0		£469	5	0

Audited and found correct. J. E. WOLFHAGEN, Hon. Treasurer.

F. B. BATTLE, Auditor.

In moving the adoption of the balance-sheet, I congratulate the Hobart Congress on the great success they had. They not only sent to us the amount of money they received from Brisbane, but exceeded it by about £30. (Cheers.)

DR. BEESTON (Newcastle): I second the adoption of the balance-sheet.

The motion was carried.

DR. ODILLO MAHER.

THE PRESIDENT: Before we pass on to the next business I should like to make reference to the regret we experience at the absence of Dr. Odillo Maher. He, unfortunately, has been prevented from being present by a family affliction, and we regret his absence from our meetings. (Hear, hear.)

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Congratulations.

The President: I should also like to communicate to the Congress that there has been received from the late Mayor of Hobart (Mr. G. Kerr), a telegram—which reads as follows:—

> "Mayor of Hobart congratulates President, and wishes Medical Congress now assembled successful deliberations."

Those who were at the Hobart Congress no doubt will remember that they were greatly indebted to the mayor for the interest which he took in its proceedings, and particularly for the hospitality which he, and those connected with him, extended to all the members. (Cheers.) If any member of the Congress has any motion of urgency, now is the time to bring it forward.

Sectional Resolutions.

Dr. W. T. Hayward (Adelaide): I suppose at this Congress, like previous ones, we shall have resolutions brought up from the different sections. I have been struck with the apparent incongruity of Congress passing resolutions on the last day, when everybody is hurrying away to an interesting picnic or something of that kind, and those resolutions going forth with the imprimatur of Congress, without consideration from the Congress as a whole. It would be much better if the sections which pass these resolutions took the responsibility of them. If they did that, the resolutions would, I believe, stand a very much better chance of being carried out; because they would be dealt with by the energetic secretaries of the sections, instead of being left to the Executive Committee of the next Congress, which would probably not be appointed for some two or three years afterwards. I was rather amused on looking over the Congress report the other day, in order to find what was done in the other States, to see that I had been appointed a member of a committee to consider certain subjects. That was the first intimation I had that I had been appointed. In these circumstances I propose:—

> "That all resolutions adopted by sections shall be reported to the Congress, but that discussions shall take place only on such as have been specially recommended by the section for adoption by the Congress."

I admit that there are certain resolutions that require the whole weight of Congress behind them. These, I think, should be adopted by Congress, but a great many of them are technical, particularly the Eye Section and Public Health, which I think would be better left to be dealt with by the section themselves.

Dr. Gregory Sprott (Tasmania): I second that.

Dr. Anstey Giles (Adelaide): Is it in order that this motion should be put without notice?

The President: Perhaps Dr. Giles is right, but there is some reason for it, and I think it is in the power of the chair to admit it. If we discuss it now, we shall have clear instructions for next Saturday's meeting, when the real motions will come up. I therefore rule that it is in order, and can be discussed now. I invite discussion on the point. It appears to me to be perfectly reasonable.

Dr. Kent Hughes (Melbourne): Would it not be better to refer the matter to the Executive Committee, rather than to the sections? A section might think a subject of more importance than would the Executive Committee.

The President: Dr. Hayward's motion intends that some things shall go on to the records of the Congress, as the opinions of the sections, but there

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may be other things that should be adopted as congressional resolutions. It leaves the matter perfectly within the power of the section to recommend it to the Congress.

The motion was carried.

THE PRESIDENT: It is now my duty to adjourn this meeting until 10 a.m. on Saturday. Will you please allow me to say that it is impossible, in a gathering of this kind, that there should not be some errors of omission and commission. Those who have been in the position of the General Executive of these Congresses will have experienced this difficulty when dealing with a large number of visitors. Disappointments are bound to take place in the arrival of so many visitors, and it is easy for the committee to make very regrettable errors in the distribution of invitations. Therefore, on behalf of my colleagues and myself, I beg of you, if there have been such omissions—and we recognise that there have been—that you will be good enough to communicate the same to either Dr. Poulton or Dr. Gunson, and we shall have every regret that the mistake has been made, and make every effort to remedy it as soon as possible.

The meeting was adjourned until Saturday morning.

THE INAUGURAL MEETING.

OPENED BY HIS EXCELLENCY THE GOVERNOR.

The inaugural meeting of Congress was held in the Elder Hall, University Buildings, on Monday evening. There was a large and distinguished audience in the body of the hall, and among those on the platform, in addition to the President (Professor Stirling), were His Excellency the Governor (Sir George Le Hunte), the Chief Justice (Sir Samuel Way, Bart., Chancellor of the University), the Premier (Hon. T. Price, M.P.), the President of the Legislative Council (Sir Lancelot Stirling), the Speaker of the House of Assembly (Sir Jenkin Coles), the Mayor of Adelaide (Mr. Theodore Bruce), the Vice-Chancellor of the University (Dr. Barlow), the Leader of the Opposition (Hon. R. Butler), and members of the Executive of the Congress, and of the Council and Senate of the University.

THE PRESIDENT: Ladies and Gentlemen—In the name and on behalf of the Australasian Medical Congress, I ask His Excellency the Governor to declare open, this, its seventh session. (Applause.)

HIS EXCELLENCY: Mr. President, your Honor, Mr. Premier, your Worship, Ladies and Gentlemen—Before I formally declare this session open I wish, on behalf of His Majesty the King and the State of South Australia, to extend to all those visitors who have come to the Congress a most hearty welcome. (Applause.) You had a welcome tendered to you yesterday in that most magnificent address which you heard from the pulpit in the Cathedral by Canon Hopcraft, and it is my great pleasure and privilege to repeat his welcome to you. About six years ago I had the honor of being present at your Congress in Brisbane, when Lord Lamington was Governor of Queensland. He began his address to you by apologising for the weather. I am very sorry that I have almost to follow in His Lordship's footsteps. However, that is a matter which is beyond our control, and even beyond the control of that excellent Executive Committee who have worked out so well and so fully the details of the Congress for you. I am sorry for one thing—that the time of the Congress was obliged to be fixed just at this particular period of the year, because had you come here a little later, we would have been able to show you a wealth of color of flowers in our gardens which just now are at their very worst. Adelaide is at all times worth visiting and seeing, but

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when it has its summer garb, or as our commercial friends would say, "when we open up our summer lines," there is no city in the Southern Hemisphere which, at any rate, could surpass it. But though the warmth is not on the surface, it is in our hearts towards you. (Applause.) It is a very pleasing moment for South Australia, that after eighteen years the cycle of time should have brought around this Congress to where it was first born. Those who took part in that Congress, and were instrumental in giving it its first start, must feel extremely gratified at the results of their efforts and their foresight. I will not keep you any longer now, because others have to speak, and we are looking forward to the address of our able President, Professor Stirling. I now have the honor to formally declare this seventh session of the Australasian Medical Congress duly open. (Applause.)

THE PREMIER: Your Excellency, Mr. President, Ladies and Gentlemen—It affords me great pleasure to perform my portion of the proceedings this evening, and welcome the Congress to South Australia. It falls to my lot because at the present time I happen to be in the position of Premier of the State. When I got that position I never thought I should have to appear on a platform like this, before such an audience. I am sure I could get plenty of men who could perform this part of the programme better than myself, but I want to assure you that, although they may be able to do it better, none could do it more heartily than I do it now. (Cheers.) The people of South Australia welcome you here because we believe that you are engaged in a work that is to the advancement of humanity generally, and because it is not a close movement—this great movement of medicine. It is a movement on behalf of mankind generally. We are all indebted to you for the amount of work you put into it, and for the sacrifices you make. I want to say to-night that we have much to thank the medical profession for—in performing their duties in attending to the wants of society—especially among the poorer classes of the community. I know it from my own experience that the doctors in the poorer parts of the cities are men who are nobler than missionaries, and greater even than those, in my opinion, who occupy our pulpits, because they put practical work into their lives. I am sure the same feelings animate the whole of the medical profession throughout the world. I have known within my own knowledge of a medical man who paid the most minute attention to cases where he never expected to get a farthing out of them. That comes close to my home. I remember my mother being afflicted with a certain trouble, and a doctor in Liverpool took great interest in her. For five or six years he attended to her, and at last had the satisfaction of seeing a perfect cure. I remember when she was told that she was not to go any more. And I remember also the scene at home, and the thought also of how much we would have to pay, and how we were going to pay it. I remember the answer that came back—"Do you think two guineas is enough?" And there was rejoicing in that home. As a rule the honest poor are very anxious to pay their liabilities. When there is generosity to them, as in the case I have mentioned, there is rejoicing. The medical profession are blamed sometimes for charging too much, and thank God, some of them know where to charge and where not to charge. I hope that you will be able to discover not only new means of treating disease, but means of making the people pay you when you have done your work. (Laughter, and cheers.)

THE MAYOR: Your Excellency, Mr. President, your Honor, Ladies and Gentlemen—In the position I hold to-day it affords me great pleasure to welcome the members of the Congress to South Australia, and particularly to the city of Adelaide. The citizens of Adelaide are particularly proud of and pleased with the town in which they live. We look upon it as being

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de facto one of the most healthy towns in Australasia, and the credit of that to a great extent is due to the number of very able medical men we have in our midst. (Cheers.) The doctors in South Australia are, I believe, such a very excellent lot, and so worthy of imitation, that I have not the slightest doubt that those gentlemen who have arrived from the other States will carry away with them a great deal of information that they otherwise would not be able to obtain. (Cheers and laughter.) I am extremely pleased to know that the first Medical Congress was held in the city of Adelaide, and was presided over by Dr. J. C. Verco, whom I am glad to see on the platform to-night—(Cheers)—and of whom it might be said in the words of the immortal bard—

Time cannot wither nor custom stale his infinite variety.

I remember going to Victoria, and consulting a medical man there for a complaint, and he told me that it was unnecessary for me to go there, because we had medical men over here who could combat equally with those in the sister State any disease known in the cyclopœdia. I hope during your stay in Adelaide you will have a good time. I believe a good many fetes and entertainments have been provided for you, and I hope the weather will hold up, so that you may have a really enjoyable time. I am perfectly sure, knowing as I do know so many members of your profession, that your deliberations will be fraught with and provocative of doing a vast amount of good to the people whom you desire to benefit. There is no profession among the learned professions that I admire so much, or in which I take so much interest, as the medical profession. It is *the* one, if I may say it, except the Church, that has for its object the raising of humanity, the amelioration of suffering, and the assistance of mankind generally. I hope and trust you will have a good time, and that your deliberations will be provocative of a great amount of good to the honorable and learned profession of which you are all such distinguished members. (Cheers.)

PRESIDENT'S INAUGURAL ADDRESS.

The President of the Congress (Professor E. C. Stirling, C.M.G., M.D., F.R.C.S., F.R.S.) then delivered the inaugural address on "Medical Science and Social Problems":—

Your Excellency, Members of the Australasian Medical Congress, Ladies and Gentlemen.—The first and very pleasant duty that falls to my lot as President of this Congress is to welcome, in the name of the medical profession of South Australia, our brethren from beyond our borders. If these, to some extent, still divide us politically, yet they form no barriers to the confraternisation of the members of the great army of medical science whose common object is the relief of suffering and abolition of disease. On our visitors do we, in this numerically small State, very greatly rely for the success of this meeting which they have honored by their presence. Though South Australia cannot, indeed, offer the scenic attractions and advantages of some of the other States, yet we, its citizens, like to think that we are not inferior to our neighbors in the warmth of the personal and social welcome we endeavor to extend to those who favor us with their too infrequent visits. On such an occasion as the present we may be relied upon to do our best to uphold what reputation for hospitality we may be thought to possess. May I also be the mouthpiece of the Congress in welcoming to this meeting all those who, though not belonging to the medical profession, yet have shown their interest in our proceedings and in the cause we represent by being present at a gathering which can scarcely have appealed to them as a recreationary attraction.

For my second duty the more difficult task presents itself of acknowledging, in an adequate manner, the compliment which has been conferred

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upon me by my election to a presidential chair that has been occupied by some of the most distinguished members of the profession in Australasia. I take this earliest opportunity of expressing my deep appreciation of the highest honor which lies in the power of my medical colleagues to bestow on one of their number. May I be found as worthy of their confidence as were those who have preceded me in the dignities of the position, in its duties and, will not all my predecessors agree with me if I add, in its great anxieties and responsibilities. In this particular instance such an acknowledgment almost requires the inclusion of an apology, seeing that I, standing for some years outside the active practice of our profession, feel myself in some respects unqualified to address my experienced colleagues on many matters which lie nearest to their thoughts and work. Instruction or inspiration is not in my power to offer, but I console myself with the thought that there may, perhaps, be some propriety, or even advantage, in an arrangement whereby, every now and then, the opportunity of speaking *ex cathedra* should be sometimes given to those whose daily work consists in the consideration of the healing art rather from the point of view of its underlying principles than from that of their adaptation to the requirements of actual practice.

This, your Excellency, ladies and gentlemen, is not an ordinary anniversary; I am glad to think by the faces I see before me that there are many here this evening who need no reminding that the Institution of Intercolonial Medical Congresses, to give them their original name, was founded in South Australia, and that in Adelaide, in 1887, was held the first of the series of meetings that have continued held uninterruptedly ever since to the great benefit of ourselves and, I would fain think, of our calling. Now, after these eventful years it returns to us for the second time. It is pleasant to see here, older, by those eighteen years that have passed since he held office, but still in the prime of intellectual life, the first President of the first Congress—Dr. Verco. So also do we welcome those other past Presidents—Dr. Batchelor, President of the New Zealand Congress in 1896; Dr. John Thomson, President of the Queensland Congress in 1899; and Dr. Butler, President of the Tasmanian, and last meeting, held in 1902, who, in their respective States, have so worthily upheld the dignity of their country and their profession as to make their example difficult of emulation for their successors.

Still with us, also, is the real originator of the system of Congresses, Dr. Poulton, the Secretary of the first meeting, as he is again of this. Neither has he lost his superabundant energies of eighteen years ago, which, to our great advantage, he now, once more, places at our disposal. And other distinguished participators of the first Congress I see, now become the Nestors of their profession, whom we gladly welcome to Adelaide for the second time.

How, then, shall I use this, an opportunity that comes once only in a man's lifetime, when he may address not only his assembled colleagues, but those now numerous members of the public to whom the widespread dissemination of scientific information has brought some knowledge of the romance of medicine and surgery.

On occasions such as these, which serve as milestones in our lives and in our profession, it is a time-honored custom for him who stands in the position which I have the honor to occupy to-night to review the progress of that particular branch of the profession with which his life's work has been identified. For one whose special interests were, for more than a quarter of a century, centred in the practice and principles of surgery, it is a tempting theme to exalt the horn of his calling by dwelling on its phenomenal advance during a period of which his own student days saw the beginning. But that has been an oft-told tale; it has been told by some of those who themselves

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were in the forefront of that triumphant march which has brought surgery to a pitch of success undreampt of even only half a century ago, and I need not repeat it.

The thaumaturgic proceedings of the operating-room have, indeed, always appealed to the popular imagination, and their results have become perfectly familiar to the intelligent public, either as the outcome of their own personal experience or of the general spread of information which is one of the characteristics of this age of publicity. Indeed we know how each remarkable discovery is served up to us by the daily press on the morning following its announcement in the country of its origin.

The brilliancy of the results of surgery not infrequently gives rise to disparaging comparisons between its progress and that of its less spectacular sister, the science of medicine. But this disparagement is not wholly justified, for medicine, if it has been later than surgery to respond to the vivifying influences of the ancillary sciences, has now at last entered upon an era of remarkable discovery pregnant with infinite possibilities for the future.

If surgery, as a craft, has, in the period in which we live, reached its golden age, it is possible to predict with great assurance that the present century will witness an equal if not greater advance on the part of medicine and its collateral branches. Indeed, as we can plainly see, that advance has now begun. Already the new therapeutics and the new science of preventive medicine has accomplished unexpected results, while offering boundless promise of achievements in the future.

About thirty years ago a distinguished English surgeon, a leader of the profession, gave it as his opinion that operative surgery had nearly, if not quite, reached its ultimate limits, and that certain regions of the body must perforce remain sacred from interference. But a great deal of water has flowed under the bridge since that time. Surgery has extended its scope far beyond what then appeared possible. Almost every organ of the body, including such important structures as the spleen, kidneys, lungs, liver and its appendages, the brain and spinal cord, are now successfully invaded by the surgeon's knife; some of them are even entirely removed. The heart itself, the centre of the circulation, the slightest wound of which was, not so long ago, thought to be a fatal injury is not exempt from interference, for there have been cases in which wounds of this organ have been successfully sewn up; the feasibility has even been seriously considered of removing the obstruction to the circulation caused by the disease of its valves, which as experimental physiology has shown, need not be regarded as impracticable. Long lengths, to be measured in feet, are, with impunity, cut out of the intestines and the continuity of the remainder restored. The stomach itself, that long-suffering organ than which, as has been well said by a surgical cynic, no ass is more heavy-laden, has, by its successful removal, been proved to be one of those useless superfluities of which not a few encumber our far from structurally perfect bodies. But it must be clear that in a finite body there must be some limit to our interference with organs on the adequate integrity of which the maintenance of life depends, and, if we have not yet quite reached those limitations, we can hardly doubt that, so far as actual removal of parts are concerned, there cannot in this respect be many more worlds to conquer for the surgical Alexander. But, even if this be the case, it is quite possible that surgery may be considerably extended on the constructive side, so that we may be able to engraft, wholly or in part, complex organs as we do now in our transplantations of skin, bone, teeth, and nerve; and, of course, there is still abundant room for improvement in our methods of operation and means of diagnosis.

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May I venture in this connection to emphasise the importance of the too often neglected *minutiæ* of human anatomy. Now that surgical interference is pushed to the utmost limits of corporeal endurance, and almost every doctor considers himself capable of undertaking the most intricate operations, a refined knowledge of the smallest structural details of the human body is more than ever imperative. A due regard to those methods of operation which will ensure the least possible disturbance of parts and the least interference of normal functions may make all the difference between success and failure. But it is astonishing what the human body will endure.

> "Oh! the little more, and how much it is;
> And the little less, and what worlds away!"

To whom shall this appeal with greater and more painful force than to those whose "little less" of knowledge or of skill, that might have been the "little more," has cost a human life.

But after all, though I speak as a surgeon proud of my calling, our craft is, we must admit, in many of its proceedings, a rough and ready sort of business, seeking to cut the Gordian knot of disease rather than to untie it. Though the surgeon's knife will probably remain the *ultima ratio* of treatment of many of those injuries, deformities, and accidents to which our body is subject, partly on account of its own inherent imperfections, yet the discoveries of recent years render it permissible, and by no means visionary, to conceive of more rational and less violent therapeutics than the cutting off, or cutting out, of diseased organs or tissues. Indeed we are even now sometimes able to achieve this object by appropriate medication. Better still, the counsel of perfection would be to substitute prevention for our cures, for indeed there is no reason why the principles of the new science of the preventive medicine should not equally be applicable to the surgical manifestations of disease.

THE OLD AND THE NEW MEDICINE.

After long epochs of slow and gradual growth, interrupted by dark ages of stagnation and superstition, both medicine and surgery have, in our own times, witnessed a remarkable and, relatively to past epochs, sudden expansion, not only in their methods, but in their underlying principles. Between the old and the new medicine there is a difference not merely of degree, but, in a great measure, of kind. What, then, we may ask, is the nature of the new influences which, to so conspicuous a degree, and with a potency exceeding that of past forces, have served both as a stimulus to and as the basis of this sudden bound of progress. It may not be without some interest, even to those outside the profession, if we see what answer can be given to this question.

In ancient Greece, some time during the three centuries that preceded the Christian era, there arose, amongst the physicians of the day, a sect who, because they based their practice on experience alone, to the exclusion of generalisation, reasoning, and analogy, termed themselves the Empirics, this word being the Anglicised form of the Greek word, meaning "one who is guided by experience." Another sect, which flourished at the same time, held experience to be valueless, and founded its doctrine exclusively upon reasoning and theory. These styled themselves Methodists, because they based their practice on a simple method which we might call a "rule of thumb." To this sect Galen, one of the greatest names in the early history of medicine, belonged. A third sect availed themselves of both experience and reasoning, and were called the Dogmatists: thus, even in these early days, there were schisms in the profession, and it is not surprising to learn that between them there was much and bitter controversy. Truly our medical forefathers were very human.

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The Empirics were controversially defeated; the sect died out, and the name Empiric, no doubt from its association with what was considered to be a defunct medical heresy, became a term of opprobrium which connoted quackery, and, as applied to medicine, it still retains much of its sinister meaning.

But if the Empirics as a distinct medical sect died out, their basic doctrine of practice has survived. For long ages empiricism—using the term now in its literal and proper sense—has been one of the principal foundations of our medical practice: to the wise empiricism of our medical forefathers it has owed much, and we must admit that on a method of sound empiricism successful clinical treatment greatly rests, and must continue to rest, as indeed do most of the judgments of our daily lives.

And here let me observe that when we are apt to decry the manner of practice of our forefathers in the exaltation of our own methods, we should remember that it is questionable whether we of the present day are in all respects the equals, in powers of assiduous and attentive observation, of those great masters of the profession who so long ago preceded us. Without the multitudinous mechanical and other aids to diagnosis that we now possess, and that to some extent are substitutes for natural wits, yet these men, by their unaided powers of observation, were often able to form an extraordinarily correct conception of the conditions and course of a disease. Their methods of treatment, indeed, purely empirical though they were, had, in many cases, a success that we have not been able greatly to improve upon. So, then, let us ever remember when we boast of our modern progress that our predecessors of old were not all ignorant, nor always wrong in their methods and judgments, and that they have left us a rich legacy of recorded observations and clinical insight into disease that stand as an example of how much may be achieved by the unaided senses and by natural intelligence wisely applied. In many ways we might well ask for "the old paths, where is the good way, and walk therein."

But so long as our practice continued to be based only upon empirical methods, to the exclusion of reasoned processes which could enable us to recognise the underlying principles of which the observed facts were but scattered expressions, so long could medicine lay no legitimate claim to come within the category of the sciences. To the dignity of that position both medicine and surgery may now, with some justice, lay claim.

No longer does medical practice rest upon a chaos—an incoherent and unrelated aggregate of observed facts; no longer are we satisfied to know that such and such things are, but we have begun to ask the questions why and how they are, and when we are able to give even a partial answer to these questions our knowledge ceases to be empirical, and becomes rational or scientific, and it is this just in so far as our answers are complete. In other words, medicine no longer contents itself with the knowledge, however accurately determined, or however often recorded, that a disease has such and such a set of symptoms, and that, for some unexplained reason, such and such a treatment is beneficial or detrimental, but it seeks to know what are the intimate causes of the perturbed action, why does it arise, why does it run the definite and constant course which has been observed to characterise it, or which distinguishes it from other kinds of perturbed action, and why should a particular kind of treatment be beneficial or the reverse.

When we are able to give answers to questions of this kind in a sufficient number of corelated cases, we are able to establish certain laws of disease that embody principles which guide us in our practice, and give to medicine not only rational methods but extend infinitely its scope of action. A sound principle, or even a wrong hypothesis that has been rightly used, is worth any number of unrelated facts. *Felix qui potuit rerum cognoscere causas* is just as true for

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the physician as for the philosopher; nay, twice happy is the former who can by his knowledge project his own felicity into the bodies and minds of those unto whom he ministers.

It is part of the aims of this address to bring before the notice of those to whom the facts may be unfamiliar some conspicuous illustrations of the wide influence that scientific principles have exerted upon medical science; to show that the phenomenal progress which the last half-century has witnessed in various departments of our profession has been due to the application of these principles, and that what has been achieved already in this direction is only an earnest of further and perhaps greater triumphs in the future. Here, then, is the difference between the old and the new medicine. The old based its methods, often we must admit with great practical success, upon empiricism and irrational hypotheses. The new, while retaining what is good of the old methods, seeks for the deeper and the truer insight into the How and the Wherefore.

THE GERM THEORY.

In these days, when everyone is familiar with the extensive role played by the microscopic organisms, which collectively we call germs, in the causation and manifestations of disease, it almost requires an apology for alluding to so well-known a story. But, as no more striking example can be mentioned of the far-reaching influence that may follow the application of a scientific conception, may I be pardoned if I make some brief allusion to the subject.

Now-a-days everybody, even that much talked of individual the "man in the street," is familiar with the fact that the great successes of surgery have been due to the co-operation of two factors—the use of anæsthetics and the adoption of aseptic and antiseptic procedures. Everybody knows that the object of these methods is, in the former case, to exclude from entrance into open wounds and susceptible surfaces those noxious vegetable organisms, generally found in the neighborhood of man, which, when they do enter, are the causes of the local putrefactions and dangerous infections of the system commonly called blood poisonings. Antisepsis has the less favorable design of destroying the germs if unfortunately they should have already gained entrance. Not only have these aseptic methods permitted a vastly extended scope of surgical procedures, especially in the abdominal region, but they have also led to the practical abolition of those former scourges of surgical practice, hospital gangrene, septicæmia, erysipelas, and other forms of blood poisoning, secondary hæmorrhage, and such like surgical calamities. And if puerperal and its allies still exist more frequently than they should do, it is principally because of the ignorance and want of surgical cleanliness of those obstetric anachronisms—the amateur Mrs. Gamps, who so confidently and so gleefully preside over the occasions which are liable to give rise to this class of complaints.

Now, for the purposes of my argument, the point is that all these great results resulting from the use of aseptic methods may, with much truth, be said to have sprung from a single bio-chemical discovery which, when it was made, had no connection whatever with medicine or surgery, indeed it had reference to the very prosaic and apparently remote question of the making of beer. Nevertheless it was undoubtedly Pasteur's classical investigations into the causes and conditions of fermentation which formed the immediate starting point, and, indeed, the foundation of the aseptic methods of treatment with which the name of Lister will ever be honorably associated. For, applying the principles of Pasteur's discovery to surgery, Lister showed that just as fermentation is caused by the presence and action of certain definite, living, vegetable organisms, so putrefaction in wounds, which is only a special

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variety of fermentation, is similarly caused by certain other minute organisms, and he proved also that the same kind of methods which would prevent the one process would prevent the other. Thus the practical question, in respect to surgery, resolved itself, as we have said, virtually into methods for the exclusion from wounds of these organisms, or for their destruction should they have gained entrance. No better example could be given of the world of difference that lies between the knowledge of a fact and the knowledge of its cause. Our forefathers were perfectly cognisant, indeed they had disastrous experience, of the broad fact that putrefactive processes were apt to be set up in wounds, but, being ignorant of the cause, they were powerless to avert the fatal results.

The principles involved in Pasteur's discoveries in the field of fermentation were soon destined to receive a still wider application, for they led to the discovery that many of the diseases we term infectious were due to the entrance into, and operation within, the body of other organisms of a nature allied to those which cause the infection of wounds. And if still there are some infectious complaints in which the specific germ has not yet, with certainty, been detected, we may be absolutely sure that for every such disease there is an organic generator, and, further, there is even much to justify the view that other diseases than those now generally classed with the infectious group, such, for instance, as rheumatism, may be due to similar causation. And need I say that we are ever on the alert to discover a possible germ of cancer.

Nor is the knowledge gained from this generalisation limited in its application to man and his diseases; it has been extended to those pestilences which at times, in certain places and with such fatality, infest the domestic animals; it has been successfully applied to the improvement of economic and manufacturing processes, and, in the light of the same knowledge, we have learned the nature of those chemical changes taking place in the soil by which it becomes better fitted for the nutrition of vegetable crops. The formation of nitrates which are so valuable as fertilisers we now know to be due to the action of vegetable organisms similar to these which produce disease, and when these are absent, or deficient, we can supply them by direct inoculation.

How wide, then, and far-reaching has been the influence of this one biochemical discovery of the organic causes of fermentation, and I have but merely touched upon the fringe of an immense subject. Truly the value of a new principle, however limited its application may at first appear to be, is incalculable, and the direction which its influence may take unlimited.

It is, however, clear that these discoveries, great as they are, form but the introduction of fresh problems. It is a great step to have traced the causation of disease to specific organisms which are tangible entities that may be seen, isolated and used as we will for the experimental confirmation of our theories. But our canon requires that causation should be pushed still further back, for there arises the next question—how and why do these germs produce their effects? Into this domain of inquiry we have now begun to penetrate. But as these considerations involve questions of somewhat greater complexity, I shall most conveniently bring them under your notice in a concrete form. And, in doing so, may I seek once more the indulgence of my medical brethren for dealing with matters which to them are so familiar.

TOXINS, ANTITOXINS, AND IMMUNITY.

Diphtheria, one of the oldest known of infectious disorders, is caused by specific living organisms which, as a rule, gain entrance into the body through the nose or throat. Settling on some part of these regions, they

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rapidly multiply and, at the same time, secrete a poisonous substance or toxin. This entering the circulation is distributed throughout the body, and poisons the tissues by interfering with the normal processes by which they maintain their vitality. In short, a *contagium animatum* from without produces a *virus inanimum*—a lifeless poison which does the mischief within. Now it is found that if some of this diptheria toxin which, up to a certain degree of purity, can be obtained separately by growing the diphtheria germ in suitable culture mediums outside the body be repeatedly, and in successively increasing doses, injected into the blood of a horse, this animal becomes so affected that it acquires, at length, the capacity of withstanding, without symptoms, a dose of the toxin that would, without preliminary treatment, inevitably have killed it. In other words, using the terminology of the day, we say that the horse has been made immune to this special diphtheria poison. In this condition the serum, or fluid part of the horse's blood, is found to contain a substance which, because it is antidotal to or capable of neutralising, diphtheria toxin, is called antitoxin. And when a suitable dose of this serum is injected into the blood of a human being it may act either by preventing an attack of diphtheria—that is, it confers immunity in this respect—or, if the disease has not advanced too far, it acts as a curative agent. From this method of treatment arises the term so much in evidence at the present time—serum therapy; and speaking of this one disease—diphtheria—it may safely be said that by its timely application thousands of lives have been saved.

Now it must be understood that this conclusion as to the antagonism of toxin and antitoxin does not merely rest on the results of trials on the living bodies of man or other animals, but its main facts are based securely on experiments in the laboratory, where it can be shown that if the proper proportions of the two substances are added together in a test tube, the mixture is harmless when injected; the poisonous toxin has, in fact, been neutralised by the antitoxin.

The case of diphtheria presents us with one of the most satisfactory examples of the remedial application of the antidotal action of antitoxin on a toxin produced by bacterial action. In some other diseases, of which the now comparatively rare disease, tetanus or lock-jaw, may be taken as an example, though laboratory experiments justify similar conclusions as to the manner of formation and interaction of these antagonistic substances, yet the application of this kind of treatment has not so far yielded such satisfactory results as in the case of diphtheria. For this relative failure we can recognise reasons which in no way invalidate the general conclusions, but these it is not necessary to discuss. We find, also, that the toxins arising from bacterial action are not alone in their power of generating antitoxins, for each kind of snake poison will similarly produce its own antitoxin, which, if it can be applied in time, acts as an efficient antidote, and so will ricin, the poisonous element of the castor oil bean, and abrin, the active principle of those bright red and black seeds called crabs eyes or jequirity, both these last-named poisons being far more powerful than strychnine. In all these cases the reaction may be demonstrated in the most certain manner in a laboratory experiment.

Considered merely in the light of a system applicable to the treatment of particular diseases, these facts concerning toxins and antitoxins are of great importance; but of still greater value are certain underlying principles which have emerged from some recent investigations on this subject, with which the name of Ehrlich will ever honorably be associated, although, as in most other great discoveries, the whole result is not the work of a single mind. The great merit, in this respect, of this distinguished pathologist is that he has reduced

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the interactions of toxins and antitoxins, both *intra vitam* and *in vitro*, to principles of molecular chemistry and of chemical physics. He has shown that the interactions in question are subject, both qualitatively and quantitatively, to the known laws that govern chemical action. We are not able, it is true, with our present knowledge, to symbolise the reactions involved in the form of an equation, for we do not know the exact chemical nature of the substances concerned, and, indeed, both toxins and antitoxins have not yet been isolated in a pure condition, but we have evidence that the former, at least, appears to be related to substances closely resembling products formed in the course of ordinary digestion. Consequently it is not improbable that, notwithstanding their poisonous character, they are substances not far removed from these which form the normal food of the body tissues. Antitoxins are believed to belong to a closely allied group.

Ehrlich's theory further refers the chemical actions involved to those cells of the body on which toxins exert their influence; he shows us how, on grounds quite in harmony with known physiological laws, the formation of antitoxins depends on the power, possessed by the molecules of certain susceptible body cells, to seize hold of, and anchor to themselves, the circulating toxins, and in so doing they are stimulated to an increased activity which causes them to liberate into the blood-stream certain substances which are, in fact, the antitoxins, and these, meeting the toxins, combine with them and neutralise their effects in much the same way as an alkali can unite with and neutralise an acid. Thus in the laboratory of the body are produced the same effects as when two chemical antagonists are mixed together in a test tube.

Great as is the importance of this theory from a therapeutic point of view, it has a still wider significance, for it throws light upon one of the central problems of physiology, viz., the nature of the chemical mechanisms of those normal physiological processes by which the living cells of the body regenerate their substance by incorporating the nutritive materials which form their food, and then again by their activities break down into waste material. And the conception has still wider applications, which will be mentioned directly.

We have seen that in diphtheria and tetanus the deleterious influence of their organic generators depends upon the production of toxins which, indeed, may produce their effects quite independently of the actual presence of the germs themselves; but we also know that there is another class of diseases in which the effects are due to the actual bodily presence of the bacteria, and not to toxins which may be obtained from them. Of such a class are typhoid fever, cholera, plague, pneumonia, septicæmia, and other forms of blood poisoning, in which diseases serum therapy is still on its trial, but so far with less certain and successful results than in the case of diphtheria. In immunising an animal against this class of diseases the object is to develop in it, not a serum capable of neutralising the toxic produce, but one capable of destroying the actual living bacterial organisms. This is done by using for immunisation cultures containing the bodies of the bacteria themselves, not merely their separated toxins. To the reactions which take place when these bacterial sera, as they are called, are used, Ehrlich applies a chemico-physiological explanation similar to, but more complicated than, that which he gives for the relatively simpler affair of toxins and antitoxins; but this, again, it is not necessary to discuss.

By pursuing this line of research it has been found possible to produce in animal serums a variety of these antibodies, as they may be called collectively, each of which has some specifically destructive action not only on such organic cells as are represented by bacteria, but upon other kinds of cellular units of complex fluids or organs, such as the red corpuscles of the blood, the cells of the liver, nervous system, and so on; and here can be seen the ground

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for the hope so often expressed, but as yet unfulfilled, that by an application of this principle we may be able to develop a serum which shall have the property of destroying those cells of which a cancer or other malignant growth is composed, while leaving untouched the healthy cells, or, if cancer should be proved to be due to a germ, as some think, we would seek for a serum which will destroy either it or its poison. Koch's tuberculin treatment of consumption, the tragedy of the failure of which we so well remember, was one of the many attempts that have been made to treat this disease on the principles we have been discussing. Nevertheless, now that we know so well that in this and many other complaints we have to deal not with a nameless and unknown something, but with definite and specific organisms, with whose conditions of existence we are perfectly familiar, the hope remains that the defeat in these cases may not be final. Indeed we have good grounds for believing that the organic generator of every disease, once it is known, may be made to produce its own antidote. This surely is a magnificent conception.

Nor even do the results of these investigations stop here, for out of them have arisen new methods, more precise than any others available, for the identification of various bacterial species; means, more delicate than any other test, for detecting the presence of toxins; a means of diagnosis for typhoid fever; a method for the determination, with extraordinary accuracy, of the resemblances and differences between the blood of various species of animals, which is capable of being applied in courts of law for the identification of blood stains. The same methods can be applied also for the purpose of determining the relationships of species and groups of animals by demonstrating the fact that "there has persisted a common property in the blood of various groups through the countless ages that have elapsed since their evolution from a common ancestor, and this in spite of differences of food and habits of life." Lastly, from an exhaustive study of the phenomena of old age, to which an increasing amount of attention has been paid of recent years, the distinguished physiologist, Metchnikoff, whose name is a guarantee that he does not speak as a visionary enthusiast, and who himself has borne a conspicuous part in the investigations that we have just been discussing, has suggested that it may be possible to apply the principles of serum therapy to the arrest of those senile changes which we have been accustomed to believe form the inevitable and distressing concomitants of old age, to the end that we may prolong the usual span of life. Be this as it may, the increasing study devoted to the conditions of old age makes it abundantly clear that many of its painful incidents are of our own making, and, above all others, the deleterious effects of alcoholic excess in bringing about premature senility and decay stand out in malignant relief. In these days, when the questions of serum therapy and immunity loom so large in the foreground of medical science, we may, in the light of recent discoveries, recall the epoch-making discovery of

VACCINATION.

A little more than a hundred years ago, the Englishman, Jenner, recognised what seems at that time to have been common knowledge on the dairy farms of Gloucestershire, that persons who had contracted cow-pox—a mild disease—from their charges did not, as a rule, afterwards contract the virulent disease small-pox, and vaccination is the methodised application of these observations. It rests on the basis that by the introduction of a weakened poison, such as that of cow-pox, which is in effect small-pox modified and mitigated by its passage through the cow, it is possible to establish in the body of man a certain insusceptibility to, or tolerance of, the poison of small-pox, so that the latter disease is either not contracted at all, or, if contracted, it assumes a milder form. Thus vaccination is but a special application of what we know to be a general principle.

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I need not dwell on the fact that by the general adoption of vaccination small-pox, which, from the thirteenth century, ranked as the most destructive of pestilences, has been, if not completely eradicated, at least greatly mitigated in its prevalence and effects wherever communities have been willing to avail themselves of the protection afforded by the practice. On the other hand, we have had frequently manifested the power of mischief that may arise from its neglect, and from the policy of indulgence towards that type of person who calls himself a conscientious objector.

You will see why I have recalled this episode of medical history; it is, of course, because the basic principle of Jenner's discovery has received such ample confirmation by the recent investigations in serum therapy and immunity. But, we may ask, why did vaccination remain more than a century as the single example of an immunisation conferred by this method? The broad idea of establishing artificially an immunity for poisons is itself very old, for, as we learnt in our school days, Mithridates, King of Pontus, in the fear of attempts upon his life by poisons, produced in himself a tolerance of their effects by the habitual and continued taking of them in small doses. The term Mithridatism, still applied to the tolerance of poisons thus produced, perpetuates the name of an early practical exponent of artificial immunisation

We may, I think, safely say that the reason why vaccination remained for so long as the sole example of protective inoculation was because it was an emprical discovery in the sense that no underlying principle was discerned which would admit of its explanation or its extension to other cases. The fact of its protective influence was known, and, indeed, was so obvious that the practice of vaccination was adopted by most civilised communities, though, let us regretfully admit, with greater thoroughness by others than by the countrymen of the great Englishman who devised it; but no rational explanation of the cause of the fact was forthcoming, and this is just what Ehrlich's theory attempts to supply. Even if, as is very probable, it will ultimately be found out not to be correct in all its details, it may safely be said that its fundamental principles will never die, and nothing can minimise the stimulating influence of this very remarkable conception that has radiated in so many diverse directions. It is, then, not a little remarkable that, in these days when the principle of Jenner's discovery has been so amply vindicated and extended, and when some of its undoubted original dangers have been removed by better methods of application, that we witness a marked tendency to neglect the protection which the world's experience has shown it so conspicuously affords.

With this wide, and ever widening, scope of the principles involved in the recent theories of immunity, it is no wonder that they occupy so prominent a place in modern medicine. Nor, as we have seen, is their application limited to the processes of disordered function with which medical science has to deal, but it extends towards a better understanding of the normal physiological mechanisms of all living tissues. They illustrate the conception that is being so surely borne in upon us that medical science, like biology, of which indeed it is but a branch, requires for its elucidation the convergence upon it and application to it of fundamental physical and chemical conceptions. No better example could be given of the value to science of a well-ordered imagination and of a good working hypothesis, or of how investigations in one field may open up unexpected discoveries in many and unexpected directions. The dreams of to-day become the realities of to-morrow, and in these theories we have a germinating idea which is destined to grow into a tree of knowledge whose manifold branches will bear abundant fruit.

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THE ANIMAL KINGDOM IN RELATION TO DISEASE.

So large is the role played in disease by those minute vegetable organisms that we collectively call bacterial germs, that we are apt to forget that the animal kingdom also provides its contingent of deadly enemies to the human body. Putting aside those grosser forms of parasitism such as hydatids, tapeworms, and the like, which are not as a rule fatal, it is now well known that in another class of diseases—infectious but not contagious—such as the various forms of malaria, yellow fever, and others, the exciting cause is the presence in the body of minute organisms which belong to the lowest class of animals; and, further, that the transmitters to man of these low organisms are to be sought amongst other and higher orders of animals. In the diseases particularly mentioned it is now well established that mosquitos of certain sorts are both the agents in the transference as well as the necessary hosts in which a part of the parasite's life's cycle of development takes place. Thus the problem of the abolition of these diseases resolves itself into a question of the destruction of the breeding-places of mosquitos, which task, apparently at first sight so impracticable, has met with a gratifying success wherever it has been intelligently and persistently undertaken. That terrible complaint, sleeping sickness, which devastates a large belt of tropical Africa, is similarly caused by a minute animal parasite transmitted to man by a Tsetze fly; sleeping sickness is, in fact, the human form of that deadly complaint known as Nagana or Tsetze fly disease, which renders large parts of South Africa uninhabitable for the introduced domestic animals. Other forms of the same fatal animal disease are those known in India as Surra, and in North Africa as Dourine. The agency of animals in respect to the causation of infectious disease is also shown in plague, where there is good reason to believe that rats, and perhaps mice and fleas, act as carriers to man. We strongly suspect house-flies to be agents whereby typhoid fever, cholera, and forms of blood poisoning may be distributed; anthrax and glanders come to us from the horse, sheep, and ox; our common tapeworms through the pig and bovine cattle; and the familiar friend of man—the dog—is, as we have long known, practically the sole source of hydrophobia and hydatids. From the knowledge of these and many other similar facts it has followed, as part of our recent views, that medical science refuses more and more to be bound by the rigid limits formerly prescribed for it, viz., that the human body and its diseases are to be regarded as isolated factors to be considered apart from their organic environment. It has been compelled to recognise, what the zoologist has long recognised, that man is only a unit in the world of living things, inter-related not only in structure but in disease with his animal and vegetable surroundings. While I speak of this subject, perhaps it is not out of place if I utter a word of serious warning in respect to the danger that now confronts Australia from the ease with which some of these fatal diseases of stock, from which we have hitherto been free, may be introduced into this country. Occurring all around us, in South America, Africa, Mauritius, India, Manila, and New Guinea they are, with the frequent and increasing means of communication, knocking, so to speak, at our very doors. And if ever they, or any of them, should, through carelessness or misadventure gain a footing in this land, you may depend upon it that the result will be a destruction of our flocks and herds that is inconceivable to those unfamiliar with their deadly effects in other countries. Ask of Africa what it has lost from nagana, dourine, rinderpest and horse-sickness; India, Mauritius, and the Philippines from surra; South America from mal de caderas; and from the answers let Australia take warning before it is too late. A few hundreds a year spent now, with intelligent and prudent foresight, in securing the services of some competent scientific expert, who has had large and practical

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experience of the highly complex and difficult problems which attend the investigation and management of this class of pestilence, might be the means of saving millions of money and untold trouble in the future.

All these discoveries, then, which I have mentioned, with their extensive ramifications, constitute great and important advances not only in the wealth of added facts but also, in what is still more important, the widening of our ideas concerning the nature of living activities and those departures from the normal we call disease. Already they have profoundly influenced our medical and surgical theory and practice, while they are full of hope for the future. Together they have led to the establishment of what is practically a new science—that of preventive medicine, which in its turn has led to the virtual suppression of some diseases and the mitigation of the effects of others. Were it not for ignorance, apathy, and that self-complacency which is so essentially a characteristic of our own British race, the results would be still more marked. Indeed success in this direction has led a cynic to observe that medicine has made prodigious progress in all things except in the treatment of disease, and there is some truth in the sarcasm; but if we are not much better able to cure diseases in the full sense of the term than were our forefathers, we are certainly much better able to prevent them, and we may regard this as the higher and better function.

RATIONAL THERAPEUTICS.

We have, however, also learnt that there are other and wiser methods of treatment than by a multitudinous, and often haphazard, polypharmacy for we have a much deeper knowledge, to which the recent investigations into the conditions of immunity have largely contributed, of how great, how manifold, and how effective are the natural recuperative powers of the healthy body which were long ago recognised in the Hippocratic aphorism *vis medicatrix naturæ*. We have discovered, or at all events we better appreciate, the facts that fresh air, abundant sunlight, change of scene and climate, and more suitable dietaries are in themselves potent therapeutic agents, and not the least important discovery is the realisation that the doctor's best function often is to be simply the *homo minister naturæ*. We have successfully applied the actinic rays of light to obstinate and disfiguring complaints of the skin, and we have invoked, with as yet uncertain success, as therapeutic agents, some of those mysterious and potent electrical and radioactive emanations placed at our disposal by the extraordinary developments of physical science, which bid fair to exhaust the Greek alphabet in their provisional nomenclature. By the researches of the synthetic chemist we are able to use medicaments of absolute purity and known chemical composition, instead of crude drugs of varying and uncertain contents; while a more extended knowledge of molecular structure has directed attention to a possible connection between chemical constitution and physiological action which must eventually lead to a more accurate and scientific therapeusis.

That comparatively recent development of physico-chemical science known as the ionic dissociation theory, which gives us a rational explanation, up to a certain point, of such intricate living actions as the contraction of ordinary muscles and of the rhythmic and ceaseless beat of the heart, the transudations of physiological fluids, and has even been applied to the explanation of that mystery of mysteries the fertilisation of the egg cell, is similarly applicable to the chemical mechanisms involved in the action of drugs, germicides, and disinfectants, as well as to many of the normal activities of the living body. Again we come back to the fact that the more we learn of living actions the more do we find that they may be referred to the same laws of physics or chemistry which govern the relations of inert matter.

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MEDICAL SCIENCE IN RELATION TO THE COMMUNITY.

But while we may contemplate with much satisfaction these great advances in medical science, let us not forget that the ledger of national life has its other side, which is not so flattering to our pride of progress. The gaunt spectre cancer, with its allied malignant maladies—the *opprobrium medicinæ*—the dread of which is an added terror to the frailties of old age, still stalks the land claiming in thousands its victims of unutterable pain and misery. In spite of all our boasted progress, we have not even learned their cause; we do not know the remedy, save in so far that all experience tells us, more and more, that early and complete removal—and the earlier the better—offers a good hope of recovery. To this let all take heed, and they may take the comfort they may so sorely need. On this problem of malignant disease is now being concentrated, in an organised system, some of the best minds of the medical profession throughout the civilised world, and it may well be hoped, nay, it may be expected, that its solution will not long be delayed, to the glory of medicine and the infinite relief of human suffering.

The great white plague, consumption, in spite of our knowledge of its cause and of the conditions which will bring about its prevention and cure, still slays its myriads, far exceeding those of battle and all other pestilences combined, in the most interesting, joyous, and useful epochs of their lives; but it may also be said that if tuberculosis is still propagated it is not because of the failure of science, but because of ignorance, apathy, wilful neglect, or, it may be, genuine poverty on the part of those who still continue habits and practices which are responsible for its prevalence and spread.

We go into hysterics at the thought of the advent amongst us of some rare visitant disease such as bubonic plague or small-pox or leprosy, but we complacently tolerate the continuance in our midst of typhoid fever and diphtheria, which are perfectly preventable, and whose tale of victims is far greater than that of the former. We fail to have our children vaccinated, or to be ourselves revaccinated, by which means we might render the community secure against a severe small-pox epidemic, while our governments, with all their love of compulsory measures, tolerate the neglect, preferring rather, or at least finding it sometimes necessary, to spend thousands in the effort to eradicate what might be prevented.

We see unmistakeable evidence of the physical deterioration of the race due, no doubt, to many co-operative factors into which I may not enter except to say that one contributory cause is the tendency for people to flock to the towns, where the mothers of the future pass the most important period of their lives—that of early adolescence—under the unfavorable or even injurious conditions of crowded and ill-ventilated working rooms. The cry "back to the land" may have a wider and a deeper significance than the mere desire to fill up the blank spaces on the map with profitable industries. It is rare to see even a child with a sound set of teeth, a defect most frequently due to their neglect, from which arises many an ailment, both then and afterwards, and there is much truth in the observation that toothpowder may serve as a better national protection than gunpowder.

It cannot also be doubted that the progress of medical science has been a contributory factor in this degeneracy, by bringing about the saving and the prolongation of the lives of those who are physically or mentally unfitted to be the parents of a healthy race. This brings us within sight of the eternal disharmony between the tendency of the work of our social, political, and ethical systems, of our hospitals, asylums, and the like, which are directed towards a similar end, and the efforts of unaided nature "red in tooth and claw," which, by the elimination of the unfit, strives with unceasing purpose only for the improvement of the race and the survival and perpetuation of

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its best. And if time permitted it would be an interesting study to see what an important part has been played by disease in the evolution of the human species, and how civilised man, by his migrations, his diseases, and his meddling, has disturbed existing harmonies, and altered, often for the worse, the organic face of the earth. And that the degeneration of which I have spoken is not merely physical, but extends also to the mental organisation, is shown by the striking and progressive increase both in the number of the actually insane and in the increase of those nervous complaints of which neurasthenia and the like may be taken as the type; this phenomenon being apparently characteristic of all highly civilised communities, and for which the mental strains and stresses of modern life and intemperance are held to be chiefly responsible.

Co-relative with the question of physical deterioration is the ugly fact, for some time suspected and recently confirmed by the report of a Royal Commission in a neighboring State, that the decline in the birth-rate, which we have been accustomed to think of as a feature only of those older countries which have had to face the problems of over-population, has extended to these sparsely peopled States, and that all classes are affected, even those who have not the burden of want to offer as an excuse. Thus one of the conspicuous signs of national decadence is beginning to affect us while we are still a nation in the making. And the causes at work, clear and distinct, and for the most part disreputable, bring with them the uncomfortable idea of a concomitant general loosening of moral fibre which will inevitably radiate its malign influence in other directions. Here, at least, is a field in which that democratic panacea, compulsory enactment, is powerless.

Closely connected, in respect to cause, with the question of physical degeneration is the excessive mortality of infant life, for which, as in the former case, inadequate and improper feeding are more largely responsible than any other single cause, and this, when not due to genuine poverty, is attributable to the lamentable ignorance on the part of those who are mothers, which our vaunted system of education does little to remove. If we are powerless to counteract those evil and other influences which underly the reduction of the birth-rate, it ought, at least, not to be an insuperable problem, and indeed it is our obvious duty to find means for preserving those we have.

The hydra-headed monster, alcoholic excess, still dwells with us, bringing in his train poverty, crime, endless social and domestic misery, and physical and mental suffering, predisposing to disease, and second only to another cause, also begotten of human frailty, in bringing about those degenerative changes which lead to senile decay and premature old age. The solution of the alcohol question would be the solution of many an ill to which weak flesh has made itself the heir.

We still show little wisdom in our dietaries, either in quantity or quality; we still continue to "dig our graves with our teeth," and to lay the foundations of disease, amongst which may be mentioned the prevalent complaint appendicitis. In this respect we in Australia should pay more heed to the evils of our excessively carnivorous diet, to which many of our ills may be traced.

Reviewing these and other physical disabilities which it is the lot of man to suffer and endure, it must in fairness be admitted that the responsibility for the existence and continuance of most of them cannot be laid at the door of medical science. Where this has been able to intervene it has done so with some success, as witness the results of preventive medicine. If we except from the category cancer and some other diseases in which we must regretfully confess the present impotence of our profession, the majority of these physical ills have now, for the most part, largely become social questions,

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and their solution when the doctor, speaking in the name of science, has pointed out the cause and cure, lies in the individual and collective efforts of the people.

If, owing to ignorance, apathy, or self-indulgence, they still continue to exist, it is the community, not the doctor, who is responsible. And it would be better if society, instead of blaming science, as it frequently does, that it cannot find ways of curing disease, set its house in order, and, by abstaining from hurtful ways, automatically abolished a vast amount of suffering.

Sometimes the advice and precepts of science are wilfully disregarded, as in the case of vaccination, where the effective remedy is deliberately thrust aside, with the approval, or even the instigation, of politicians, who care more for the votes than for the bodies of their constituents.

Then think of the enormous sum of human improvement, physical, mental, and moral, that would accrue to a great mass of the people if only that one factor in the predisposition to and causation of disease, intemperance, were eliminated from their lives by deliberate voluntary restraint, for I have no belief whatever in the permanent efficacy or benefit of those prohibitive measures which are so often advocated as the only possible panacea. It is stated that two-thirds of the immense national drink bill is incurred by the working classes; how surely, then, would the diversion into healthier channels of money saved in this direction bring an automatic relief to those who are most concerned—a relief from an untold amount of grinding poverty, sickness, and mental and moral distress. And to a degree much greater than is realised, how very much longer and healthier lives we should lead if we refrained from eating so much, so often, and so unwisely. What might be done towards the relief, or even the abolition, of that scourge of home life, consumption, if only people would cease from doing those things which favor its propagation and continuance, and would do those which, they can be told in all honesty, will, with certainty, prevent its spread and bring about its cure. But how shall we blame the general public for their errors of commission and omission when those who cannot claim the excuse of ignorance, in defiance of risks that are not so imaginary as they seem to think, persist in continuing the reprehensible and insanitary practice of the promiscuous transference from mouth to mouth of an uncleansed communion cup. So, too, any betterment in respect to the unsatisfactory and unwholesome conditions which prevail in connection with physical deterioration, decline of birth rate, infant mortality, and the like is only to be expected from a rude awakening of the public conscience, and the adoption of a sane and rational system of training in those hygienic and physiologic matters which, though of such great importance for the sound mind and the sound body, yet are so inadequately treated, or even entirely neglected, in our systems of education.

The suppression of malaria and yellow fever has become, as we have seen, a question of the destruction of the right sorts of mosquitos; plague, largely, of the extermination of rats and of the filth which usually invites their presence; cholera and typhoid, of pure water and uncontaminated food; hydrophobia was speedily eradicated in Great Britain by the much-abused order for muzzling dogs; and in a hundred different ways the fact is obvious that what were once considered medical questions have now, to a large extent, resolved themselves into social problems.

REVIVAL OF QUACKERY AND OCCULTISM: INFLUENCE OF THE MIND ON THE BODY.

Whether it is to be considered as a social failing or only an amiable foible, it is nevertheless true that the boundless credulity of mankind upon subjects connected with medicine has, from earliest times, been a conspicuous

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feature in his life, and it is not a little remarkable that an age which has witnessed so great a growth of scientific precision in our medical methods and conceptions should be also characterised by a revival of superstition, a belief in occultism and quackery of all descriptions, that would be worthy of mediæval times. It is particularly remarkable that these beliefs and the habits and practices begotten of them are not confined to the ignorant, but prevail notoriously amongst those who belong to the educated classes. We see the columns of the press filled with the announcements of clairvoyants, professors of second sight, palmists, herbalists of various nationalities, and the more outlandish the more sought after; hypnotists, crystal gazers, faith healers, and a whole tribe of a similar sort, many of the designations being merely cloaks for practices much worse than is indicated by their names. We are told that imperfect noses can be corrected by absolutely painless methods; that double chins, drooping cheeks, and wrinkles can be removed; and that ruptures can be cured without operation. There are infallible remedies for cancer, consumption, obesity, baldness, alcoholism, narcomania, and numerous other diseases. Columns are filled with the marvellous and unfailing virtues of somebody's pills, potions, syrups, and other nostrums—the same article professing to cure a whole group of the most diverse complaints; while flamboyant posters portray the features of the eminent specialists who deal in them. There is an abiding faith, chiefly among the more educated, in various routine "systems" of treatment, as they are called, which pass under the names of their exploiters. And have we not seen in recent times worthy and otherwise blameless citizens pinning their faith to the efficacy of an iron ring as a cure for rheumatism?

One of the most recent developments of this revival of superstition is the pseudo-religious movement which passes under the name of Christian Science, with the Rev. Mary Baker Eddy as the chief exponent and apostle. As an example of the fatuous twaddle which passes for Mrs. Eddy's gospel, let me give you the following extract, which is part of an invocation for the cure of cancer of the stomach, one of the most real, most objective, and most distressing of complaints.

"Lord, help us to believe that all Evil is Utterly Unreal; that it is silly to be sick, absurd to be ailing, wicked to be wailing, atheism and denial of God to say I'm sick. Help us to stoutly affirm with our hand in your Hand, with our Eyes fixed upon Thee, that we have no Dyspepsia, that we never had Dyspepsia, that we will never have Dyspepsia, that there is no such thing, that there never was any such thing, there never will be any such thing.—Amen."

Surely no mumbo-jumbo of Haitian Obi-man could be more ridiculously inept, and yet this is the sort of thing that has gained the concurrence and approval of many educated people. Nevertheless, however repellant we may find the methods and practices of these charlatans, and however silly we may think them, in an age which professes to be one of reason and enlightenment, it is very evident that they must find their trade profitable, for they continually increase in number, in the voluminousness of their announcements, and, we may add, in the impudence of their pretensions.

True of all times and of all sorts and conditions of men and women, and never more true than in this connection, the Hudibrastic distich—

> "Doubtless the pleasure is as great
> Of being cheated as to cheat,"

embodies a profound knowledge of human nature, and condenses a practical wisdom which explains the facile success of many a rogue and charlatan with their confiding dupes.

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To our sorrow and discomfiture it must, however, be admitted that we doctors have all of us, at some time or another, met with cases in which so-and-so's patent pills have cured our patient when precisely the same ingredients (for the composition of all of these remedies is no secret) have failed, and do we not know, all of us at least who have served our time in a large hospital, the marvellous efficacy of the local brew of *mistura diabolica* concocted with quite other objects in view than that of scientific medication. There can be no doubt of the reason of many of these successes, which is, in fact, the principal foundation on which all kinds of quackery is based. It is that the confident assertions and brazen effrontery of the purveyor of the patent medicine has produced an expectation of, and a profound belief in, its efficacy, which contributory factors often fail to accompany the orthodox prescription. This statement brings us within sight of the great question of the influence of the mind on the body, a factor curiously enough more recognised by the laity than by the medical profesion, not only as manifested in one and the same person, but also as between two different individuals. The potency of this influence everybody freely admits, but few of us actually take it into serious consideration in our practice. We were never taught anything about it in our medical course, nor do we teach it now, and but little attempt has been made to organise and systematise our scattered but yet considerable mass of knowledge of the subject. Yet there is no doubt but that the power to utilise, consciously or unconsciously, this mental factor in medicine is the real secret of the success of many a physician who, except in this respect, has no better medical equipment than his less successful compeers. Though we ridicule the quality, yet really we all envy the possession of a good bedside manner, which is only one mode of expression for a pleasant and reassuring comportment, and it is just faculties belonging to the same category which account for much of the success of the advertising fraternity. "Two mighty powers," says the writer of a recent book, "work for good in every physician worthy of the name: what he knows and what he is; but, alas, as a rule, we only value the former." This well suggests the too often neglected powers that may lie behind the personality of the physician.

Gradually we are learning that the abundant faith which inspires to noble deeds and to the enduring of great suffering is a form of mental energy which may be diverted into the swelling therapeutic stream; but it is a pity that the chief exploitation of this potent force should have so chiefly fallen into the hands of unscrupulous impostors and charlatans ignorant of all things except the illimitable credulity of human nature.

Here, then, is a wide field of investigation for the physician of the future from which a rich harvest may be expected.

CONCLUSION.

It has been my endeavor in this address, in which you may well say that I have wandered from the Dan to Beersheba of medical and social science, to present to those who do not belong to our profession some account of its aims and methods; of its triumphs over physical ills, of its hopes and aspirations for the future, and of the vivifying influence of some principles of science which have so greatly contributed to its advance. I have endeavored to show that man must be considered only as a unit in the world of living things closely inter-related by his diseases with his animal and vegetable surroundings, and that medical knowledge, consequently, stands not apart from but part of the great body of organised science; indeed, more and more is it borne in upon us that not only every science, but also every system of truth, is intimately connected with every other. From these facts it arises that the investigations of an ever-increasing number of diseases have virtually

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resolved themselves into problems of botany, zoology, or of physical chemistry. We see how wide has become the scope of preventive medicine, and by a slight stretch of the imagination we may almost foresee a hygienic millenium in which all diseases shall be abolished, leaving only accidents and the inevitable incidents of old age. These latter we are, on the one hand, assured by one of the most eminent of living pathologists might, by appropriate treatment and a better living, be postponed to a period considerably beyond that at which we now accept them with what resignation we can command; while, on the other hand, to those of us who have passed the meridian of days, it comes, as an uncomfortable suggestion, from a man of science of equal eminence in another branch of medicine, that an anæsthetic euthanasia is the most fitting treatment for the sexagenarian. For my part I prefer to pin my faith to the optimistic view rather than to the homicidal treatment, and I fancy there are many similarly conditioned, in point of years, who will have the same preference.

Then it has been indicated, very perfunctorily, as I am too well aware, that many of the medical questions relative to disease have passed out of the domain of medicine and entered that of social and political science, in which, again, they are dependent upon individual, collective, and organised efforts scientifically directed and persistently carried out. Had I not outraged your patient indulgence by the length of my remarks it would have been interesting and possibly profitable to examine the extent to which these efforts have been successful, or, alas, too often inefficient or even unattempted.

Science has, I think, shown by its achievements and by its energies that it may be trusted to do its share in the world's work; but how shall we, in an age distinguished by its shams, shoddy, and superficialities, inculcate and foster that spirit of lofty patriotism, self-reliance, self-restraint, supreme devotion to duty, and unceasing striving for excellence in all things undertaken which are the real foundations on which a nation's greatness rests?

It is well that we should realise, before it is too late, that a nation's preeminence depends not alone on the extent to which its collective intelligence is organised and co-ordinated along the many diverse paths that lead to success, but also on the high sense of duty and self-sacrifice of its citizens. And the doctor may perhaps be forgiven if, in his capacity of a social unit of the community, he ventures to say that it is impossible to view without anxiety the prevailing tendency of the day towards the pursuit of pleasure and the decline of high enthusiasms and beliefs in ideals. In this we may see the sacrifice of many things that have made our country great, and the presence of many that have contributed to the downfall of great nations in the past. It is said of us with much truth that we are apt to be in earnest about trifles and to trifle about earnest things, and this in days when, with unexpected suddenness, our national patrimony—nay our very existence as a great nation—may become the stake for which we shall contend with others less self-complacent than ourselves.

Many lessons have been taught to a wondering and startled world by the great Manchurian drama, but they may all be summed up in the one singularly happy and comprehensive appreciation, which we who deal with the lives of men may well take to heart—"Nothing but the best will do." This after all is but the most modern variant of that venerable exhortation of the Preacher applicable to all men and for all time—

> "Whatsoever thy hand findeth to do, do it with thy might."

Dr. John Thomson (Brisbane): The task imposed upon me can best be discharged by brevity. We came to-night to listen to the inaugural address by our distinguished President. We came expecting an intellectual

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treat, and I can assure you, speaking for my own part, and speaking for all of you, that our expectations have been realised. (Loud and prolonged cheering.) In this feast he has put before us many dishes of the very highest quality, to provide food for our mental digestion and assimilation. The address has been worthy of the man—the man the medical profession throughout Australasia unanimously selected to preside over us, the man who can put after his name the mystic letters F.R.S., which but few other Australian men can do—a distinction which England bestows upon only her most scientific sons. (Cheers.) The address was worthy of the profession to which it was delivered, a profession which the brilliant preacher yesterday declared to be one of the learned professions, and associated it with law and divinity. He clearly hinted that law and divinity had been left behind, and that medicine had made marvellous strides. If, therefore, it was learned in the past, it is still more learned now. The address was equally worthy of the men in this State of yours, including Dr. Poulton, who, in 1887, to commemorate the Jubilee of Queen Victoria, inaugurated this Congress. It went through all the colonies, and came to Brisbane; and it was at Brisbane that His Majesty's representative here to-night was initiated into the mysteries of the Congress. It has come back here, and it gives us great pleasure to be here amongst you. May I ask your Excellency to put a vote of thanks to our President for his address this evening?

His Excellency: As Dr. Thomson has said, it hardly needs words from anyone else to ask you to give a unanimous vote of thanks to our distinguished President. I think I may, in the name of you all, not only those who are present, but of all who will have the pleasure of reading it and hearing of it, present to him a unanimous and hearty vote of thanks.

The vote of thanks was carried with the audience standing.

The President bowed his acknowledgments.

The Hon. G. H. Butler (Hobart): It is my pleasing duty to-night to propose a vote of thanks to His Excellency the Governor, who has shown a keen interest in our deliberations and discussions. The fact of one in his high position and attainments showing such an interest in our Congress must be an incentive to all of us. There is no doubt that one advantage of this Congress is that we learn so much from each other when we meet. I also, on behalf of the visitors, thank His Excellency for the cordial welcome he has extended to us on our visit to Adelaide.

The motion was carried with acclamation.

The proceedings closed with the National Anthem on the Conservatorium organ by Professor Ennis, Mus. Doc., Director of the Elder Conservatorium.

FINAL GENERAL MEETING.

The final general meeting of Congress was held at the Prince of Wales Theatre, University Buildings, on the morning of Saturday, September 9th The President (Professor Stirling) occupied the chair.

The President: In the first place I have to read you a resolution from the Synod of the Church of England. It is as follows:—

"Church Office, Adelaide, September 6th, 1905.

"The President of the Medical Congress, Adelaide.

"Dear Sir—I beg to forward hereunder a copy of a resolution passed unanimously by the Synod of the Church of England at its annual meeting yesterday.

"I am, dear Sir, yours faithfully,

"Charles Marryat, President of Synod.

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"THE RESOLUTION.

"This Synod, recognising the dignity and greatness of the medical profession, its unique opportunities, its immense responsibilities, together with its countless deeds of unknown and unacknowledged kindness to the sick and needy, tenders its respectful and hearty greeting to the Australasian Medical Congress, assembled for the second time in the city of Adelaide. The Synod trusts that, by the blessing of God, the deliberations of the Congress will minister to the progress of advancing science and consequent amelioration of some of the world's distresses. It also hopes that the visit to Adelaide of members of the Congress from other States will be one of pleasure as well as profit to themselves."

We shall take means a little later on in this meeting to send a suitable reply to this message. The next business is dealing with the resolutions from the various sections of Congress. Before I deal with them specifically I may be allowed to make the following remarks. It is no doubt a difficulty that we experience at these meetings that we have no standing orders under which to work, and no rules of debate. That is a difficulty which has occurred to us, and a little later will follow a resolution which will seek to remove it. Seeing that we are obliged to conduct this meeting without guidance or rules of debate, perhaps you will allow me to lay down the following rules, which I hope you will not consider too draconian. We are all anxious to give due consideration to the business of the resolutions, and it will be understood that as the various resolutions have been considered by the sections in which they have arisen, we may reasonably anticipate that full discussion has taken place on them. I therefore say that no speaker shall speak for more than five minutes and that at any time, on its being proposed and seconded—"That the motion be now put," the motion shall be put without discussion. (Loud cheers.) That will enable us to see whether we want to decide one way or another. May I take it by those cheers that you agree? (Cries of "Aye.") The first resolutions are from the Section of Medicine.

SECTION OF MEDICINE.

DR. JARVIE HOOD (Sydney): I have much pleasure in moving—"That this section is of opinion, judging from the complex and sometimes dangerous symptoms produced by X Rays, Radium, and Finsen light, that the use of these agents should be in the hands only of members of the medical profession. That this resolution should be forwarded to the general meeting of Congress for its consideration." We discussed it thoroughly in the Section of Medicine, and there is no need to discuss it further now.

DR. MURRAY (Melbourne): I second that.

DR. LOVE (Brisbane): I move an amendment to add—"Or under their direct supervision." Since that resolution was adopted by the Section of Medicine a conversation has taken place, and we believe that the amendment will overcome a difficulty that presents itself in the original resolution.

DR. MURRAY (Melbourne): I hardly think it is an amendment. Dr. Hood and I are agreeable to accept that the words be inserted.

The addition was agreed to, and the motion as amended carried.

SECTION OF PUBLIC HEALTH.

THE PRESIDENT: The next is a series of resolutions from the Public Health Section, but I notice that they do not come to us with a recommendation to be forwarded to the General Congress.

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RESOLUTIONS.

1. "That in view of the profound influence of school life and school training upon the physical future of the race, the hygienic construction and management of schools, the training of teachers in school hygiene, and the systematic practical instruction of school children in elementary personal, domestic, and civic hygiene are subjects which demand the earnest attention of the Governments of the various States of the Commonwealth."
2. "That for each State Education Department there should be available a whole-time medical officer, possessing special knowledge of applied hygiene in relation to schools, to whom should be referred all questions relating to the hygienic construction and management of State schools, to the protection of teachers and pupils from preventable disease, and to the methods and scope of hygienic instruction and training."
3. "That applied school hygiene should form a compulsory subject in the training of State teachers, and that a system of medical inspection of schools should be introduced at an early date, particularly in the larger centres of population."
4. "That the compulsory notification of phthisis is advisable in the interests of public health."
5. "That in view of the large number of deaths from phthisis in Australasia, this Congress urges upon the Governments of the various States the necessity of establishing and maintaining sanatoria for the treatment of cases among the poor."
6. "That it is necessary to provide a fund for assisting families among the poorer classes where the breadwinner is suffering from phthisis, and has entered a sanatorium for treatment, and this Congress urges upon the Governments of the various States the necessity of taking steps for providing such a fund."
7. "This Congress protests against any action which has the effect of interfering with a bi-daily delivery of milk in Australian cities, and considers any such action as seriously prejudicial to the public health."
8. "That it be a recommendation to the next Congress that special consideration be given to the attaining of uniformity in the compilation of vital statistics in the Australian capitals."

Dr. Springthorpe (Melbourne): I think the error mentioned by the President is one made by the printer. I know my resolutions were for recommendation to Congress. I move to add—"That these resolutions be forwarded to the general meeting of Congress for its consideration."

Dr. Elkington (Hobart): I second it.

Carried.

Dr. Armstrong (Sydney): I propose the adoption of the resolutions by Congress.

Dr. Worrall (Sydney): I second that.

Carried.

Section of State Medicine and Medical Ethics.

Dr. A. A. Hamilton (Adelaide): I beg to move to add to these resolutions—"That these resolutions be sent on for adoption by Congress."

Dr. Gibson (Brisbane): I second it.

Carried.

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Dr. Rennie (Sydney) moved—"That a committee be appointed, consisting of representatives from each State, with power to add to their number, to consider the best means to be adopted to amalgamate the various medical organisations throughout Australasia and New Zealand; two representatives to be elected by the local branches of the British Medical Association in each State, by the Victorian Medical Society, and in Tasmania by the Medical Section of the Royal Society, and by each Medical Defence Association in Australasia. That the Secretary of the Section of State Medicine and Medical Ethics should communicate the above resolution to the various bodies, with a request to send him their nominations."

Dr. Marten (Adelaide): I second it.

Dr. Rennie (Sydney): I should like to point out that there was an addition to the resolution—that the committee report to the next meeting of Congress.

The President: Perhaps we can have that as a separate resolution.

The motion was carried.

Dr. Rennie (Sydney): I move—"That the committee appointed by the foregoing resolution bring up a report to the next meeting of Congress."

Dr. Sprott (Hobart): I second that.

Carried.

Dr. Springthorpe (Melbourne) moved—

1. "That in the opinion of this Congress the future provision for the care and treatment of the insane in Australasia should be based upon a recognition of the threefold requirements of (*a*) up-to-date mental hospitals, (*b*) farm and industrial colonies, and (*c*) benevolent asylums."
2. "That in the opinion of this Congress there is imperative need for the establishment in the different States of epileptic colonies, in which epileptic children should receive education, epileptic adults be suitably employed, and epileptics generally find a home—as well as the most successful treatment."

There is no machinery by which these resolutions shall reach the persons to whom they ought to go. There should be a general instruction to the General Secretary that they should be forwarded to the proper authorities. They should go to all the State Governments. I move to add—"That the General Secretary take the necessary steps to forward these resolutions to the proper authorities."

Dr. Howard (Melbourne): I second the resolutions with the addition.

Carried.

Dr. Rennie (Sydney): I beg to move—"That it is unethical for medical practitioners to grant interviews to representatives of the lay press on medical matters, in connection with which their names are allowed to appear. That a copy of this resolution be forwarded to the lay press."

Dr. Springthorpe (Melbourne): I second it.

Carried.

Dr. Springphorte (Melbourne): There is one by Dr. Syme, which is accidentally omitted.

Dr. Syme (Melbourne): There was no resolution afterwards passed that it should be presented to Congress.

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Naval and Military Medical Officers.

Surgeon-General Williams: I move the adoption of the following resolution:—"That in the opinion of this meeting it is desirable to hold a Naval and Military Section at future Australasian Medical Congresses. That this resolution be forwarded to the general meeting of Congress for its consideration."

Dr. Giblin (Hobart): I second that.

Carried.

The Eye Section.

Dr. Lockhart Gibson (Brisbane): There was a resolution passed unanimously by the Eye Section, but there was no decision that it should be forwarded to the Congress to pass it. It was proposed by Dr. Kent Hughes and seconded by Dr. J. W. Barrett. It was as follows:—"That permanent blindness from lead-poisoning amongst young children in Queensland has been recognised; that it is highly probable that paint used on verandah railings, &c., especially when exposed to weather influences, is the source of the poison; and that this matter should be brought under the notice of the proper authorities." I beg to move that that resolution be reported to Congress.

Dr. Kent Hughes (Melbourne): I second that it be reported.

Carried.

Standing Orders.

Dr. W. T. Hayward (Adelaide): I have to move—"That it be a recommendation to the Executive Committee of the next Congress to draw up Standing Orders for the conduct of debates and other business of Congressional meetings, and that the same be reported to the first general meeting of the next Congress." I do not think I need say anything about it.

The President: We are only asking that something may be done to remove the difficulty I have mentioned.

Dr. Astles (Perth): I second it.

Carried.

Next Meeting of Congress.

Dr. Jamieson (Melbourne): I suppose, Mr. President, that it is because I am a member of that diminishing band whom you mentioned in your address as Nestors of the Congress that I have had this resolution put in my hands. I suppose we have to confess that it is greatly on account of the parsimony of the Commonwealth Parliament in refusing Western Australia its rights to a direct railway that we are unable to visit that State at present. I think it almost follows as a matter of course, then, that, since Melbourne succeeded Adelaide on the last occasion, it ought to succeed it on this. As those of us who were present at that first meeting of Congress carried away very pleasant recollections of that meeting, we tried when the Congress came to us in Melbourne to make the meeting a pleasant and successful one, and we did so. (Cheers.) We had nothing but pleasant recollections of that first meeting, and we shall go away again with grateful remembrances of the unparalleled hospitality that has been extended us on this present occasion. (Loud and prolonged cheering.) Melbourne is not now what it was in the days of the boom, but still if we cannot altogether imitate the almost regal hospitality we have received on this occasion, in our humble way we will try to do what we can I have to propose therefore—"That the next meeting of Congress be held in Melbourne—provisionally—in 1908." It will be left to the local authorities to fix the date. I have pleasure in inviting the Congress to Melbourne.

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DR. SYME (Melbourne): I have very much pleasure in seconding the resolution. I can only repeat what Dr. Jamieson has said—that Melbourne feels so overwhelmed by the hospitality and kindness received in Adelaide on this occasion that we feel it is a very difficult matter to follow in the same regal way. We can only do our best, and we extend to you a very cordial invitation to come to Melbourne next time. (Cheers.)

Carried.

THE NEW PRESIDENT.

DR. RENNIE (Sydney): I have much pleasure in moving—"That Professor Allen, Professor of Pathology and Anatomy at the University of Melbourne, be the new President of Congress."

DR. LOCKHART GIBSON (Brisbane): I second it with much pleasure.

Carried.

VOTES OF THANKS.

THE HON. G. H. BUTLER (Hobart): I have much pleasure in moving—"That a hearty vote of thanks be accorded to His Excellency the Governor for his patronage, the very keen interest he has taken in our proceedings, and also for his most royal hospitality since we have been here."

THE PRESIDENT: No seconder is required for these resolutions.

Carried.

DR. WILTON LOVE (Brisbane): I move—"That a vote of thanks be passed to Sir Samuel Way, Lieutenant-Governor of the State, for his patronage in the first place, for his liberal support and the interest he has taken in the Congress, and lastly for his hospitality." Those who were at the conversazione last evenin will carry away pleasant recollections which will last a very long time.

Carried.

DR. J. C. VERCO (Adelaide): I propose—"That a vote of thanks be passed to the Government of the State, the Premier, and the Acting Deputy Postmaster-General." The Government have promised to print our transactions—(cheers)—they have afforded very considerable facilities for visiting the State institutions, and also granted concessions in railway travelling. (Cheers.) We associate the name of the Premier. who gave us a very hearty welcome at the inaugural meeting, and made kindly reference to the medical profession, and expressed his very good wishes on behalf of the State. We thank him for that, and especially for his hope that we might discover some means whereby patients should be made to pay. (Laughter.) We also include the Acting Deputy Postmaster-General, for the conveniences he has afforded us in postal matters.

Carried.

DR. ROSS (Sydney): I move—"That a hearty vote of thanks be given to the Mayor of Adelaide for his warm welcome, and for the interest he has shown in our proceedings."

Carried.

DR. ASTLES (Perth): I have very much pleasure in proposing—"That a hearty vote of thanks be given to the Chancellor and Council of the University for the free and full use of their buildings and scientific apparatus, and also to the members of the staff for their active assistance, particularly the Registrar, Mr. Hodge, and Mr. Fuller and Mr. Addison." I am sure that without these buildings, and without the active courtesy and assistance of the staff, this Congress would not have been the success it was.

Carried.

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Dr. Jeffrey Wood (Melbourne): It is my pleasing duty to propose—"That a very hearty vote of thanks should be accorded to the Synod of the Church of England for their very friendly message." I am sure also that I am only echoing the feelings of members of this Congress in also thanking the Dean and Chapter of the Cathedral, the choirmaster, and organist for the splendid service which the members of Congress had the privilege of attending on Sunday last, with special mention of Canon Hopcraft, for his sermon. (Cheers.)

Carried.

Dr. Thomson (Brisbane): This fair city of yours has been invaded by hordes from all the States, but you have captivated and captured us. Someone has said that the work of the Congress was getting beyond bounds, but don't you think the entertainments are getting beyond bounds? I look upon this place as a paradise for practitioners, and it seems to me that all your medical men are millionaires. They live in mansions instead of homes. They don't drive traps, but they drive luxurious equipages and motors—so, ostensibly, they are able to do so. (Laughter.) I move—"That a hearty vote of thanks be accorded to those ladies and gentlemen who have entertained us, with especial mention of the Chief Mechanical Engineer, Mr. T. Roberts, the Superintendent of Public Bildings, Mr. C. E. Owen Smyth, and the Hydraulic Engineer, Mr. C. A. Bayer." Every lady in the place has done her level best, and I wish to include in the motion all the fair sex who have been connected with the Congress.

Carried.

Dr. Jarvie Hood (Sydney): I have to move—"That a hearty vote of thanks be accorded to the Press for their full reports of meetings, and for their general information and support."

Carried.

Vote of Thanks to the President.

Dr. Batchelor (Dunedin): I feel that the motion entrusted to me is a very important one. It is—"That we accord a hearty vote of thanks to our President for his welcome."

(Members rose, and gave three rousing cheers.)

Any attempt to eulogise the work done by our President would be presumption, and I shall not attempt it. But we must congratulate ourselves on the way the work has been got through; and so long as we have leaders of the profession such as Dr. Stirling we may look hopefully for the maintenance of the status of the profession in Australasia. (Loud cheers.)

Carried.

The President: Gentlemen—I should be more than human if I did not greatly appreciate the compliment that you have been good enough to pay me this morning. It is quite true that as an individual I have done my best to make this Congress a success. (Dr. Thomson—"And you have succeeded.") Judging from your expressions of approval, we have not been altogether unsuccessful. (Cheers.) But let me say this—that although the executive head of a meeting of this kind may count for something, yet full well do I realise that any efforts would have been perfectly unavailing towards success but for certain contributory factors. I have been fortunate enough to have behind me the united medical profession of South Australia. (Cheers.) I have been extremely fortunate in having around me the most painstaking, devoted, and sagacious officials that possibly could help any President. You know their names—Dr. Hayward—(Cheers)—Dr. Poulton—(Cheers)—Dr. Gunson (Loud cheers)—and Dr. Symons, Chairman of the

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Reception Committee. (Cheers.) When I had those men alongside me with their experience, with their personal friendliness, and their indomitable energy—which is indeed necessary on these occasions—I had the assistance of most valuable men. And I say, gentlemen, when you are good enough to thank me for what I may possibly have done, I wish that it should be recognised how much we owe to these gentlemen for their kindly assistance throughout the whole of this Congress. That, I think, is all the acknowledgment I am able to make; because after all the best acknowledgment has been your kindly tributes towards our efforts, and it is very gratifying to find that you, as visitors, are prepared to make the best of any virtues we may possess, and turn a very blind eye to the many failings that you must have discovered in ourselves. ("No.") We have had a very unanimous Congress, and I think it has been a pleasant one. (Loud cheers.) I thank you all for the unfailing support you have given us. That now concludes the Adelaide Congress. (Loud cheers.)

SECTION OF MEDICINE.

TUESDAY, SEPTEMBER 5TH.

At 10 a.m. DR. COLQUHOUN (Dunedin), President of the Section, read his address.

At 11.30 a.m. DR. RENNIE (Sydney) read his paper on "Diet in Relation to Kidney Disease."

Discussed by Drs. Verco and Jamieson.

Discussion adjourned until Wednesday, to permit members to hear Professor Welsh's paper on Cancer.

WEDNESDAY, SEPTEMBER 6TH.

At 11.10 a.m. the PRESIDENT took the chair. (The Section did not meet earlier owing to members wishing to hear Mr. Bird's address on Surgery.)

Dr. Abramowski (Mildura) resumed the discussion on Dr. Rennie's paper.

DR. SPRINGTHORPE (Melbourne) read his paper on "The Use and Abuse of Mental Therapeutics."

Discussed by Dr. Rennie and the President. Dr. Springthorpe replied.

DR. ABRAMOWSKI (Mildura) read his paper on "The No-food Treatment of Typhoid Fever."

An animated discussion followed, in which the following members took part, viz.:—Drs. Hornabrook, Hayward, Jarvie Hood, Verco, Springthorpe, and the President. Dr. Abramowski replied.

DR. RENNIE read his paper on "Epilepsy: Is it Incurable?"

DR. JAMIESON (Melbourne) read his paper on "Notes of a Case illustrating the Symptoms of Tumor of the Occipital Lobes of the Brain."

Discussion on these two papers was adjourned until Thursday.

THURSDAY, SEPTEMBER 7TH.

The PRESIDENT took the chair at 10 a.m.

An interesting discussion took place on Dr. Rennie's paper. Drs. Jamieson, J. Hood, Verco, and Sinclair taking part.

DR. SINCLAIR moved and DR. JARVIE HOOD seconded—"That this Section supports the resolution submitted by Dr. Springthorpe, in the Section of

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State Medicine, viz., 'That, in the opinion of this Congress, there is imperative need for the establishment in the different States of Epileptic Colonies, in which epileptic children should receive education, epileptic adults be suitably employed, and epileptics generally find a home as well as the most successful treatment.'"

Carried *nem. contrad.*

The resolution was forwarded to the Secretary of the Section of State Medicine.

Dr. McIntyre Sinclair (N.S.W.) read his paper "The Prognosis of Pulmonary Tuberculosis."

Dr. Stoney (Echuca) read his paper "Five Years' Records, Victorian Sanatoria for Consumptives."

Dr. Camac Wilkinson (Sydney) read his paper.

Dr. Gault (Adelaide) read his paper "Sanatorium Treatment of Phthisis."

An interesting discussion was entered into by Drs. J. Hood, Gill, Lawrence, Abramowski, and the President. The readers of the papers replied.

Friday, September 8th.

At 9 a.m.—Clinical exhibits. Drs. Verco and Swift exhibited several cases of interest.

At 10 a.m. Dr. Herman Lawrence (Melbourne) read his paper "Radium and Electro Therapeutics in the Treatment of Skin Diseases."

Dr. Lawrence at the same time exhibited an "X-ray bath."

Dr. L. H. Harris (Sydney) read his paper on "The Present Position of the Röntgen Rays in Medicine and Surgery."

Dr. F. Clendennin (Melbourne) read his paper.

Discussion.—An animated discussion was taken part in by Drs. Springthorpe, Wilton Love, Closs, J. Hood, Reissmann, Benham, and the President.

Dr. Jarvie Hood moved and Dr. Murray seconded—"That this Section is of opinion, judging from the complex, and sometimes dangerous, symptoms produced by X-rays, radium, and Finsen light, that the use of these agents should be in the hands only of members of the medical profession, and that this resolution be forwarded to the General Meeting of Congress for its consideration."

Carried *nem. contrad.*

Resolution forwarded to the General Secretary.

Dr. Jarvie Hood read his paper on "The Transposition of the Organs verified by *Post-mortem* Examination."

Drs. Stoney, Astles, Reissmann, and the President remarked that they had each seen one case, and Dr. Verco said he remembered two cases.

Dr. Astles (Perth) read his paper "Is there such a Disease as Croup?"

Drs. Stoney, Gill, and Benham discussed it. Dr. Astles replied.

Dr. J. McDonald Gill (Sydney) read his paper on "Leukœmia and Allied Conditions in Children."

Discussed by the President.

Dr. Gill read his paper on "Two Cases of Lead-poisoning in Children."

Drs. Stoney and Morris discussed it. Dr. Gill replied.

Dr. Benham (S.A.) read his paper on "A Fatal Case of Acute Pancreatitis."

Dr. Benham laid his paper on "The Medical Treatment of Appendicitis" on the table, as there was no time to read it.

Dr. Reissmann did the same with his paper on "The Principles of Chemistry and the Study of Medicine."

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STATE MEDICINE, ETC.

WEDNESDAY, SEPTEMBER 6TH, 1905.

Presidential Address by DR. SYME. PROFESSOR STIRLING in the chair.

THURSDAY, SEPTEMBER 7TH, 1905.

At 9 a.m. DRS. VERCO and SWIFT had several clinical cases on view, but owing to the counter attraction of a visit to the crematorium, very few members attended.

DRS. VERCO and SWIFT exhibited a man suffering from idiopathic muscular paralysis.

DR. SWIFT showed a series of cases of icthyosis that he had treated with extract of thyroid gland and arsenic. He also brought forward three cases of psoriasis that had made very little progress under several different modes of treatment, but improved very rapidly when given thyroid extract and arsenic.

DR. SWIFT also exhibited two girls suffering from chronic progressive enlargement of joints, with enlarged glands (still), or so-called rheumatoid arthritis of children.

SEPTEMBER 7TH, 1905.

DR. SYME in the chair.

10 a.m. Paper on "Hospital Abuse" (DR. H. W. BRYANT).

10 a.m. Paper *re* "Taking of Payment from Hospital Patients" (DR. W. MOORE).

In the absence of the writers, the above papers were read by the secretary of the Section.

Moved by DR. HOWARD that DR. Rennie's paper be read before above were discussed.

"The Role of Provident Medical Associations," by DR. GEO. RENNIE.

Proposed by DR. RENNIE (Sydney), seconded by DR. BEESTON (Newcastle) "That a committee be appointed, consisting of representatives from each State, with power to add to their number, to consider the best means to be adopted to amalgamate the various medical organisations throughout Australasia and New Zealand, two representatives to be elected by the local branches of the British Medical Association in each State, by the Victorian Medical Society, and in Tasmania by the Medical Section of the Royal Society, and by each Medical Defence Association in Australasia; that the secretary of the Section of State Medicine and Medical Ethics should communicate the above resolution to the various bodies, with a request to send him the names of their representatives; that this committee bring up their report to next Congress."

Supported by DR. HOWARD; carried unanimously.

Moved by DR. RENNIE—"That the above resolutions be sent on for adoption by Congress on Saturday morning."

Proposed by DR. SYME (as suggested by Medical Defence Association of Victoria), seconded by DR. SPROTT—"That it is unethical that autobiographical or biographical notices of medical practitioners should be published in the lay press during their lifetime."

Supported by DR. BEESTON; carried unanimously.

Minutes taken as read, September 8th, 1905.

Confirmed—G. A. SYME.

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SEPTEMBER 8TH, 1905.

Moved by DR. RENNIE, seconded by DR. SPRINGTHORPE—"That it is unethical for medical practitioners to grant interviews to representatives of the lay press on medical matters in connectian with which their names are allowed to appear."

That a copy of this resolution be forwarded to the lay press.

That this resolution be sent on for adoption by Congress.

Carried unanimously.

Moved by DR. SPRINGTHORPE, seconded by DR. HOWARD—"That, in the opinion of this Congress, the future provision for the care and treatment of the insane in Australasia should be based upon a recognition of the three-fold requirements of (*a*) up-to-date mental hospitals; (*b*) farm and industrial colonies; and (*c*) benevolent asylums."

2. Moved by DR. SPRINGTHORPE seconded by DR. RENNIE—"That, in the opinion of this Congress, there is imperative need for the establishment in different States of epileptic colonies, in which epileptic children should receive education, epileptic adults be suitably employed, and epileptics generally find a home as well as the most successful treatment."

As regards the foregoing resolutions, there was a consensus of expert opinion as to the methods that should be adopted in the future in the way of housing and treating the insane and of dealing with epileptics; and there was, in addition, an especial need of having this opinion confirmed by Congress, so as to bring it to bear upon different State Governments. In the absence of any such authoritative resolution, other than scientific reasons might dominate future action, leading to great waste of public money, and the perpetuation of unsuitable modes of treatment. The resolution suggested would do much to strengthen the hands of all those who were working for reform in our lunacy administration. He had much pleasure, therefore, in moving the adoption of both resolutions.

That above be forwarded to Congress.

Papers by DR. ALLWORK—"The Ethical Relations between Town and Country Practitioners," and DR. T. C. MOORE (Napier) —"The Friendly Societies in Napier, New Zealand."

Both taken as read.

ARMY MEDICAL CORPS.

MEETING OF NAVAL AND MILITARY MEDICAL OFFICERS.

A meeting of Naval and Military Medical Officers was held in the Prince of Wales Theatre, University Buildings, on Friday, September 8th. Major Ramsay Smith, P.M.O., South Australia, presided. Lieutenant Hornabrook, South Australia, acted as Secretary.

SURGEON-GENERAL W. D. C. WILLIAMS, C.B., read a paper on "The Organisation of the Australian Army Medical Corps on a Peace Footing with its Expansion to War Requirements," which is printed in full elsewhere.

LIEUTENANT-COLONEL EAMES, C.B. (Newcastle): I move a vote of thanks to Surgeon-General Williams for his excellent paper, and for putting the matter so plainly before us. I take it also that I may move—

> "That it is desirable that a Naval and Military Section be held at future Medical Congresses."

MAJOR BEESTON (Newcastle): Do you wish a discussion in general, as to the best method of obtaining officers?

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SURGEON-GENERAL WILLIAMS: Any point you wish; but I think we should settle first the matter of the section at the Congress.

MAJOR BEESTON (Newcastle): Then I second the motion.

The motion was carried.

MAJOR RAMSAY SMITH (Adelaide): It is almost a pity to enter into any discussion on this paper. Any discussion which we may initiate or carry on will only tend to make the subject appear lopsided after we have finished with it. It is better, on the whole, to make up our minds to carry out the scheme as indicated. If we do that we shall have a first-class reserve of officers. That is very essential, and it is a thing which we should all welcome. Many of us know men who have done yeoman service in the war as consulting surgeons and physicians who do not meet us as officers. We all regret it, and I think the scheme Surgeon-General Williams has outlined to bring them in is one that deserves our support. The other point that is new to us is the extension of our service to supply a Red Cross institution. That also is an admirable suggestion, and ought to be carried out. Here in Adelaide I am glad to say that the ambulance associations are very closely connected with the Army Medical Corps. It has come about in a natural manner from single members of both associations, and I do not think it would be difficult to follow in the same course in the other States. You will forgive me for not initiating a discussion. The time will be better occupied in carrying resolutions which I hope members of the Congress will bring forward.

COLONEL THOMSON (Brisbane): My great idea is to get medical men to join the service. I do not know that it is a very alluring service. A man has to sacrifice a great deal of time to it, and to make it a success he must take it up as a hobby. Unless he does so, he is better out of it. Once he adopts it as a hobby he will find it very pleasant, very instructive, and, apart from the military aspect, he will find certain social surroundings that give it a certain charm. The reserves in some of the more populous States will be obtained more quickly than in others, and will find more ready acceptance. We have a very huge territory in Queensland, and the units in the various services and brigades are a long way apart. In connection with the 15th Light Horse there are 700 miles between the various squadrons, and it is almost impossible to get them together, and equally impossible to get medical officers to join. It should be less difficult to get the reserves, and there would be no difficulty to get the men enrolled. I do not think they would get much work out of them in peace or in camp; and it is in camp that the best kind of instruction is to be obtained. Many men are better surgeons and physicians, and are better qualified to look after patients, than those who have stars on their breasts and wear shoulder straps. But those men know nothing earthly about camp discipline, or about giving an order and seeing that order carried out. That is altogether beyond them. It is a difficult thing sometimes to get orders carried out, because some men with higher positions in the service resent carrying out orders. This association in camp, and learning the duties of camp, are of vital importance. I really think our nursing association should be extended. In Queensland we have fourteen nurses, and I see no reason why other nurses should not be on the reserve list. We can use them when opportunity offers. I do not see why some of those associations should not amalgamate, or why we should not recognise some of the civil ambulance corps. We have a very extensive transport corps in Brisbane, and they have something like eight or ten small wagons. The place is fitted with electric batteries, and a bell rings, and out they come, and go down the street like a fire brigade. That is a great advertisement. They are under discipline, and it would be a grand thing if we could get the whole of the civil medical men enrolled as a reserve, and the whole of the

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nurses commandeered as well for the reserves. Every man ought clearly to understand the organisation in his own State—not only the organisation of the A.M.C., but the organisation of every unit in the service. You are responsible for the medical examination of the whole of the men, so that you are really brought in touch with every branch. If you do not really understand the whole organisation of a brigade, then necessarily you are at a disadvantage. It does not take much time to learn it. Having learned it, you can more readily understand the work of the G.O.C., who has made a homogenous whole out of the old disintegrated parts in the various States, each of whom was working under its own Act. He has done it in a marvellous way, and secured great harmony. I have paid great attention to the scheme of organisation that was drafted by General Hutton, and the last words he said to me were—"If we could get Williams and Bridges home in London, they would reorganise the whole British Army."

SURGEON-GENERAL WILLIAMS: I am extremely gratified at the unanimity of opinion that existed over the motion that we should at future Medical Congresses have a Naval and Military Section. That will give us in the next three years a chance to bring up matters of organisation, material, transport, camp sanitation, and other things; and we ought to occupy three or four days of the Congress if this movement is accepted by the Congress.

MAJOR RAMSAY SMITH (Adelaide): I think we should give a vote of thanks to Surgeon-General Williams for his interesting paper, and bringing this matter before us.

DR. GIBLIN (Hobart): I second that.

The motion was carried unanimously.

Surgeon-General Williams bowed his acknowledgments.

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SECTION OF MEDICINE.

ON THE STANDARD OF ETHICS AND WORK IN MEDICINE.

By Daniel Colquhoun, M.D., M.R.C.P.,
President of Section.

Mr. President and Gentlemen—My first duty is to express my strong sense of the honor conferred on me in electing me President of the Section of Medicine in this Congress. While I recognise that the appointment is mainly a courteous recognition of your sister State of New Zealand and its Medical School, I none the less gratefully thank you, my colleagues of South Australia, both personally and on behalf of those I represent.

I confess that I have found great difficulty in deciding on what subjects to speak, and recognise how natural is the expression with which a well-known song begins—"That first of all we'd have you know how hard it is to write." What I say must be technical, because I am addressing an audience of experts: It must not be too technical, because I am addressing also an audience whose interest in our art is general and not particular. Nor can I forget that there is no part of the science of medicine that has not been fully dealt with from hundreds of platforms, and that I am following a company of distinguished men who have spoken on similar occasions from this chair, and who have initiated the discussions in the section of medicine with wisdom and eloquence which are hard to imitate. If therefore, in the selection of the theme and in the handling of it, I fall below the dignity of the occasion, I bespeak in advance the kindly consideration of the members of my own profession, and those other ladies and gentlemen who honor us with their presence.

In choosing to speak of medical education in these States I have been guided by the fact that this is a subject on which I have some special knowledge, and that it is a matter of importance to the whole community—lay as well as medical. *We* are interested in the quality and character of the young men who are daily joining our ranks as colleagues, and who are to be our successors, and those who are not of the profession are concerned in all that relates to men and women with whom at one time or another they must be brought into contact, and to whose skill and conduct they must probably at some time trust their lives and happiness.

To the public the technical details of our studies are of little importance: the wise man leaves law to the lawyers, medicine to the doctors, and navigation to sailors. But in every art and science the public is the supreme judge of its usefulness, and there are questions concerning our own art that have to be propounded and answered anew from time to time, almost by each generation. The most important aspect of the relation between medical practitioners and the public will always be that which is concerned with ethics. Does the profession maintain a high standard of conduct among its members? A second question, or series of questions, concerns the working side or efficiency of the profession. Is education thorough, here and abroad? Are the teachers and pupils alive and quick to grasp the needs of the hour, and alert to what is required of them? And, curiously enough, do doctors really know anything about disease and the curing of it? We have no

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objection to these questions—not even to the last: they are as necessary for those who practise medicine as for their patients. Many people behave at any rate as if the answer to the last question must be in the negative. They take drugs in enormous quantities on the strength of advertisements, they entrust the care of their health to professed healers who have no qualifications except unbounded self-assertion, and they adopt theories of the most grotesque and absurd character. But large as this class is, it is not to it we appeal. It is to the sober and reasonable part of humanity; and, imperfect as the result may be, I shall try to define our position, both as to Ethics and Education.

There has been no little misunderstanding among all classes about Medical Ethics and Medical Etiquette; but there is really no mystery about either. As they have come to us, they are the product of many generations; they have been evolved from the experience of a profession which has always had special opportunities for studying the structure of organised human society. It has been represented at the universities and schools, and has known the best of scholastic learning; it has been engaged in the affairs of war and of peace; it has known the homes and lives of the rich and the poor, the powerful and the dependent, the most and the least intelligent, the good and the bad among men. The conclusion the profession as a whole has come to concerning their relation to one another and to their patients is the result of this wide experience. Its philosophy has been of the inductive rather than of the intuitive school; but it is interesting to notice that, with regard to the practical conduct of life, these conclusions are the same as have been arrived at by the highest religious teachings. "Do unto others as you would wish others to do unto you" is the foundation of all medical ethics. And this rule has been reached, not on sentimental or emotional grounds, but as a deliberate and—one might almost say—a scientific opinion as to what is necessary for the carrying on of human society. A deep sense of the duty of the doctor to his patients has been impressed on their students by all the best of our teachers.

The oath of Hippocrates (born about 460 B.C.) is famous and well known —"With purity and with holiness will I pass my life and practise my art. Into whatever houses I enter, I will go into them for the benefit of the sick, and will abstain from any voluntary act of mischief and corruption. What I see and hear in connection with my professional practice or not in the life of men I will not divulge." Such oaths are no longer taken or required, but they are the law of the profession notwithstanding, and have been taught by our great teachers by the best of methods—their own example. From Sydenham and Harvey to Simpson, Paget, and Jenner, the English School of Medicine has followed the Greek in this respect, and the law has added its powers to the moral forces. If any medical man is guilty of conduct deemed infamous in a professional sense his name is removed from the register of qualified practitioners, on proof being given of his guilt. But the moral force is the stronger of the two. Few men are to be found who will face the condemnation of their own profession, and every practitioner knows that he will deserve and get that condemnation if he breaks the moral code laid down in effect in the Hippocratic oath.

Elaborate codes of ethics are in existence, and perhaps they might be more studied in detail in our schools and societies than they are; but they deal only with details, and they are all founded on certain broad principles. Thus, no man may have anything to do with secret remedies or secret methods —he must make no profit out of such things. The Chamberlain brothers invented obstetric forceps, but they are remembered chiefly for their infamy in concealing, for personal profit, a discovery which was of universal value. If a new remedy is useful for the relief of suffering it should be placed in the

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hands of all who can use it. If, as often happens, the supposed discovery is a mistake, it is the more quickly detected; if it is useful, its range of usefulness is more quickly determined.

All the great discoveries of our time have been improved by others than the discoverer. Indeed the advance of any science would be impossible in the face of secrecy and jealousy in dealing with new facts.

In the complicated business of professional life many minor regulations are necessary. The need of courtesy and fair dealing between man and man is nowhere more needful than in the relations between doctors. There is nothing which more surely stamps a man with the seal of mental and moral inferiority than the habit of decrying the work of his professional brethren and exalting his own. Even without that there are numerous causes of quarrel and misunderstanding between the best-intentioned of men. Every association of medical men knows by experience how hard it is to keep the peace among a few of its members. The code of ethics tries, at least, to do this, and the more nearly it is followed the chances of misunderstanding become lessened.

Another feature of the code is its condemnation of advertisement—either direct or indirect—among medical men. I will not dwell upon this beyond saying that this conclusion is a well-reasoned one, not arrived at from any pharisaical belief that we are better than our neighbors. Methods which are unobjectionable in ordinary business are not suitable in ours.

But, if we object to a policy of hustle, we are fairly determined not to be hustled. We believe that our work is important, that for its best development it must be individual, and that the man who can do good work ought to be paid for it. We are at issue on some of these points with some very respectable gentlemen. The profession has always unstintingly given its services freely to hospitals, and there is no medical man in large practice who has not on his list a number—sometimes very large—of free patients. A system has grown up whereby men of small income may get cheap medical attendance by a process of mutual insurance—the well-known club system: and this has been developed with the entire approval and assistance of medical men. But many men and women, both in town and country, have not hesitated to take advantage of the free work done in the hospitals and elsewhere. They sometimes defend themselves by saying they pay taxes. So they do for charitable aid, but they do not seek to take this out in doles from benevolent boards, and the hospital is as much a charity as the benevolent asylum.

A scheme, which may be called the Medical Syndicate, has found wide favor in some quarters. It proposes to hire a doctor for a salary, not too liberal. and assign to him the task of caring for the sick of a given district. Everybody is eligible for membership, and every member is of course entitled to his full share of the doctor's time and skill. Such syndicates strike at the very root ideas of our profession. We believe, if they were to be successful, they would destroy us by taking away individual effort, ambition, and independence.

The club system, admirable as it is, has been terribly abused. Especially in country districts, well-to-do men, lawyers, bankers, men of business, farmers, &c., have become members of working men's lodges, and have saved their pockets at the expense of our profession. We believe that this development of the system is bad, for the same reason that the syndicate system is bad, and that the chief sufferers in the long run will be the public. Not that the club doctors are inferior to their fellow practitioners in any way; that is not the case: but because too much work is thrown upon them—often of a trivial nature. They have little leisure time for holidays, and that personal

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bond which is needed for sympathetic work is very often absent. We have to fight the policy of hustle in all these forms; and it is not altogether for our own sakes. We are of no use to anybody if we are not efficient, and we trust to that fact for the destruction of systems which would end in making us inefficient.

These differences of opinion are to be regretted, but, as quarrels go, ours with a section of the public is not a very bitter one. And as we are not a weak or disunited body of men, and only desire to see justice done, we believe we can discuss most of these matters with our temporary opponents with a good prospect of coming to an understanding.

It would be impossible to discuss the ethics of our profession without referring to these matters, which greatly affect the lives of all of us. The student of to-day will be guided less by any sentiments we profess than by our actions. On the whole, I think we are inclined to stand by one another—the strong tend to help the weak, the senior man the less experienced, in all legitimate ways; and it may be fairly claimed that the influence brought to bear by the profession itself on its younger members is as high as it has ever been.

Of this we may be sure—theories of practice and science will vary. We shall add our portion to the lumber heap. Many of the details on which we pride ourselves will be as dead as Galen's twenty-five pulses or Lister's spray—as absurd as Don Quixote's attack on the windmills—to other generations. But, both for ourselves and the public, it will never cease to be of the first importance that our standard of conduct should be high. We may use the Hippocratic formula, "With purity and holiness" or the mordern colloquialism, "To play the game," but the meanings are identical and the requirements are the same.

The part played in the education of the student by the times in which he lives—by the world spirit—is hardly second to the professional influences of which I have spoken. It was not by accident that the epoch of the Hippocratic school was the epoch of Socrates, Plato, and Phidias—of all that was best in Greek life—that age produced the School of Medicine which guided and dominated science, until science, philosophy, letters, art, and religion were overthrown by barbarism and superstition. Under the vigorous rule of the Arabs —while they were a conquering and expanding people—medicine, with other sciences, again prospered and advanced. A long time of mental stagnation in Europe ended with the Renaissance. It was a true re-birth when Italy—and, through Italy, Europe —found again the old learning of Greece and Rome: and this not only because of the infinite value of the art and literature of the past, but because it widened the horizon of the human mind, cramped by the centuries of struggle for existence which followed the downfall of the Roman Empire. These struggles were to decide whether a Tartar, or a Saracen, or a Christian force should be the master of Europe, and, in the clash of arms, Laws and Letters were silent. Then Dante and Petrarch spoke, and all Europe listened. The arts became alive again, and modern science was born. The new world of America was less to mankind than the new worlds which were thrown open to students who followed the teaching of the universities of Europe. Authority yielded to observation—not with meekness, we know; but the new force was irresistible. The revival of the old learning in medicine was followed by an earnest desire on the part of students to follow the methods of the Greek and early Alexandrian Schools. Nature must be studied, and above all the student of medicine must know the material on which he has to practise. But it was not until the sixteenth century, 1,400 years after Herophilus had taught human anatomy in Alexandria, that Vesalius in Brussels aroused the enthusiasm

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of all the students of medicine in Europe by his anatomical demonstrations, and that the real study of anatomy began, and with it was laid the foundation of scientific medicine.

It is no part of my programme to consider details of medical history, except in so far as they illustrate the influence of environment on medical education. The force of the Renaissance has continued: it has moulded the whole constitution of society, and made possible the discoveries of science, the explorations, and the colonisations of the nineteenth century.

There has been much talk of political federations, but steadily, and without any apparent deliberate design, an intellectual federation of the whole world is gradually taking place. Each nation is learning that it has much to learn from every other nation. Every new discovery in science or philosophy is international, and has to stand the scrutiny of a thousand investigators. Theories which have stood for centuries are subjected to the same criticism. If a nation is backward in growth, if it has allowed itself to be smothered by sloth, corruption, and ignorance, every wind of heaven smites it, and—by reformation within or enemies without—it must be changed, or die.

We have seen but yesterday a new nation enter the field. If Japan has gained much from the Western peoples, she has added much to the common store—examples of patriotism, valor, honor, and thoroughness in all her enterprises, which cannot fail to stimulate and raise the standard of national aspirations.

There have been no such "spacious times" since the world began. Into this scene of international activity Australia has entered as a comrade and fellow-worker with other nations—it may be only on trial, for she has yet to prove herself worthy. But it is well to note that there is now, as there has always been, another spirit abroad. (It is impossible, in this connection, to avoid the language of metaphor—it expresses at any rate what we want to make clear). There is lust which seeks only its own pleasure, greed which seeks its own gain, a sneering devil which can pull down and destroy, but can build up nothing. There are tyranny, envy, and ignorance in abundance—evil influences which have called themselves by many names, porphyrogenitus born in the purple, autocrat, democrat, friend of the people: and all these influences are at work trying to destroy whatever of good the ages have built up for us. As this battle with the nobler spirit of the age goes, so shall we rise or fall.

A matter which deserves extended consideration, now and at other times, in connection with all medical schools and universities, is the health of the students. A sickly medical student is a satire upon our art. If a man cannot keep that body over which he has sole control in good order, how can he expect other people to trust to his skill in *their* ailments?

We talk about *mens sana in corpore sano:* about "health being a man's best asset." and so on. What good does it all do? Before a man has learned the meaning of such phrases he has often established faulty habits of study and living which stick to him. Too many men break down in their student career and afterwards; too many men graduate and find that they have lost mental and physical elasticity in the process of learning—not to regain it, perhaps. for years. Is there nothing to be done, systematically and steadily, in the way of attending to the students' health? Should we be content with the old rule-of thumb methods?

The making of Utopias has always been a fascinating pursuit, even if the ideal is never realised. The university might well start with the fundamental idea that it was its business to teach its students the art of living as well as learning—and through its students the whole community. This was the old Greek ideal, expressed in the words "Music and Gymnastic"—the care of the body as well as of the mind.

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There would need to be a residential college. Remembering that we are dealing with young men, not with boys, a great deal of the management could be done by the students themselves. The senior medical students might form a health committee, whose business it would be to carry into practice the lessons they had been taught in their anatomy, physiology, hygiene, and medicine classes. On his first entry into the college each man would undergo a careful physical examination—surely as important a thing as matriculation; height, weight, chest expansion, muscular force, and other important matters being observed. Monthly, thereafter, his record would be noted, so that the earliest sign of any failure in health might be detected.

A sanitary committee would supervise the household arrangements, drains, ventilation, baths, &c.

A gymnastic committee would see that the right kind of exercise was prescribed for each student.

A catering committee would supervise the food arrangements.

We preach the virtues of fresh air: the bedrooms would be as well ventilated as those of a sanatorium phthisical patient.

We preach early rising and cleanliness: The student would rise at 7 in the winter, and 6.30 in the summer; and begin his day with some form of exercise and a cold bath.

We preach moderate diet: Here there would be room for observation and experiment. The food would be the best that could be bought; the cook the best that could be hired. In these directions there should be no false economies. But the food should be plain, suitable for students, who need no heavy or late dinners, no stimulants to appetite.

It is not beyond hoping that a university might be found bold enough to establish a school of domestic economy in connection with such a college; and if women could only be induced to believe that there was as honorable and profitable a career for them in this work as in nursing, or any other occupation, one at least of the crying social wants of the day might be satisfied. If the ideal could be carried out, such a college would be a practical school of hygiene. The doctor who had been trained in it could say—"I have done these things I recommend." He would know from practice much of what he now only learns in books or lectures. He would learn something about administration of affairs and responsibility.

Many possibilities might flow from such co-operation. We might see summer camps formed for boating, shooting, and fishing, for the study of nature, and as health resorts for those who were run down from any cause. For non-resident students the college would be a club-house, a place where a good meal could be obtained, and social intercourse encouraged. We might see fewer men going to cricket and football matches, and more men playing football and other games. Surely there is nothing more lamentable in modern life than the spectacle of thousands of able-bodied young men contenting themselves with watching others playing, and having no pursuits of their own. To the university man, at least, the study of "Gymnastic," ought to be an integral part of his daily life.

The influence of such an institution would not, and could not, end with the student himself: it would extend into the daily life of the whole State; and if our universities are destined to have their rightful influence, they must be prepared to play their parts in a wider field than they have occupied heretofore, and to take a broader view of their duties.

Much would depend upon the students themselves, but not all. They cannot be expected to have either the experience or the initiative of their elders; but if they discuss these matters among themselves, and show a desire to try such an experiment, they will be supported by all the special

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knowledge and skill that the medical side of universities possess—of that we may be sure. But the young man is above most things conventional. He does not like trouble, he hates ridicule, and he has a wholesome conservative dislike of innovations. We are told that, in a state of nature, if a pack of hunting dogs has marked down a quarry and one of their number turns aside from the hunt on private business with a hare or a rabbit, the rest of the pack will tear him to pieces. Young men—perhaps all men—are pretty much like dogs in this respect. But if the quarry is likely to furnish meat for the whole pack, there may be found leaders who will try for it.

I have often thought the student at a university is too much like an egg in an incubator—so much heat, so many days, and the chicken steps out ready to fight for himself in the world. The university might do something better for him. It might encourage comradeship, make him feel more like a child who has an alma mater which educates him, cares for him, corrects him, perhaps, but is proud of him as a son who will one day be proud of her.

Leaving this question of the health of the student, let us now turn to the machinery whereby his technical education is determined. The day has gone by when the need of local medical schools might be questioned: the matter has been settled. Victoria has granted degrees in medicine to 665 students, Sydney to 285, Adelaide to 49, and New Zealand to 88. Most of these graduates are now in active practice in the colonies and elsewhere.

For an Austral student there is much to be said for an education in one of his own universities. The standard of education taught and required is not inferior to that which obtains elsewhere: the cost is less. The young man, at the most plastic stage of growth, is not cut off from those home and national influences which tend to bind him to his home and to his country. He studies the actual types of disease, and the local conditions he is most likely to meet with in practice. For the States themselves it is of importance that they should be able to produce their own doctors at home: it means that an honorable career is open to many who could not adopt it if the education could only be procured abroad: it means also a higher standard in the profession both among the teachers and those who are not engaged in teaching. For the poorer classes it means more abundant means of getting the benefit of special knowledge for the prevention and cure of disease, and for raising the standard of health.

On the other hand, a good many things may be urged in favor of educating the student in Europe. Twenty-five or thirty years ago society throughout these States was much more cosmopolitan than it is now. Most of the men engaged in active life had travelled at least from Europe to Australia, and many had a wide experience of nearly every part of the world. By the passing away of this older generation it cannot be denied that a strong tendency to provincialism has sprung up, and this has manifested itself, among other ways, in an inclination to magnify our own institutions, our own people, and, to some extent, a failure to understand our relations with the mother-country. It may well be urged that no better means of counteracting this spirit could be found than to strive to send as many of our young men as possible to seek at least part of their education in Great Britain. Not only would they in this way secure the best teaching by the best men in institutions of historical importance, but they would gain a larger knowledge of the world in which they live, and would be more likely to become citizens of the Empire rather than of one small State.

Probably things are best as they are. Some men go home, and return with a leaven of wider experience. Some remain in the States and are stimulated to better work by the knowledge that they have to compete with men from other schools.

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In Australia and New Zealand there are four universities which have medical faculties. These universities are practically supported by the States in which they are situated. This arrangement is, perhaps, the best which can be imagined. It allows of unlimited expansion through affiliated colleges, while retaining the standard of education unaltered, and secures for the universities an independent position. When we compare our condition in this respect with other countries, we can see, I think, that it has many advantages. In America, where great wealth has been given and is being given to educational institutions, the diversities of the teaching standard are very great —while the best schools are among the best in the world, the worst are among the worst. The average payment of teachers is small, so that in many cases the professors have to be men of private means, or be content to live in comparative poverty. A recent writer, Mr. J. A. Hobson, writing in the *Independent Review*, speaks strongly of the harmful influence exerted on university teaching by the endowments of millionaires. I would not venture to criticise any American institutions in an unfriendly spirit, but it may be well to see what an expert has to say about the dependence of universities on private benefactions. He declares that the independence of many of the universities has been greatly imperilled. He speaks of professors removed from their chairs because the teaching was unpalatable to those who had endowed them; and, further, states that while it is of great importance to attract money to an institution, the heads of the university are often chosen less for their academic distinction and capacity for government than for their social powers and ability to please wealthy men. Who pays the piper calls the tune; and Mr. Hobson thinks that a privately endowed university is not in a position to discuss freely many urgent social problems in ways which might be distasteful to the moneyed classes.

In Great Britain, although the Medical Council has done much to raise the standard of education, the great number of bodies which grant licences to practise medicine has not always tended to a high standard of efficiency.

In other ways the State universities have certain advantages over those of older countries. They have been able to pick out the best from many systems, and representatives of nearly all the educational bodies at home have from time to time helped to mould our newer ones. Not Oxford or Cambridge, Dublin or Edinburgh, or London can be said to have dominated our universities. Something different from any of these has been produced—I do not say something better. The main idea of the Austral universities is that their functions consist of collecting and dispensing knowledge of all kinds that can be useful to man. In medicine they have aimed at maintaining a high standard of preliminary education, instituting examinations which shall test the work done by students and teachers, and finally deciding when the students are fit to become practitioners of medicine. It is to be hoped that they may long continue to be free from sectarian and political influences; from the presence of the idle rich, who, in so many cases in Europe and America, lower the standard of education and increase its cost—I may almost add, from the endowments of millionaires, which might lessen the independence both of the teachers and of the students. A wise statesmanship will aim at supporting freely its own universities without asking charity from any man, while always welcoming free gifts which may extend their power and usefulness.

Our universities demand either a degree or a matriculation examination modelled largely on that of London University.

While recognising that it is useless and unjust to launch a student into the enormous mass of detail which forms the medical curriculum without some evidence that he has the mental training and capacity to grapple with

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it, opinions will continue to vary as to the nature and extent of this required preliminary training. The present state of the subject must be looked upon as provisional only. If time were no object few would be found to deny the reasonableness of requiring that Greek, the source of nearly all modern and ancient thinking, should be one of the foundation subjects; that Latin, once the universal language of learning, should also be studied; that logic should be taught formally; and that French, German, and English, and the elements of Physics, Mathematics, and Biology should be known to the student.

But art is long and time is short, and there is a limit to the knowledge that one small head can contain. When scholarship meant Greek and Latin the field was limited, and it was possible to be profound. It still is, for the specialist; but common experience has demonstrated that the time spent on the dead langnages is for most young people time wasted, or at least not adequately represented by the knowledge gained. As far as the medical curriculum in Australia is concerned, they seem likely to be eliminated entirely.

One may regret the loss of Greek, but it does not mean that the Greek spirit need go—the spirit that urges men to be artists in Plato's sense, not craftsmen: and when Latin goes it will leave behind it a sense of loss as if an old friend and servant had left our house for ever. It was not only the key to a great literature, but it was once the universal language of learning. Among many disruptive forces it was once for all Europe the common bond which made the united work of science possible. But to-day things are different. The growing demands of science need the utmost flexibility of speech—an accuracy and precision which are not possible in an alien tongue, however carefully studied. If a man has anything to say he must say it in his own mother tongue.

For our students English is the language they ought to learn to use and understand first of all, and if any fault is to be found with preliminary examinations it is they do not sufficiently recognise this. Far too often we meet with students who can neither write good English nor give an account of their own literature and history. Much has lately been said and written about the study of nature, but the most important part of nature for us is the right use and understanding of words. I hope if we are doomed to lose Greek and Latin we shall have a more thorough study of English in their places. French and German are second in importance. The man who spends his scanty leisure in studying the latest discoveries of science in German, French, and English, and perhaps in other tongues, is somewhat of a fancy picture. Here, as in other branches, we have to depend on the specialist in journalism to keep us acquainted with what other nations are doing; and we can be sure that nothing of importance is published in any language which is not quickly translated into English. It is good for those who possess a knowledge of these and other tongues, but we want no collection of smatterings. If they are demanded let the standard aimed at be that of Baltimore—a good working knowledge of French and German, both for colloquial use and for reading.

Once the student has passed the preliminary stage he finds himself at the beginning of a five years' course of hard work, the general arrangement of studies being the same in nearly all civilised countries. One year is devoted to preliminary science training; he learns something of physics and mechanics biology and chemistry—subjects useful in themselves, and which help him to know something of the scientific conception of the universe and the forces which act on it, and useful also in helping him to methods of study. Purely scholastic methods which have prevailed in his earlier education now become united largely with laboratory work. Of the wisdom of this part of a student's training there can be little doubt: it is, in some ways, one of the most practical parts of his course. Year by year it is more necessary to know something

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of electricity, light, and heat. The X-rays have demonstrated how, from unexpected sources, new power may be lent to therapeutics. Finsen's experiments and practice with various colored lights on living organisms is only second to the X-rays in value; and in other departments of physics the studies are at least of equal value.

No less important are the lessons derived from biology; and perhaps the special application of these sciences to medicine is not always born in mind by teachers. We are still somewhat under the domination of the school which regarded botany as being specially concerned with medicinal plants, and with the naming and identification of species. More important is the consideration of the vital phenomena of plant and animal life, especially of the lower organisms and their relation to the higher living things. These lower organisms are now known to play an important part in the production of most of the diseases which attack men, animals, and plants. From a study of these diseases we can learn much of those which affect the human body. Pestilences occur among plants such as the potato or the vine. Examination shows that the causes of these diseases are to be found in the presence of a fungus with enormous powers of reproduction. There are two ways of dealing with the growth of this fungus: one is to attack it by germ destroyers, spraying, fuming, &c., and the other is by seeking to improve the condition of the plant and the nutritive properties of the soil in which it lives. These two methods are analogous to the two great methods in use in medicine: we can either attack disease by strengthening the organism against attack, and enabling it to throw off the germs which try to live upon it, or we can try, by the use of antiseptics and antitoxins, to destroy the lower organisms in and out of the human body. The causes which lead to the degeneration of individuals and of species can be studied freely, and, in the case of plants at least, without hurting the susceptibilities of any society for the prevention of anything: and the lessons to be learnt are invaluable, for life is one, and the laws which control the welfare of one set of organisms are the same as the laws which control the lives of all.

These studies are productive of valuable general notions which bear on the science of medicine, and are also becoming daily more essential for their practical details. The methods of investigating the life histories of bacteria and the lower animal organisms is now essential to an understanding of many diseases, and is begun at least in the biological course. The same things may be said of chemistry in all its branches, but especially where it deals with organic materials. It is true that all these subjects are so wide in their scope that the medical student can only touch the fringe of them. But, in his future career, he will be confronted from time to time by problems which must be referred to the chemist, biologist, or physicist, and he must be taught at least to know these problems when he sees them.

To a few students, no doubt, such studies are mere taskwork, to be got through mechanically, with as little effort as possible, but to many it is the introduction to that knowledge of nature which it has been the object of the wise of all ages and nations to strive to attain—a glimpse of that harmony and beauty "which will never die into nothingness," and which will be a retreat for him throughout life from the daily cares and worries of his business.

The studies which follow are all determined by his life and business, and are on the same lines in all medical schools. Anatomy and physiology, disease and the anatomy of disease, methods for the prevention of diseases, and their cause, the legal aspect of medicine—these are the objects of work for four busy years—or five, if the new proposals of the greatest English examining bodies are carried out. Of the details of most of these studies it is not my business to speak, but there are some general features which demand attention on such an occasion, both by the public and the profession.

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We have always been expected to have accurate knowledge on all subjects connected with our work, and we have always been hampered, and often prevented, from gaining that knowledge. Here I would like to disclaim any idea of making a divinity of knowledge, and sacrificing anything and everything to gain it. It is bought too dear if it is purchased at the cost of cruelty to man or beast; and, unfortunately, the minds of men are so constituted that unlimited power of experiment and observation have led to abuses, and occasionally to horrors, which have brought about reactionary resistance to legitimate observation and experiment. I know of no profession or business which is not the better of outside criticism and restraint. Our medical schools and training hospitals are the better for lay management and criticism—our professional work is the better for it. We need never expect to be free from such influences; but it is our right and our duty to protest when we find superstition and sentimentalism and loose thinking interfering with the objects of our existence.

I have alluded to the long struggle which anatomists had to engage in for centuries. Much of the opposition was and is purely atavistic. It is rooted in the ideas of primeval man as to his future state. If his body was mutilated in this life or after it, he entered the spirit world a maimed being: if his body was treated with disrespect, his angry spirit would haunt and injure the survivors who permitted the dishonor. Consciously, or unconsciously, these ideas have stood and still stand in the way of the rational scientific study of the structure of the human body in health and disease. And only as the wiser view gains ground, viz., that the body is no more than the outward garment of the man, can we hope to find an absolutely scientific practice of medicine. Our knowledge of disease is founded mainly on our knowledge of normal anatomy and the alterations caused by diseased processes. We must have freedom in the States to examine with the scalpel and the microscope if we are not merely to become the depositories of other men's observations. This is one point on which we have to complain often of the attitude of hospital managers and of the public, and it cannot be too strongly urged that the loss is theirs as much as ours when these examinations are neglected.

Another point on which there has sometimes been misunderstanding is as to the free admission of teachers and students to public hospitals. This has sometimes been opposed on the supposed ground of the interests and feelings of the patients. I may say I have no sympathy with any who would claim for university teachers a monopoly of hospital appointments. Much of the most valuable work, both in practice and teaching, has been done by men who have had no connection with the universities. A public hospital should be open to all duly qualified men who are willing to give their services. It is true that unsuitable men are sometimes appointed, but the same thing applies to the duly accredited teachers. The advantages for good professional work are great in all hospitals, and these advantages should not be monopolised by any body of men. But it is not likely to be denied by anyone conversant with the subject that the standard of work is always higher among all the staff where there is a medical school than where there is none: thus it is in the interests of the patients as a whole to have such schools. But there are some principles of selection which ought to be observed, if the right men are to be chosen. Other things being equal, the man with special surgical qualifications should be chosen for a surgical post, with medical for a medical.

The senior staff should be recruited from the junior. Each hospital of any size should have a senior staff and a junior staff in all departments. It should be the business of the junior staff to assist the senior, to take their places during absence for holidays or other reasons, and in this way they

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should be trained for the responsibilities of the senior position when their time comes. The present method in many hospitals of appointing men to the most responsible positions—those of physicians, surgeons, and specialists—without any preliminary training to demonstrate their fitness, is in every way absurd, unfair to the patients, and unfair to those practitioners who are prepared to take trouble to fit themselves for doing the very best kind of professional work.

There ought to be some continuity of work. Annual elections by hospital subscribers mean touting for votes, and that appointments are often given to the most unfit. Where medical schools exist in connection with hospitals the governing authorities of these schools should have a fair representation on the hospital boards. This is not undemocratic: it is only recognising that the medical school really represents the whole of the people who may have to depend in sickness on the skill and honor of its graduates, and who, through Government subsidies, pay a large part of the cost of maintenance of hospitals.

We in New Zealand, at least, have reason to recognise gratefully the attitude assumed, especially of late years, by our various hospital authorities towards our Medical School. If friction has occurred it has been temporary, and never serious. In advocating representation by the university on the boards of hospitals used for teaching purposes it is therefore not done in a carping spirit, but because I believe the change would result in a better understanding of the needs of the university on the part of the hospital trustees, and a better understanding on the part of the universities of the difficulties which have to be overcome.

If the presence on a hospital staff of specialists engaged in teaching is an advantage to the institution and to the patients, by increasing the precision and accuracy of the work done, this is largely due to the presence of students eager to learn and critical of methods. But besides this, the students themselves are, I believe, a great gain to the hospital. Less instructed than their contemporaries very often in the business part of life, there is no class of the community which has anything like the training and knowledge of human nature which these young men get. They bring into the wards an atmosphere of youth and energy and kindliness which is quickly felt even by those patients who have most dreaded the experience. Children delight in them—older patients talk freely to them; they get at facts, often important, which their seniors would never get hold of; they keep in touch with all the details of their cases, and render invaluable help in treatment. Follies they have, no doubt, and things could be, and are, cited to their discredit—as Paley said, the man who is never foolish is probably a fool all the time. But I know of no men who are in closer sympathy with the sick and the poor than are the medical students, nor who are more welcome and more trusted by them. And yet we have often had trouble to get them duly instructed—trouble arising chiefly from theorists and morbid sex-obsessed persons. They are the men who are to be the help-bearers to the sick and to women in their troubles, and yet we are told it is indecent, indecorous, improper, that they should have the opportunities of learning what these sufferings are. It is pitiful that we should have to protest against these objections. If we have to protest in vain, then let us shut up our medical schools and take again to amulets and charms and spells, or their modern equivalents—electrical belts, magnetic rings, and faith-healing.

At one time a man might hope to be master of the whole of the healing art, but gradually medicine has been split up into a great number of departments: and while it is possible and necessary for a man to have an intelligent comprehension of them all, it is not possible for any man to attain to expert knowledge in all.

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As nearly all these departments are represented at the Congress, I need not speak of that special branch whose deliberations are inaugurated by the assembly. All have their foundation in anatomy and physiology, and all depend on a close study of nature, on the observation of innumerable details, and on the philosophical consideration of facts accumulated.

Let us look at the task of the student of medicine. First he has to learn from books and lectures the main facts about disease which have been accumulated by others. It has been the fashion—it may still be in some quarters—to sneer at books and lectures, to say that only practice can teach medicine, that the book man is good for nothing, and so on. All this is pure nonsense. Everything that we have in the way of knowledge is to be found in books, if we only had the art to extract and apply it. Theory and practice are not opposite and incompatible, but are complementary to each other—each is incomplete without the other.

The book man may be dangerous, but he is not half so dangerous as the so-called practical man who neglects books. Serene and imperturbable in his ignorance, he recognises nothing that has not come within his own narrow experience.

The student must study in books, and if he cannot transfer into his own head more than a small part of what he reads, he will at least know where to look for his facts when he wants them.

Good text-books are, fortunately, abundant, and it is due to the modern co-operative system of work that these text-books can be produced with the latest facts in pathology aud therapeutics in every part of the world, set clearly before the student. If we compare a text-book of to-day with that of a hundred years ago we shall see the difference in method.

Cullen, of Edinburgh, was one of the famous Edinburgh teachers, and his first "Lines of the Practice of Physic," 1777, had a great reputation well into the early years of the nineteenth century. The edition of 1816, edited by Dr. Reid, is a logical methodical book, clear and sensible always. Specially valuable and interesting are the references to digitalis and its uses, to Fowler's recent introduction of arsenic in the treatment of intermittents, to Currie's views on opium in fever, of affusion in scarlet fever, and to Baillie and Hunter on hydatids of the liver; but one is struck with the smallness of the circle of observers, and its limitation to practitioners in Scotland and England. Laennec's great discoveries are not mentioned, although we know they had then revolutionised medical work in France and on the Continent. This speaks partly of conservatism, but more of the want of communication between different centres.

If we look at a modern text-book we find everywhere the traces of the work of those useful specialists who devote themselves to collecting, from all sources, the work of each observer. No journal is too obscure, no language too little known, to be searched: no matter where a man lives, he has the means of knowing what workers are doing in any branch of science he is interested in.

The advantages to the student are clear. He is no longer a man of one school, however great—Edinburgh or London, Paris or Baltimore. Other factors, of course, come into play in the making of the text-book. The free communication between countries, the journals, the societies, all do their part; but the great bulk of the material produced makes it difficult to keep in touch with even a part of it, and makes the work of the co-operative specialist absolutely necessary.

If the personality of the teacher is a less dominating factor than formerly in the medical school, his duties are by no means at an end with the evolution of the most perfect of text-books. In the nature of things these books can be

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no more than abstracts of knowledge, and they must be supplemented by the living voice, which will illustrate by accounts of cases, pick out the more from the less essential, emphasize by reiteration where it is needed, question, and demonstrate.

Not fewer, but more, lectures are needed—lectures on cases, on treatment, on underlying principles. The spoken word, representing experience, lives in the mind, attracts the attention, when the written word imparts but the shadow of a recollection. The business of the university is to teach: not merely to point out to the student where he may get his knowledge.

The same methods of co-operation have produced for the more advanced student—the practitioner who does not disdain to add to his knowledge—many admirable systems of medicine and surgery, consisting of monographs written by specialists who know their own subjects as thoroughly as they can be known. The man who in his former student days has learned to read can find in these volumes help and instruction in all the experiences of his medical life; and if he systematically seeks that help he will find that, along with the practice of his profession, he in a few years will have educated himself to the highest standard that can be attained. We all know men who have done, and are doing, this in every department of practice.

There are two faults in all our books, which will probably never be entirely removed, but which might easily be lessened—the use of foreign words where English could be used, and the increasing use of personal names in describing diseases and symptoms. Why should we say Erythrocyte and Leucocyte instead of "red cell" or "white cell"? And so on, with hundreds of words. The Greek used the simplest and the most direct language he could command to describe what he saw—white skin, red skin, swellings. With no knowledge of Greek, we use Greek to describe what we see, or, with as little reason, Latin, or French, or German; and this method of naming things does not help to clearness of ideas. More mischievous is the abuse of proper names. Who can hope to keep up with the increasing torrent, however gratefully we may recognise the new facts which have been added to science? Even the oldest and most venerated names become a snare to the student, instead of a help. What is Potts or Collis to him, that their names should be given to special fractures, for his undoing at examinations? He does not care whether Bright was a statesman or a physician, and he is quite as pleased to think that the nodes of Parot are so called because they resemble the beak of a parrot as that they commemorate a distinguished French surgeon. And really such names are useless. If we want medical history, let us make it part of our curriculum. There is something to be said for that; but there is nothing for this quite ineffective way of keeping great men in remembrance. A really serious effort to purify medical language is worth making, and I think that we would find that English could be used with advantage in a majority of cases of disease and symptoms.

There is still another class of books for which there is a future. Hutchison has led the way in his excellent "Archives." Byron Bramwell has done something of the same kind in his "Clinical Studies," and in his "Atlas of Clinical Medicine," a costly book, but surely the most magnificent volumes that have ever been produced in pure medicine. Lawyers have their "Leading Cases": why should we not have the same thing done systematically in medicine? Would it not be possible, and perhaps profitable, to the publisher to have the whole field of practice illustrated by actual cases, as Bramwell has done for a part? In the medical journals of the world there is abundance of material which might be made available. There one might find thousands of cases accurately observed which could be selected, and added to, where necessary, by fresh observations and findings in pathology and therapeutics.

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Of great interest and value would be the records of the original cases which led to the definitions of various forms of disease. Such records are to be found in the writings of nearly all original workers. Graves would yield the brief notes he made on exophthalmic goitre, Addison his cases of adrenal disease, Bright his observations on renal trouble, Jenner, and others of his time, their records of typhoid and typhus. Such volumes might be illustrated where necessary—not as elaborately as Bramwell's Atlas, perhaps, but well enough to help the text.

Such a record would never be out of date. Systems and theories change from time to time, but a case well studied is always of value.

When we are puzzled in practice, it is from the details of actual cases that we learn, rather than from general abstracts of knowledge, however skilfully done.

If I have tried to emphasize the value of bookwork in our plans of medical education, it is because no more dangerous attitude can exist among students than that of despising books, and not to exalt that branch above the equally necessary practice study which must go on at the same time in the wards and in the clinical laboratories. In these branches we have no new principles, nothing that our predecessors did not recognise as fully as we do. We have to study nature, and observe as closely as we can the actual facts which are before our eyes; but we have the advantages of innumerable helping methods which were unknown to them, and which are continually being added to. The mastery of these methods is a great part of the student's work. If his training has been sound, he knows what a healthy body is: he has now to find out in what ways that standard may be departed from. Gradually every organ and every function of the body has been brought under exact observation and analysis. Thanks to Avenbrugger and Laennec the lungs and heart can be explored, the thermometer reveals the exact degree of fever present, secretions can be analysed, the microscope shows the minutest changes in the blood and tissues and the presence of harmful organisms, the Röntgen rays show change in the bones and soft tissues. It is not claiming too much for medicine to say that its part is to estimate the health value of the human body as the engineer estimates the strength and value of the materials he has to use, and the strains they may be subject to, and that he has, as a rule, the means at his command to make that estimate. We sometimes hear that medicine is not a science—that it is purely empirical, that we pour drugs, of which we know nothing, into bodies of which we know less. Never were gibes more empty and meaningless. What is science if medicine is not a science? We know there is no such thing as absolute knowledge among men. But we do not say there is no science of navigation or shipbuilding because ships are lost at sea, or that there is no science of engineering because of a Tay Bridge disaster.

If science means the systematic accumulation of correct knowledge by observation within the limit, but to the full extent of, human powers, then medicine need not fear the test, and can claim its place with the other departments of human knowledge. And not only that, but it must remain where it has always been—at the head of all the branches of the science of healing. Its business is to take the most comprehensive view of the human body in health and in disease, and of all the methods whereby the one may be maintained and the other eradicated or modified. Whatever may be the skill of the specialist, the surgeon who deals with all the body or a part, it is at his peril and that of his patient that this view of the relations of the whole organism are disregarded. No one values the skill of the surgeon more than the physician; and I believe that, equally, the wise surgeon recognises that in most of his cases there is a medical aspect which is better observed by the physician

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than by himself. And, striking as are the achievements of the surgeon, it is still the province of the physician to deal with the greater part of the maladies of the body; and the public may rest assured that the study of all the means which can help to make men efficient is being pursued in all parts of the world with an energy and enthusiasm which is exceeded in no branch of science.

It may be asked if the curing art has kept pace with the acknowledged advance in the science of disease. I have very little hesitation in answering "Yes" to this question. The most striking proof of general improvement in the healing art is to be found in the improvement in the value of life during the last hundred years, or less. In the early part of the nineteenth century it has been estimated that the average age of death was about 20 years; it has now been raised to at least about 40: and this has been obtained through the application of commonsense methods to daily life. Human beings flourish when they get a plentiful supply of fresh air, sunshine, simple food, and exercise. This has been the reasoning which, from the time of Hippocrates, doctors with more or less success have tried to instil into the public mind. In the matter of the life of the individual medicine has been no less successful. It has made it its first business to get an accurate grasp of the real nature of disease and degeneration; and, having done that, it has aimed at combating these causes. Nothing is easier than to despise the past and to see nothing in it but its blunders; but our whole knowledge is founded upon the work done by those who have gone before us, and they have left behind a vast treasury of material, which is of the greatest value in therapeutics. If we have dropped much of these methods we have retained more. Only those who know little about medicinal substances deride them, on the principle that it is the bad workman who quarrels with his tools. There are few diseased conditions which may not in some way be helped or modified by the judicious use of the medicines which are at our command. We are learning something day by day—something of the limits of our art and many new possibilities of its extension. There are obscurities and difficulties, for are we not dealing with Life—the greatest of all mysteries? There are half truths and whole truths, false theories and true, in abundance, and it needs a cool and wary head to disentangle them from one another.

Once the student has finished his career at the university, and has duly passed his examinations—and, whatever may be said against the examinations, they remain at present indispensable as the only means whereby we can judge whether the student has made a right use of his opportunities or not—it is not to be supposed that the work of study is done, and that he is for all time a workman completely equipped for anything he may have to do. The view we have taken of this matter in New Zealand is that it is the duty of the local graduate to pursue his studies in other countries if possible; in his own, if that is the only way open to him. More than 50 per cent. of our graduates have so far proceeded to Europe after graduation, and I think we may say the others are only waiting their opportunities to do the same. Of these about 10 per cent. have taken their F.R.C.S. of England, and most of the others have taken the conjoined qualifications of the English colleges of physicians and surgeons.

Whatever may be said in favor of the local study of the elements of the profession, it seems certain that great advantages must accrue to the student who knows by practical experience what is being done in other schools than his own. No one who is conversant with the history of recent medical work can fail to recognise the excellence of the great schools of France, Germany, Italy, and Spain. But to men whose mother-tongue is English there can be only two countries which make a supreme demand upon them, and those

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are the great mother-land and the two Americas—Canada and the United States. Notwithstanding much idle talk, Great Britain still stands in the very first rank of those nations where the highest thinking and the best work is being done. If you want the best of anything—books, machinery, clothing, or instruments for scientific or any work—you know that you will get it in Great Britain. If you want the clearest ideas, the best exposition of new and old methods, I believe you will get them nowhere better than in that country we are all pleased to call "Home," and, above all, you get it in the tongue you know best.

A visit to America is of the greatest possible value for post-graduate instruction. The student will see there a great nation full of energy and enterprise. In all the large cities of the States and of Canada universities and medical schools are filled with men who are eager to put into practice the newest and best methods of universal learning. No men are more cosmopolitan in their teaching, and nowhere would the student see more excellent practical work. It is perhaps a fault of their virtues, that they are so eager to try whatever has been discovered in Europe that they have not produced that amount of original work that might have been expected from their numbers and their energy; but it is easy to over-estimate the value of originality. The discoverer of new methods and new principles is born not made, and it seems clear from the history of the last hundred years that the atmosphere of the old world is more favorable to the production of original work than that of the new one. It was said of the Portuguese in the early days of their geographical discoveries that they marked the quarry, and other nations ran it down. America has run down much quarry marked by the older nations. An Englishman discovered the anæsthetic power of nitrous oxide, and said it might be used some day for surgical operations: an American used it. Boerhave spoke of ovariotomy as a possible operation: McDowell of Kentucky did the operation. Laveran, Manson, and Ross showed the nature and method of malarial infection, and Americans made Havana a healthy city. Pasteur and Lister founded antiseptics, and nowhere in the world is the practice more thoroughly carried out than in America.

One of the greatest of modern teachers, Osler, late of Baltimore, now of Oxford, lately speaking of Johns-Hopkins College, said it had been his ambition to make it equal to one of the great German Clinics: and it is true that he has left Baltimore, with the help of his colleagues, in the first rank of teaching institutions of the world. But the saying is characteristic, I think, of the American view. You would not hear such language in Great Britain. There Edinburgh is Edinburgh, Dublin is Dublin, Guy's is Guy's—each with a great history behind it, doing its own work and looking to its own future. In Great Britain, in London, Edinburgh, and Dublin, the student finds himself in the middle of practical work where the ideas which have pervaded the world can be learned at first hand. Nowhere will he see better hospitals or better methods. Nowhere is there more abundance of special work in every department of his art, and the arrangements for the instruction of colonial visitors and the friendly spirit which actuates teachers and students alike in their dealings with visitors is above all praise. Such an addition to the education of the colonial graduate needs no arguments in its favor; and it is only by carrying out such a class of study that we can hope to found a high level of attainments throughout the colonies, and keep it in coming years. Nor are other reasons wanting in such an argument. The student who has passed through such an experience is likely to be a better man, not only professionally, but in every other way—less provincial, more in touch with modern ideas, better acquainted with the great Empire and race of which he is a part. Nor should he neglect to visit the other great European

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countries. If he gains nothing else he will at least learn, if he is teachable, to lose some prejudices against foreign peoples and some conceit about his own.

Men from British schools, from America and from Australasia, go in great numbers to the schools of Germany, Austria, and France and meet together, and with the teachers and students of these countries, to the great advantage of all. In France the schools are practically free to everyone. If a man has been foolish enough to think of the French as an idle or frivolous people, twenty-four hours in Paris ought to be enough to destroy that idea. Now, as always, France is foremost in its work in all branches of medicine. It is a country of high ideals, of logical methods, of original and systematic work. The visitor will see in Paris every variety of hospital practice—while the idler has hardly begun his day, teachers and students have already done a day's work. You may visit the Salpetriere, where Charcot taught, and where his successors worthily carry on his traditions; the great hospital and museum of St. Louis, with its wonderful models by Baretta; the Hotel Dieu, and the other great general hospitals; the Pasteur Institute, with the adjacent hospital for infectious cases—a very "holy chapel" of hospitals, as perfect in every detail of construction as art and science can make it. And everywhere the visitor will be received with courtesy and kindness, for there are no more perfect gentlemen to be found anywhere than among the great teachers of the French schools.

I need not speak of what we owe to Germans in all the sciences, but especially in medicine. In the framework of their minds I think they are nearer to us than the French: we think and work together. The best men of both nations deplore all the influences which would separate us, and rejoice in everything that tends to make us understand one another. Medicine is one common ground, and whether in Berlin, Vienna, or Hamburg, the student will find abundance of practical help in all his studies. The man is fortunate to whom the difference of language is no barrier; but even for the man who speaks no language but English there are few difficulties. The Germans are good linguists—they give us cause for shame in this; but it makes study among them come near to be as advantageous to us as study in England or America.

The great International Congresses may not have fulfilled all the hopes of their founders, but they have done something towards breaking down national jealousies, and replacing ignorance by knowledge and mutual respect. I have had the privilege of attending two of these great meetings—the last at Madrid, two years ago. It is easy, of course, to find fault with details, but the man must be incased with self-confidence as with triple brass who does not feel humble in the presence of the famous workers of all countries who take part in these deliberations, and submit the results of their labors for criticism and consideration by their fellows. These international meetings are the outward and visible sign of that federation of the nations in the common bonds of science of which I have already spoken—they are crippled at present in their usefulness by the difference of tongues, but we must hope that our successors will overcome that difficulty by a better knowledge of German and French, or that we may find a common language, such as French, which may take the place of Latin for international intercourse.

There are many other agencies which help to keep up the educational standard of the doctor after he has gone into practice—such as books, journals, and medical associations. Of none of these is it necessary to say much; their usefulness is apparent. It is only to be regretted that so many members of our profession do not avail themselves, as they might do, of the advantage of local societies and such meetings as this for the discussion of their common interests and of their daily work.

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We are sometimes told that our medical associations are great trades unions. I am afraid those who use this phrase do not always mean it as a compliment—even when they are unionists themselves. Many of us would be glad if our associations could emulate the virtues of the best of these great unions—the loyalty of their members to each other, and mutual helpfulness. Perhaps they might also learn some lessons from us, and we might then see the spectacle of one of these unions meeting, as we do, to discuss new ways of improving their work; trying to find out what other nations are doing—European, American, or Asiatic—and selecting the best of these methods for the improvement of their own work.

There are many problems before us in medicine when we have mastered the knowledge that is available from the work of the past. We must still seek after new definitions of disease, and endeavor to understand better its near and remote causes. We must continue to seek for new methods of cure and prevention of disease. But, bearing all these things in mind, I am inclined to think that a great deal of the most useful work of the future must begin with ourselves—with putting our own house in order. If we can first of all teach our students how to live sensible health-bearing lives they will carry the habits into mature life with them. We have left these things to chance, to the uninstructed parent or pupil. We might as well leave any other branch of knowledge to the same influences. Logically, we ought to begin with the schools. We should examine the teeth, the eyes, the lungs, the digestive organs, the muscular systems of the pupils. But we know we cannot do this. We can, however, begin with the medical schools if the students will help us; and, unless our theories are false, we ought to be able to turn out a body of young men whose physical and mental condition will be the best argument that can be used to persuade other classes of society to carry out the same system.

To go back to the vegetable kingdom for an illustration: we find that the best authorities tell us that for stamping out and avoiding disease among plants we want rather abundance of manure and food than mechanical methods of destroying parasites.

With human beings also disease is most often a manifestation of imperfect nourishment, due to causes often to be traced back for many years—not truly to the mere local parasite, which has found the tissues weakened, and has attacked them successfully.

I have tried, imperfect as I knew the result must be, to lay before you the standard of ethics and of work of the profession of medicine of to-day. I do not think we have reason to be ashamed of either, but individually we must know how far short we fall of carrying out these ideals. "The artist," said Plato, "works for the supreme end of his art, whatever it may be; the craftsman works for his wage." We try to do honest work, to be help-bearers to the suffering, to shun greed, envy, and malice, and self-conceit, to take large views of life—and we fail often enough; but because we try, I do think we gain, as few of our fellow men do, the trust and kindly feelings of those among whom we work, and that after all is our highest reward.

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THE PRESENT POSITION OF THE RONTGEN RAYS IN MEDICINE AND SURGERY.

By L. Herschel Harris, M.B., Ch.M.

Mr. President and Gentlemen—Skiagraphy has already passed through two epochs of its history since the discovery of the X-ray by Professor Röntgen. The first epoch of discovery and publication was one of unbounded enthusiasm, during which wonderful tales were told of the new force and its posibilities. The expectations of the public and profession were raised to the highest plane, only to be followed by the second epoch of disappointment and condemnation.

During the first epoch, unfamiliarity with the characteristics and actions of the X-ray was the cause of many accidents and disappointing results from its employment. Accidents which occurred during the first two epochs, in the form of severe X-ray burns, dismayed many early operators, and caused a feeling of suspicion as to the expediency of its employment. Idiosyncracy was given a very prominent place, which later investigation has shown to exist only in a moderate degree; burns having been oftener produced from over-long or too frequent exposures in the hands of those who were unfamiliar with its actions.

Surgeons began to doubt its value in localising foreign bodies, as they had often failed to find the object in the spot indicated on the skiagram. Up to this time it had not been appreciated that the relative position of a body, with reference to the anti-cathode and the sensitised plate, often produced a deceptive location of the shadow of the image upon the skiagram. The causes of distortion have since become better understood, and, by cautious methods, localisation is made more accurate.

During the second epoch, the therapeutic uses of the X-ray began to be studied by a few early observers, but received no particular attention or credence till within the last four years or so.

The third, or present, epoch is the one of technical scientific study, and a better appreciation of the limitations of an agent supposed at first to possess more remarkable qualities. There is also, with the further realisation of possibilities, an accomplishment of results which tends to restore confidence and again bring the measure into universal favor: it is the epoch of realisation and substantial recognition of this valuable diagnostic and therapeutic agent.

There are still many questions calling for more careful study of the scientific application of the X-ray as a diagnostic means. At the present time, however, its use in accurately locating foreign bodies, diagnosing calculi, fractures, and various visceral conditions has become too certain to leave a doubt as to its value. What is now required is greater skill in its application, which cannot be expected of the general practitioner or student of medicine, who has not received the proper technical instruction in its different applications.

The subject is a progressive one, and in the near future many improvements will be made in appliances for more definite and precise localisation, rapidity in making exposures, and development of the sensitised plate, and improvements in tubes; as well as other features which will tend to perfect the methods in present use.

It often occurs to me, when I see anyone now starting X-ray work, how very easy everything is made for them. With a 10-inch coil, a good

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interrupter, a suitable electric supply, and a choice of any number of first-rate tubes, it is possible for anyone to rapidly master the technique of ordinary radiography. The purely photographic side can be reduced to simplicity itself; whilst, with regard to the treatment of cases, such an enormous amount of information has been published by one and another worker, that it is comparatively easy to acquire sufficient information; so that at any rate one can use the X-rays without the risk of doing very much harm. This is the view I take of radiographic practice at the present time; and, thinking of the early days, from 1896 onwards, when there were innumerable difficulties, mistakes, and disappointments, one is inclined to envy the happy individual who is now only just starting the work.

At the same time, there is the other side of the question: and there is the pleasure of thinking of the conquered difficulties, the gradual acquirement of the knowledge—often gained by bitter experience—which enabled one to conquer those difficulties and problems; the keen delight which successful, early, and first results brought; the intense excitement of the first coin seen stuck in an œsophagus, and easily and safely removed with the certain knowledge that it was there and to be removed; the scarcely less pleasure and excitement when a case of rodent cancer was seen to yield to X-ray treatment—when those successful cases could be counted on the fingers of one hand. All those pleasures are lost to, and not to be obtained by, the present day beginner, who has to look for his pleasure in the more or less easy way of following where others have led, and his consolations in looking at his beautiful X-rays, full and rich in detail, and in comparing them with the unfinished, detailless, and indifferent radiograms that some of us were so proud of seven or eight years ago. One of my most valued possessions is the first X-ray tube I used, which I zealously preserve; and, when looking at it often wonder how it was possible to obtain any result at all with it. Nevertheless it served its purpose, and helped to locate many a needle in the palm, and diagnose fractures in the extremities.

Professor Sylvanus Thompson, in his presidential address in 1897, said:—"Excepting only the introduction into surgery of Lord Lister's antiseptics and the discovery of anæsthetics, no discovery in the present century has done so much for operative surgery as this of the Röntgen rays." This was a somewhat bold statement made at that time, but I think most surgeons would agree with the opinion thus expressed.

First, let us consider the use of the X-rays in the diagnosis of injury to bones and joints.

I think I am pretty near the mark when I estimate that 90 per cent. of all bone injuries are now subjected to a radiographic examination. The reason for this is very obvious when one comes to consider the numerous complaints made by many patients, even though a good result be obtained, and the numerous lawsuits which frequently follow. With many patients now there appears to be an idea, after they have sustained a most serious accident involving one or more fractures, that if they cannot sue the Government for damages their medical attendant is the next best mark, no matter how good the result, and how little the deformity.

In the Sydney Hospital we make it a rule to have every fracture screened or skiagraphed. With the indoor cases a copy of each skiagram is attached to the patient's case-sheets, and the plate is carefully numbered and kept for reference at any time. Sometimes one case may be skiagraphed four or five times, if necessary. Needless to say, the diagnosis is always made first and confirmed by the rays. A word of warning here may not be out of place. It is very easy to adopt the attitude "Oh, here is a bone injury; we must

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have it X-rayed, and the diagnosis and treatment must depend entirely on the report." The man who would systematically and as a routine follow this rule will, in time, put himself at the disadvantage of not having cultivated and brought to perfection those valuable qualities of careful observation of symptoms, &c., which enabled our pre-Röntgenite forefathers to diagnose and locate fractures with such great skill and success.

I would apply this suggestion not only to the use of X-rays, but would even go further and assert that there seems to be a decided tendency at the present day to trust rather and altogether to modern "instruments" of diagnosis, such as thermometers, microscopes, radiograms, inoculation of animals, exploratory operations, and so on; and so, almost unconsciously, to get into a habit of practice which certainly does not tend towards a training of, and increasing of, a careful and methodical faculty of observation.

Necrosis in bone and malignant disease can both be discovered by the X-rays. I remember well my first case of myeloid sarcoma of the radius, in the case of a lady who had sustained a fracture of this bone a few months previously. The diagnosis first made was "excessive callus pressing on the nerves." An operation followed the skiagraphic examination, and, as far as I know, the patient is still alive and well—nearly seven years ago.

Since the discovery of the rays there has been an apparent increase in the number of cases of congenital dislocation of the hip. Many of these cases were looked upon as acute anterior poliomyelitis, and treated accordingly; especially, too, if the case were bilateral, and consequently showed little or no variation in the length of the limbs. Tubercular lesions of bone are often well seen; though I can safely state that if the lesion is minute, and early, it is generally difficult to make a definite diagnosis.

Before leaving the subject of skiagrams of fractures or injury to bones in particular, and to all skiagrams in general, it is necessary that, if such are to be produced in a court of law as evidence, they must bear some mark of identification. My method is to take, say, one yard of ordinary fuse wire, and work it with the fingers into some much-twisted shape, and, having taken the skiagram with the wire superimposed in one corner, to hand this "brand" to some responsible person for safe keeping. It would be impossible to forge such a "brand"; and, without some such precaution, it is quite impossible to identify to the satisfaction of the court that a skiagram produced is the one taken of the patient in question. I also have small letters and numbers cut out of lead, and place these on the envelope beforehand—R. indicates right, L. left, and so on. The date follows.

It is marvellous to think of the strides made by radiography in connection with stone work. In 1896 Henry Morris wrote in the *Lancet*:—"Owing to the position of the kidneys, close to the vertebral column, the depth of the cavity, and the thickness of the overlaying tissues, it has so far proved impracticable to obtain a radiograph of renal calculi." In June, 1898, Mackenzie Davidson, in a short note, asserted that he had been able to see a stone *in situ* on the screen. This is probably the first record of this having been done, though, not long afterwards, several of us here will recollect our first cases when renal calculi were detected.

Strange to say one of my first successful cases was one in which I discovered a small ureteric calculus. I published the skiagram in 1900, which, so far as I know, was the first one published, showing a ureteric stone; but since then I have only had one other case in which the calculus has been in the ureter. I still maintain that about 4 per cent. is a fair average to allot to cases of ureteric calculi, and the remainder are invariably in the renal pelvis. Leonard, of Philadelphia, maintains that 50 per cent. are ureteric. Perhaps

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no man in the world has done greater or better work in connection with renal calculi than Dr. Lester Leonard. He still adheres to the method he has used now for some years, viz., a long exposure with a low tube, using a high amperage, and he bases his negative diagnosis on the axiom "that in a negative possessing a differentiation in the shadow of tissues less dense than the least dense culculus, no calculus can escape detection."

In an interesting letter written to me by Dr. Leonard at the end of 1902, he states: "I do not claim to make the absolute negative diagnosis in all cases, only where the definition and contrast in the negative warrant it . . I, however, always tell very stout patients that they are difficult subjects, and that I may not be able to render an absolute negative diagnosis or to find a calculus."

About the same time I heard from Mr. Henry Morris, who, in the course of his letter, says: "In my opinion the Röntgen ray is a scientific toy in renal cases, and that our practice of surgery ought to be based upon other factors in every case," &c., &c. I think by now, probably, that he will admit that the X-rays are of the greatest service to renal surgery, for a negative diagnosis does not necessarily indicate that the patient should not be operated upon; it is still, I maintain, of the same value as a negative result in the case of examining for tubercle bacilli in the case of a suspected phthisical patient.

Then, again, I can quote five cases in my own experience where calculi have been located on one side, whilst all the symptoms were on the opposite side. Before the days of the X-rays we can all remember cases being operated upon for renal calculi and found—how often? Now it is the routine practice for any suspected case to be submitted to an experienced radiographer for an opinion; and rightly so, too.

The medical men who send their cases should themselves most carefully examine the resulting plates, and so gradually train themselves to interpret the shadows. By this means they will be in a position to give an opinion as good as the operator; and, indeed, this applies to skiagrams generally.

So far as my own experience goes, I have detected calculi in seventy cases, and they have been removed. I have made, so far as I know, twelve mistakes, and examined about 400 cases.

To Dr. Albus-Schonberg is due the credit of introducing a pressure tube apparatus through which the exposure is made for kidney cases especially. There are two advantages obtained by it. Firstly, by cutting off all but a small direct stream of rays, and by cutting off secondary rays, a much sharper picture is obtained; secondly, by the pressure of the tube, the respiratory movements are modified on the side being examined. This method I have tried, and can recommend it in other cases too, where a small area only is required—as in joint cases. Of course when the compression tube is used, only a small area can be exposed at a time, and if the result is negative, then subsequent exposures on different plates must be made. This is the great drawback to this method. I think, perhaps, it is better to expose as usual on a large plate, and to use the compression tube subsequently, if necessary. The exposure, too, is very different now to what it was. Whereas a good result in a suitable case can be obtained in fifteen seconds or so, a couple of years ago we would have given half an hour. The tubes have improved, and also the method of producing the rays; and, in this connection, I always feel deeply indebted to Dr. MacCallum, of Geelong, for having induced me to use a "Caldwell" interrupter. Even now at times I still use an accelerating screw. Judiciously used with a low tube, it is of great assistance. The heavy anodal tubes for the strong currents are very admirably made, and recently a water-cooled anodal tube has been placed on the market, which has many advantages.

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The majority of operators, when examining for stones in suspected cases, make the patient lie flat on his back with knees drawn up and head and shoulders slightly elevated. Others simply let them lie flat on their back.

For versical calculi the rays are of not nearly so much assistance as in the former cases, for these can usually be easily detected by other means. As for biliary calculi, one or two men claim to be able to detect them. I have my doubts, and should very much like to see the original plates.

Turning now to the medical aspect, it may be as well to recall to your memories a statement made by Dr. Mansell Moullin in his presidential address, in 1899, when he said, " I am convinced that, at no very distant date, the examination of a patient's chest with X-rays will be considered as much a matter of routine, and as little to be neglected in all doubtful cases, as an examination with the stethoscope at the present time." And he enumerated a long list of cases in which their value from the diagnostic point of view was very great; and this list can scarcely be added to at the present time. But, owing to the improvement in apparatus, and to the better recognition of what can be seen with the screen, and by the study of good plates, and to the more exact knowledge of the meaning of the differences from normal that can be seen and radiographed, his remarks as to the value of this method of diagnosis now carry more weight.

The fluorescent screen examination has proved of the greatest service in detecting aneurism in the thorax, and other mediastinal tumors. Pulmonary hydatids have been seen, and, in several cases, a diagnosis has been correctly made of a hepatic abscess having ruptured through the diaphragm into the lung.

It is generally admitted that a diagnosis of pulmonary consumption can sometimes be made by the screen before any physical signs are evident. Besides the dull areas seen, the most important point is the diminished excursion of the diaphragm in the affected side.

So far as the treatment of tuberculosis is concerned by means of the Röntgen rays, it is now a matter of much controversy. In this connection I feel I cannot do better than quote some extracts from the presidential address of Dr. Bullitt, delivered at the Congress of the American Röntgen Ray Society at the end of last year. My results agree entirely with his, in spite of most glowing accounts being published every now and again by some new man.

In tuberculosis of lymphatic glands the term " cured " can hardly be understood to mean the entire disappearance of the affected and enlarged glands. It rather means a diminution of the size of the glands, and a disappearance of all symptoms which indicate any activity of the disease process. In the comparatively few detailed reports which have been made of cases of this kind the statement has been made in all, that while the condition had improved and the glands had diminished much in size, they still were present as palpable enlargements.

Some men have published most startling accounts of the results of treating phthisis pulmonalis by means of the rays. In all the cases mentioned the usual hygienic treatment, rest, diet, &c., have been included, and no doubt they did well in spite of the rays.

To sum up the treatment of tuberculosis generally, I agree with the statement that, " while the reports are encouraging, and while the probability seems to be that this method of treatment may be shown to be of great value, the statistics obtainable are too uncertain and not sufficiently explicit to warrant such an assumption." And let us not forget that our effort and duty must not be to prove that the Röntgen method is of value in the treatment of tuberculosis, but that it is to ascertain the truth as to whether or not the method is of value.

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Passing on to diseases of the appendages of the skin, such as hypertrichosis, alopecia-areata, tinea tonsurans and Favus, sycosis, acne vulgaris and comedo, and inflammatory diseases of the skin such as eczema, psoriasis, lichen planus, lupus vulgaris, and erythematosus, prurigo, &c., we all know what good results have followed suitable X-ray treatment in these cases.

I must make special mention of the treatment of Keloid and hypertrophied scar tissue by the X-rays. Since I had the privilege of reporting the first case treated I have had many such cases, and if treatment be persevered with long enough, the cure is practically certain. Several severe reactions must be produced in every case.

Perhaps the most important consideration of the rays is in connection with the treatment of malignant disease. Further experience in the application of the X-rays to the treatment of rodent cancer, and other forms of superficial cancer, has tended, on the one hand, to clear up some points which were previously obscure; and, on the other hand, to raise fresh issues, upon which we still require enlightenment, and of which we have much to learn. There can now exist no doubt as to the beneficial effects to be obtained from scientific application of the X-ray in many superficial lesions, but we still have much to learn as regards the best methods of application and the proper dose to administer.

In many cases the X-rays act almost as a specific; but, like all "specifics." they occasionally fail to produce the desired result. Those few exceptions may, however, be taken as those that go to prove the rule.

An important question is that of recurrence in cases which, to all intents and purposes, have been cured, and this frequently after only a few months' freedom from distressing symptoms.

Mr. Malcolm Morris, in a paper read before the Harveian Society, has pointed out that the general principle of the up-to-date treatment of superficial skin lesions may be expressed in the single word "reaction." "Reaction," says Mr. Morris, "is a force which can work wonders if properly directed and controlled. In the present state of our knowledge we are but imperfectly able to control its operation. The clinical phenomena seem to me to warrant the belief that some part at least of the beneficial effects of the X-rays is due to the reaction which they excite." I think that most of us now hold this view: in fact, we generally, in treatment of our cases, produce a dermatitis of a greater or less degree, and thereby get some idea of proper dosage.

Dr. Hall-Edwards, in an admirable paper on this subject, in the *Journal of Medical Electrology and Radiology*, speaks of the advantages of a "soft" tube. I think I am safe in saying that these are not generally employed now in conjunction with a mechanical mercury break. With an electrolytic interrupter tubes become hard so quickly that the expense of procuring fresh ones would be endless. If "soft" tubes are to be used, so as to obtain their full effects, they must—even under the most perfect conditions—be constantly replaced by new ones, as no tube which has commenced to get "hard" can be again brought into its original condition unless it be filled with air and be then re-exhausted. A common plan often adopted—and I myself once was a culprit—is to keep the old tubes for therapeutic work. The results thus obtained will not bear comparison with those in which new tubes are employed. The vacuum of a tube can certainly be lowered by means of certain subsidiary tubes, which give off vapor or gas when the spark is made to pass through them. In these self-regulating tubes the vacuum may be lowered even past the point of "softness," yet they never produce again the results which were at first to be obtained from them. This, I think, is explained by

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Sir Oliver Lodge, who has pointed out that, in addition to X and other rays which come from an excited tube, a certain number of cathode rays pass through the glass, and these carry with them ions knocked from the atoms contained in the vessel. No matter how many atoms are admitted to replace the lost ions, the contents of the vessel will ever afterwards consist of a mixture of (what we may term) damaged and complete atoms, and the conditions which at first existed cannot be reproduced unless it be filled and re-exhausted. Some manufacturers assert that the same conditions can always be repeated by measuring the amount of current taken by the tube. This must be incorrect, for as a tube is ever and always changing from a "soft" to a "hard" condition, it must follow that one state cannot be maintained by any regulation of the amount or tension of the electrical current passed through it. Could we by any means maintain a given condition for even a limited time, we should be in a position to administer measured doses with at any rate some degree of certainty. There is no doubt that the measurement of the amount of current passed through the tube is of importance, and gives a much better idea of the condition of things than did the old method of measuring the amount of current taken through the primary of the coil.

To Dr. Deane Butcher we all owe a debt of gratitude for having introduced for discussion before the Röntgen Society of England a paper dealing with the means of accurate measurement in X-ray work. He pointed out that in X-ray work it is useful to distinguish three distinct individuals—the maker of the focus tube, the driver of the tube, and the user of the tube. Of the maker of the focus tube we may fairly ask that he shall give us —(*a*) A standard tube of definite size and make, with a certain definite resistance, measured in ohms: (*b*) Means of maintaining this resistance constant: (*c*) Means of measuring the resistance and varying it at will. Of the driver of the tube we require that he may be able to maintain—(*a*) A constant current through the tube: (*b*) A constant vacuum in the tube.

Lastly, the user of the tube requires only two instruments of measurement: one to assure himself of the quality of the rays, and another to measure their quantity.

So far there are several methods in use. Holznecht uses a capsule of gelatine, impregnated with a salt which turns to a deeper shade of green under the influence of the rays. This color is measured by comparison with a painted scale of twelve different tints. Holznecht's chromoradiometer is very expensive, and difficult to procure, and from time to time he alters the composition of the capsules.

Freund uses a 2 per cent. solution of iodoform in chloroform, which darkens under the influence of the rays.

Sabourand uses a portion of an ordinary fluorescent screen, with the same object.

Baroist has supplied a radio-chromometer to determine the penetration of the rays. This instrument is based on the fact that the relative transparency of silver and aluminium to the rays varies with the quality of the rays considered.

Dr. Leonard, in his work, employs a milliamperemeter, which can be placed in circuit with the tube. The milliamperemeter measures the volume of electric energy passing through the tube, and hence the proportionate volume of Röntgen ray energy given out. The increased volume which is shown to pass through low tubes, he states, corresponds with their greater photochemic activity, and also with their effect in therapeutic work.

Leonard employs the following formula in expressing his standard dose in the treatment of malignant disease:—10m, 8″, 2′g, 2ma; meaning ten minutes treatment, with the platinum 8 inches from the skin, the tube

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having an equivalent air resistance of 2 inches, and energised by a current of two milliamperes. Like any other algebraic formula, an alteration in any of its terms alters its value.

This attempt of standardisation is a step in the right direction; but most of these methods have been threshed out by French physicists, and it was decided that they were unreliable, because the inconstancy of the tube defied all efforts of measurement. Mr. Dean, of tube fame, attributes one cause of this to the fact of not employing pure platinum in most of the tubes on the market. There is certainly something in this; and I think that if more care were taken in the manufacture of tubes our results would be much better than they are.

Some enthusiasts have gone so far as to measure the glass broken from different parts of the globe of a tube, and found great variation in the thickness. Let us hope that, as time goes on, and competition becomes keener amongst the manufacturers, our tubes will be considerably improved.

In Australia our best results have been in cases of rodent cancer. This disease with us is very common, especially with the working classes. In America and on the Continent it appears that no distinction is made between rodent cancer and epithelioma, and nearly all the cases which we call rodent cancer here they call epithelioma: hence the cure of this disease is spoken of by our confreres as a simple process. Dr. Grubbe, when addressing the members of the Röntgen Ray Congress last year, defied the profession in Chicago to produce ten cases of rodent cancer there.

In the treatment of these cases of rodent cancer there is great variation, though the ultimate object is reached by many eventually. I generally follow the following line of treatment:—As a preliminary, a thorough curetting of the affected tissue with subsequent antiseptic dressing. Two days afterwards begin the X-ray applications, with a soft tube, at 8 inches distance to start with. Applications are daily for ten minutes. During the treatment I constantly paint the part with pure carbolic acid: this serves as an antiseptic, and closes up the lymph channels, as well as assisting to hasten the "reaction." I never employ therapeutic tubes specially made, nor do I use special tube-shields: these have been found to interfere with the proper passage of the current through the X-ray tubes, which rapidly deteriorate and become useless. I use any good make of tube, and protect the patient with several layers of tinfoil. They are instructed to spread some mild antiseptic ointment on china silk, and cover over the affected area. When a good reaction is produced the sittings are discontinued for the time being, and, if not cured, a second reaction is produced, and so on. Great care should be taken to include an area around the affected region, to be on the safe side. To show how necessary this reaction is, I may mention that two of my cases some few years ago developed cellulitis during treatment, and when they recovered the rodents were also cured. Before discharging a case, the part should be perfectly smooth. Lately I have been applying a solution of fluorescin to the part during the application of the rays, hoping thereby to shorten treatment, as well as intensify the rays.

My results, on the whole, have been very satisfactory, showing only a very small percentage of recurrences within the past six years. I do not think statistics should be published till such cases have been treated for ten years, and even then they are misleading.

One statement I wish to make, which I have not seen mentioned before. When any bony sturcture is attacked by rodent cancer, an operation should be insisted upon. I have seen such cases heal over, apparently cured, and later on break down again. This will happen every time. The probable explanation for this is that the curative rays are the soft ones, and the bone

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salts prevent them from acting on dense structures. So, no matter how little bone be affected, always insist upon surgical interference first, and subsequent ray treatment. I make it a rule of having every case examined microscopically, and often, to my surprise, I find that I am treating a case of superficial epithelioma. These respond just as well to ray treatment as do the rodent cancers, and operators all the world over agree on this point.

Glandular involvement should be carefully sought for, and, if present, nothing less than a thorough operation should be performed.

The treatment of sarcomata by the rays has not been so satisfactory, and though several successful cases have been reported, there is some doubt as to the diagnosis in these cases being correct, and also too little time has elapsed to speak with any authority on the success of such cases. My own results have been disappointing.

Passing on to malignant disease of the breast, one of the commonest malignant affections amongst women, I think we can justly and proudly claim that the X-rays have done very much good in the treatment of many such cases. Hundreds of such cases have been treated in various parts of the world, and the results throughout are good, on the whole.

There is some difficulty in considering seriously the numerous published results, for, as Dr. Bagg, of Pittsburg, says, "the Röntgen-ray workers may be classed under three heads: 1. Those who have made a careful study of electricity and physics, and are able to duplicate results, either in diagnosis and treatment. 2. Those whose work is chiefly confined to some other specialty, and who use the Röntgen rays without making any claim of having paid any particular attention to the subject. 3. Those whose work is done by head nurse, or some other inexperienced party."

All conscientious radiographers agree that no malignant case should be treated by the rays without previous consultation with and sanction of an experienced surgeon. I am afraid, however, that this rule is not always carried out—and, to be lenient, let us say owing to ignorance on the part of the radiographer. I do not consider that any malignant case should be treated by the rays unless pronounced to be inoperable, or as an adjunct to a surgical operation. Pre-operative applications of the rays are not justifiable.

In these days of asepsis, I think we should insist on every breast tumor being subjected to an exploratory operation, at least. Why, Dr. Halstead, of the Johns-Hopkins Hospital, in a systematic cutting of the entire tumor in twenty-eight cases of apparently benign breast tumors, found foci of malignant disease in them all! A careful study of the results obtained by Röntgen treatment in malignant disease of the breast shows that it has been most effective where it supplements, rather than supplants, operation—that is, where the focus of disease has been removed.

The Röntgen treatment has the advantage of being conservative and reconstructive, rather than destructive. It possesses prophylactic and palliative properties that make its first operative application of the utmost importance. It helps to reconstruct normal tissue, and hastens the healing of wounds. It can be applied immediately after opertaion in all cases without doing harm, and with the assurance that much benefit will be derived. It is the most potent palliative and prophylactive agent known in the therapeutics of this disease. In inoperable and hopeless cases, seen in the late stages of the disease, it affords relief from intolerable symptoms, lengthens life, and is the greatest chance that offers to the patient. That this is often very great is shown by many remarkable cases: patients given up as inoperable and hopeless have been restored to comparative health, comfort, and activity. In such cases local and constitutional treatment upon sound medical and surgical principles

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is absolutely essential to success. Often in these cases a toxæmia, so common in malignant disease, occurs, and manifests itself in rheumatic pains, biliousness, and gastric attacks, with a general increase of nervous symptoms. In some instances, in the early stages, it can be combated by vapor baths, provided the vitality of the patient will permit. Tonics and sedatives are of value. Quinine is of special value, for its tonic and antitoxic properties. Some operators give quinine for its fluorescent properties as well, and it seems only feasible to think that fluorescent solutions absorbed by diseased tissues should cause them to re-act quicker and better. A good plan is to prescribe the patient a pill of quinine, iron, arsenic, and strychnine, a careful diet, and to drink large quantities of liquids.

Other forms of malignant disease have not given such satisfactory results as these just mentioned. Practically every form of malignant disease has been subjected to the rays, but the results with the deep-seated ones have been doubtful. In most cases, at any rate for a time, a stimulating or tonic effect follows the treatment. This is accounted for by Dr. Snow as being due to the disposition of the vibratory influences of the rays to first overcome local stasis, restoring tone to the muscular coats of the arterioles, and at the same time inducing a more active local metabolism.

I think, gentlemen, this briefly describes the principal uses of the X-rays since their discovery ten years ago, and their progress—though hampered by a few unscrupulous workers—has been slow and sure. What the next decade will produce it is hard to predict, for just now even an altogether new subject, that of azoo spermia, is engaging the attention of scientists.

All these considerations make us ask the question—In whose hands should the Röntgen rays be? And I think unhesitatingly you will answer, in those of qualified medical men.

Let me close by quoting Dr. Thurston Holland in his presidential address before the Röntgen Society of England last year. He says: "Obviously it is possible for anyone to acquire the necessary knowledge to show, say, a fracture, or coin, or foreign body in the body; but even in these simple cases, if the application is to be made of the fullest use and mistakes are to avoided, a medical man skilled in the use of the rays must direct the examination: I would even go further than saying that a qualified medical man only must use the rays for medical and surgical purposes, and would say that he must be at the same time an expert in radiography. Even in the hands of medical men a caution is necessary. Large numbers, now, so to speak, 'dabble' with X-rays. They order a good apparatus, and do not trouble to get proficient at the work; as a consequence, their X-ray work is most inefficient, and when it comes to X-raying hips, giving an opinion on a chest case, or attempting the diagonsis of kidney stone, the results they show are quite worthless, and tend to bring X-ray work generally into disrepute amongst those physicians and surgeons who depend on others for this work. However, the use of X-rays for cases of this kind may be almost said to be unimportant when compared with their use for treatment; and yet, on all hands, we see unqualified persons using powerful apparatus in this manner. And yet who is to be blamed for all this? Primarily, medical men themselves, who have from the first encouraged instrument-makers, chemists, and generally non-qualified individuals by sending cases to them. The position of X-ray work, in my opinion, is this:—Any practitioner can set up an X-ray apparatus, and use it for his own special work—a surgeon for foreign bodies, fractures, and so on; a physician, for chest cases, &c.; a dermatologist, for the treatment of his cases. But there remains a field for the X-ray expert, who is ready and competent, to use the X-rays properly for any and every class of case to which they can be applied: and this man ought necessarily to be a qualified medical man."

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VICTORIAN SANATORIA FOR CONSUMPTIVES.—NOTES ON FIVE YEARS' RECORDS.

By R. Bindon Stoney, M.B., B.Ch.

Having at my disposal the records for several years, I thought I might be permitted a few minutes to summarise these case-books and see if any lesson could be derived from them. Our sanatoria consist of a summer establishment in the mountains of Macedon, where a few carefully picked cases stop on during the winter, and a winter sanatorium in the dry northern district of Victoria, right on the Murray at Echuca. It is of this latter I am medical officer, and have charge of forty-five to fifty patients from May to November in each year.

As to climate, Echuca is usually dry, and affords a maximum of clear sunshine. The nights are clear, and slightly frosty in the middle of winter; we are thus able to give the patients the greatest amount of light and fresh air with a climate that enables them to be out of doors as much as possible.

The rules of the institution are that only cases in the incipent and early stages are admitted, but, unfortunately, we get a good many cases sent to us from Melbourne that are anything but in the early stages, and most of these have to be sent home disappointed after a short residence with us.

A certain number of cases of miners' lung come to us, but are all practically hopeless cases. Another bad class are the undeveloped, rickety, strumous youngsters, who certainly improve with fresh air and good food *up to a certain point*, but never do us credit.

The history of a large number of cases that come under our notice is:—1st. A course of patent medicines prescribed by himself, his chemist, or his friends, that does no good. 2nd. A course of disinfectants, under the family physician, of creosote, guiacol, &c., trying to poison the bacteria in the lungs, but, instead, destroying all the natural ferments and vitality of his digestive organs; or he is saturated with cod oil and such like till his liver refuses to act properly. 3rd. He gets a course of tuberculine lymph, electricity, massage, or other so-called specific under the specialist, to the material benefit of the specialist.

It is difficult to say which of these courses is most harmful, as they give the patient the idea that he is being cured, yet daily his hope of recovery is getting less and less: finally, when he is thrown up by the chemist, family physician, and specialist, he is thrown on to the sanatorium as a last resort, or he goes to the incurable hospital, or dies at home.

All our experience is that it is exceedingly important that cases should be recognised in the incipient stages, and that no time must be lost. Time should not be wasted in purely medicinal treatment, or he soon reaches that stage when recovery is impossible. Of all the factors in the treatment of consumption fresh air and sunshine stand first, good nutritious diet and rest second, reserving medicine for complications.

During the five years under consideration, 1900-1904, 552 patients were admitted to the sanatoria. Now the question of real value to us is, what percentage of those *are cured to that extent* that, after several years, they are able to carry on their work and earn their own living *without difficulty?* How many are barely able to so do, and what number are totally invalided or dead? And, if possible, to see the condition of those who have done best, and what treatment has been carried out in their cases.

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With regard to death-rate. Of the 552 cases treated up to end of last year 247 are known to be dead, being 44½ per cent. of total. Or, taking them year by year :—

1900.....	66	cases—	39	dead,	59 per cent.
1901.....112	"	54	"	48	"
1902.....105	"	44	"	42	"
1903.....118	"	59	"	41	"
1904.....151	"	51	"	33	"

Showing that, after the first two years, the balance shows a much slower increase of death-rate. Of those alive 46 per cent. are well and able to do their work, 16 per cent. with difficulty struggle along, 26 per cent. are invalided, and of the balance, 12 per cent., we have no recent information. That is to say, of all the patients who have gone through the sanatoria in those five years, not quite 21 per cent. are now well enough to be able, without difficulty, to follow their daily occupation.

The next question is as to the conditions under which patients have done best: and great prominence has been put on the question of the patient putting on weight. A recent work states that a patient who increased a stone in weight was fairly safe. But what do we find? Take the year 1901 (the oldest we have weight records for), during which 112 patients were admitted to our sanatoria (fifty-six to each); of these sixteen put on a stone and over in weight during their residence with us, yet after five years what do we find? That 50 per cent. of these are, *to our knowledge*, dead, while of the whole 112 we know of only 48 per cent. of deaths. So that those who have gained over a stone in weight show a larger percentage of deaths than the rest of the patients; in more recent years, though numbers are not quite up to foregoing, yet the percentage of deaths among the stone and over is more than those who have put on from 5 to 14 pounds; and, on the other hand, the patients who lose weight, or do not put on at least 5 pounds, show during the four years the very large mortality of 62 per cent.—the totals being :—

One stone and over	85	cases—	23	deaths,	27 per cent.
Five to 14 pounds gained....149	"	35	"	23	"
Nothing to 5 pounds gained..131	"	77	"	60	"
Lost weight.....105	"	70	"	67	"
Weights not recorded 1900 .. 79	"	42	"	—	
Total.....552	"	247	"	44½	"

This is still more accentuated when we go through the cases individually, and we find those who have been best fitted for the struggle of existence are those who have put on 7 to 10 pounds, not those who have been fattened up like prize oxen for the slaughteryard.

It sometimes happens that those who are sent away home as hopeless cases nevertheless pick up strength, return to work, and are comparatively well; while, on the other hand, cases that appeared to have done remarkably well go to pieces as soon as they get out in the world again. For the latter the reasons very largely are, as far as I can see, that the patient has had such an easy lazy time in the sanatoria that his muscles are quite unfit for work. He gets among companions and drinks perhaps a little more than he ought, or, even where he takes every care of himself, the worries of this world and the fact of being tied down to his work, of having to do his daily round whether he likes or no, whether he feels fit or no, of weather and climate, and such like, which he has to put up with when he goes back to work, and a large number break down before they have been at home long.

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Weight lists are liable to a great variation, and it is a mistake taking a week's weight list by itself, neglecting those previous—taking each patient's gain or loss for the week. The aggregate weight increase will be most materially altered by one or two bad cases, or if one or more are in bed and not weighed, or if those who may be dismissed just before weighing day are losing or gaining fast. Another thing is that a sudden change in the weather will cause a general alteration in clothing, which, though slight in each case, makes a considerable difference in the totals. Another trouble is that patients are weighed once a week on a particular day after dinner, and if one week's dinner is not quite so tasty, or another a little more so, it may affect the weekly list. Hence, I prefer as long an aggregate as I can get—or, as I have done above, to take each patient's weight at arrival or departure—if we want a true idea of how things are going.

As to heredity. In 52½ per cent. we get a family history; this to the patient means probably a congenital delicacy with increased opportunity for infection. In many cases we have two or three out of one family. As far as we can see, they do not do as well as other cases; but it is impossible to separate the figures, as the family history does not necessarily imply heredity, as infection may have been acquired quite independently.

Temperature is one of the most important indications, and I feel inclined to classify them as:—1st. Very high and irregular temperatures, often showing an alteration of two or three days' rise and fall; look out for dysentery, tubercular enteritis, or other acute complication (hopeless). 2nd. More regular than the last, may show a tendency to a three or four day periodicity running up to perhaps 103, generally with streptococcal complication (very hopeless). 3rd. A two or three day rise, say to 101.5, no mixed infection (hopeful). The temperature in complications runs much higher often than symptoms seem to warrant; a bilious attack, a slight cold, may run it up two or three degrees.

Hœmorrhage, when slight, has but little bad effect on a case if we do not knock the patient up with starvation and bed. Severe hœmorrhages are very rare with us.

The mental aspect should always be carefully considered—plenty of amusement to interest them; and we must remember that if we give a patient no bottle medicine he goes away and says he was neglected. He feels so, and it does him harm. Give him something to treat his mind.

Mild and convalescent male cases, who are smokers, are, I think, all the better for their pipe in strict moderation, if it does not make them cough.

What then are the cases that have done best? They are cases where one, or at most two, lobes are affected; where there has neither been any very rapid increase of weight or the reverse—where the weight has slowly increased to the full weight of the individual and stopped there; when the patient is naturally of an energetic and hopeful disposition, and has been both hopeful all through and energetic as far as allowed as soon as any temperature he may have had was gone. He does not worry about the past, present, or future, but cheerfully takes his part in any work or amusement that is going on. These are the cases that, before they go out, harden their muscles by fresh air, cold water, moderate regular exercise, and good food, so that when the work of this world has to be undertaken they are ready and willing to take their share in the battle of life. The grumbler, the loafer, the flabby fattened up case, the case who has had so much rest cure that he never gets fit to work again—they go out, and soon the last case of those men is worse than the first.

I ask for the digestion that it be not ruined with antiseptics and irritants, that the liver be not over-loaded with oils and fats, that the muscles be not

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allowed to degenerate for the want of exercise, that the mind be not allowed to grow either apathetic, morbid, or introspective, for the want of rational interest and amusement. I ask that, while thinking of the tubercle bacilli, you forget not the diplococci, streptococci, the germs of rheumatism, influenza, etc., for it is on these other organisms that the prognosis and prominent symptoms of consumption are largely due.

A little less theory, a little more common sense; and remember always that when a patient leaves the sanatorium he has to take his part in the work-a-day world, or go under.

THE PRESENT POSITION OF THE SANATORIUM TREATMENT IN AUSTRALASIA.

By Arthur H. Gault, M.D., Lond.

The sanatorium treatment of consumption as far as Australia is concerned may still be said to be on its trial. Each of the larger States has a public sanatorium, but the palatial buildings of the old countries find no counterpart here: we have only small institutions—indeed, for the most part, colonies of cheap and temporary structures. For the working man a sanatorium is acknowledged to be the best place, but for the private patient the Australian practitioner is not yet convinced of its necessity. The general advice to the early consumptive is, to have as much fresh air as possible—to go for a change or sea trip, to reside in the north, or in a tent, and when these have failed try the sanatorium. It is hardly fair to judge of the value of this treatment under such conditions. I think I am correct in saying that the patient has been suffering from his disease for at least a year before, as a last resource, he comes under treatment in a sanatorium. The poor man works as long as he is able; the rich tries everything else before he yields himself to this treatment. The sanatorium enthusiast believes that he is adopting one of the most valuable therapeutic advances of the last century, and one destined to play an increasingly important place in the treatment of the disease, and he looks to it for the solution of the tuberculosis problem. It may seem strange that the death-rate from tuberculosis has not fallen in these States as it has in other parts of the world, the reason obviously is that the influence of this form of treatment has not yet asserted itself. I venture to predict that in another decade, when it has had a chance, the tubercular death-rate will decline here as it has done elsewhere. It is the duty of every medical man to make himself personally acquainted with the methods and results obtained, and thus be in a position to tender advice.

No doubt there is a strong prejudice on the part of the patients themselves; they think they could not stand the severity of the treatment—the exposure to draughts and cold, the over-feeding, the rigorous discipline. They say it would make them worse to be associated with so many suffering from the same disease as themselves; that life would be so monotonous away from home comforts and friends. All such fears are dispelled by actual experience. Everything is planned for the comfort and happiness of the patients, as well as their good. Patients are never allowed to feel cold—they take the food they like—there are no irksome restrictions. The presence of others suffering from the same disease, but so much better than themselves, acts as a continuous inducement to persevere. The quiet, non-exciting, yet, at the same time bright and cheerful, life of a well-conducted sanatorium affords conditions most favorable for recovery.

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There is no doubt that a climate like Australia makes it easier to live an open air life, and therefore do without a sanatorium. The winter is the time when the advantages of a sanatorium are the most obvious. We would consider Samoa a most unsuitable climate, and yet it was just the one that suited R. L. Stevenson, because it was one in which it was impossible to live anything else but an open-air life: and, no doubt, the improvement that takes place on going for a change is due to the fact that the patient is so much out of doors. Our climate ought to give us a great advantage in the after treatment of this disease.

Briefly let me describe the Australian sanatoriums.

1. PUBLIC SANATORIUMS.—New South Wales has adopted an excellent idea—men and women are to be treated in different institutions. Thirlmere is being transformed into a modern sanatorium for women, the original substantial three-storied building will have to be modified, the large wards for twelve patients divided, and something done to get over the disadvantage of stairs. Thirlmere accommodates about forty patients, and is under the care of Dr. McCredie.

Kings' Tableland Sanatorium, Wentworth Falls, is under the care of a resident, Dr. McIntyre Sinclair, and provides accommodation for forty-four male patients. The buildings consist of pavilion blocks built of wood connected by corridors, some single shelters, and some for five patients. At an elevation of 2,700 feet one would think the climate rather severe in winter; but as most of the cases are early ones, it does not seem to interfere with results. Dr. Sinclair is to be congratulated upon the large percentage of recoveries—equal to anything obtained elsewhere and testifying to the value of a resident physician.

Victoria in its provision for consumptives is also unique in having a winter and a summer residence. There is a good deal to be said for this arrangement, but it is unnecessary if a moderate climate be chosen. The extra expense could only be borne by a rich State like Victoria.

The winter residence at Echuca and the summer one at Mount Macedon have each accomodation for about fifty patients. The buildings are of a temporary character—wooden shelters supplemented by tents; but they seem to serve the purpose, and are quite insufficient for the number of patients applying. Echuca is under the care of Dr. Stoney, and is looking out for a larger site. Mount Macedon labors under the disadvantage of having no medical man in constant attention.

The new Government Sanatorium at Greenvale has only been opened a few months. Seven tents have been erected to hold five patients each. More permanent wooden buildings are in course of construction.

Queensland is well supplied by the Jubilee Sanatorium at Dalby under the care of Dr. Stewart. The building is a simple one. more like a large verandah, but the results seem equal to any. Dalby provides accommodation for forty patients. Dr. Stewart has recently investigated the after history of patients discharged as cured. Most have gone back to their original occupations, and, in spite of the unsuitableness in many cases, have maintained perfect health.

The New Zealand Government has recently opened an important sanatorium at Cambridge, with accommodation for fifty-eight patients, under the care of a resident, Dr. Pentreath. This sanatorium is splendidly situated, and has a large area of land (1,000 acres) in connection with it. The buildings are of wood for six, two, or one patient, chiefly single rooms. There is a separate colony of shelters for paying patients. We have no doubt the advantage of an experienced resident doctor will make itself evident in the excellence of the results.

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There is also a small sanatorium at New Brighton, the Nurse Maude Camp, providing accommodation for twenty patients in separate shelters and tents. In addition to this, the general hospitals at Wellington, New Plymouth, Blenheim, Invercargill, and Waimate have annexes for consumptives, from which suitable cases can be drafted to a sanatorium. Now that hospitals do not admit consumptives, this is an arrangement which ought to be adopted at all such institutions.

South Australia has the Kalyra Sanatorium, Belair, with fifty-three beds, erected and largely maintained by the estate of the late James Brown. The buildings are all of stone of a substantial character. The three new wings are built on Nordrach lines with single rooms.

Western Australia has no sanatorium, but there is a small annex to the Perth Hospital.

Tasmania: A similar arrangement is in contemplation for the Hobart Hospital.

There is great need in all the States for colonies to provide suitable occupation for those in whom the disease is arrested.

II. PRIVATE SANATORIUMS.—*New Zealand*—Avon Pine Sanatorium, Christchurch, under the care of Dr. Greenwood; for eighteen patients. It consists of isolated wooden shelters and tents, arranged in a colony. Flagstaff Sanatorium, Dunedin, under care of Dr. Stephenson; with accommodation for fourteen patients. The building is of wood, with central administrative block connected by covered-in corridors with the wings, which are composed of single rooms.

Queensland—None.

New South Wales—Leura, Wentworth Falls, a small home, under the care of Mrs. Robison.

Victoria—Belle Vue Sanatorium, Yackandandah, under the care of Dr. Johnson; for twelve patients. Buildings of wood, isolated shelters for each patient.

South Australia—Nunyara Sanatorium, Belair; with accommodation for thirteen patients. The building is of stone, with central administrative block and converging wings for male and female patients connected by verandah, and facing due north.

In comparing the various sanatoriums we would like to note a few points as to—

Altitude.—The favorite elevation seems to be about 1,000 feet: King's Tableland is, however, 2,700 feet, and Dalby 1,500 feet; Greenvale is only 600 feet, Echuca 400 feet, while Avon Pine and the Nurse Maud Camp are practically at sea level.

Aspect.—Generally N., N.E., or E. In the southern parts of Australia I am strongly convinced that the main building should face due north, with the wings closing in somewhat: this gives the full advantage of sunshine in winter, and affords shelter from winds. In rooms facing east the morning sun must disturb the patient's rest in summer.

Building Material.—As a matter of economy, wood has generally been chosen; but, if the building is to last any time, I think stone will be found cheaper, while it has the advantage of equalising temperature. Some sanatoriums have tents and shelters of a very light description, but, if the other States are like South Australia, strong winds must make them at times uninhabitable. Thirlmere is built of stone, but its three stories are quite out of keeping with Australian tastes. The Belair Sanatoriums are also of stone: the tiled roof of Nunyara is picturesque, and invaluable for mitigating extreme summer heat.

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Rooms.—In the older institutions large wards are provided, and, for the sake of economy, some of the newer ones have rooms for four to six patients; but I think that every patient should have a room to himself. Verandahs, or their equivalent, are found in nearly all: the seclusion insisted on at Nordrach does not seem to suit Australian tastes. Artificial heating is, for the main part, not attempted, rugs and hot-water bottles being relied on. Open fires and stoves are found at Thirlmere, Cambridge, Avon Pine, and Kalyra, in dining and recreation rooms; but are seldom used. Nunyara has a complete system of hot-water radiators, with hot and cold water laid on to each bedroom. Lighting is generally by kerosine lamps. Cambridge has electric light, and Nunyara acetylene gas.

Drainage.—King's Tableland, Cambridge, and Nunyara have septic tanks; the rest more primitive methods of sewage disposal.

Treatment.—There seems a general disposition to avoid anything in the way of specific treatment, and to rely solely on simple hygienic measures. Even cod liver oil is never seen in most of the sanatoriums. Systematic inhalations are used in one institution, while two indulge somewhat in oil-massage.

The question of temperature is a most important one, but would require a paper for adequate discussion. The oral temperature is accepted as sufficiently accurate in all except King's Tableland and Greenvale, where the rectal temperature is recorded. It seems agreed that a temperature of 98°.8 in the morning, or 100° at night, necessitates absolute rest. The question of rest and exercise is, after all, the most vital to success, and one which requires the constant supervision and matured experience of the medical superintendent. It is in this matter, above everything else, that home treatment errs on one side or the other: it is very difficult to get people to realise the importance of this matter; one so frequently comes across patients with active disease who are practising deep-breathing, and thereby unknowingly doing their best to extend their trouble. When active disease is present, the quieter the lung can be kept the better, provided that perfect oxygenation of blood is taking place, The degree of lung movement varies with the amount of tidal air, and I have been experimenting to find out how much this is affected by different circumstances. It is very difficult to measure tidal air; but, by trying the experiments on different patients who had no idea of the nature of the experiments, I think our results are fairly accurate. I might summarise thus:—

Normal tidal air	500 c.c.
Patients walking gently up moderate incline ...	700 c.c.
After resting for half an hour	400 c.c.
Patient walking gently on flat	500 c.c.
After resting an hour	300 c.c.
Patient with limited lung disease, but at absolute rest on account of elevated temperature .	150-200 c.c.

We must remember that laughing, coughing, or forcibly inhaling disinfectants cause a great increase in chest movements, and ought to be limited as much as possible.

Dr. Haldane has pointed out that the effect of breathing impure air is simply to lead to deeper breathing, and, in healthy persons, he suggests that this may be really a benefit: I disagree entirely with Dr. Haldane's conclusions; but his facts are undeniable. Impure air causes deep breathing; the perfectly pure air of a sanatorium, combined with perfect rest when lying down, enables a patient to reduce the amount of tidal air, and therefore the chest movements to a minimum. As the disease becomes quiescent, very slowly and gradually, and under constant supervision, the patient is

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permitted to increase the respiratory movements up to the normal. I think I am right in saying that this is a process which can never be properly carried out except in a sanatorium.

In this connection I might say something about *Recreations* and *Amusements*. It is absolutely essential for success that a patient's life be non-exciting, yet at the same time bright and varied. Walking is by far the most important form of exercise, and its pleasurableness depends very much on the variety and picturesqueness of the walks. Croquet is allowed in most places for the good patient. Motoring forms an important alternative at one sanatorium. Games—such as draughts, cards, even billiards—and music are permissible, if they can be carried on without excitement. At one institution private theatricals are indulged in; but I think that is too risky.

In conclusion, I would like to bring before your notice the desirability of agreeing to a unifom system of classification. Statistics are misleading: all classifications are open to objections. But, for public reports, we must have both. One classification consists of four classes, according to the number of lobes affected: Another, Class I., good cases; Class II., hopeful cases; Class III., cases which might be benefited, but cannot expect complete recovery; Class IV., unsuitable cases. Dr. McIntyre Sinclair divides his cases into Early, Intermediate, Advanced. While the generally accepted Turban's classification—Stage I., slight affection, not more than one lobe or two half-lobes; stage II., slight affection of not more than two lobes, or severe affection of not more than one; stage III., all beyond stage II. "Class," I think, is a better word than "stage," and four classes give more latitude, without being cumbersome. I therefore propose a modification of Turban's.

Proposed Classification.

Class I. Incipient. Infiltration of one or both apices. Closed. No bacilli. With or without hoemoptysis.

Class II. Cases in which not more than one lobe is involved, or half of two lobes. Limited softening.

Class III. Cases in which not more than two lobes or equivalent amount, or cases complicated by superficial disease of larynx amenable to treatment.

Class IV. More extensive disease than class III., or such as are complicated with deep laryngeal trouble, intestinal disease, or persistent albuminuria.

DISCUSSION ON USE OF TUBERCULIN.

Dr. Gault thought that tuberculin might be more extensively used in sanatoriums, for the purpose of confirming diagnosis in doubtful cases. As for treatment, it was admitted that tuberculin was only successful in early cases. So long as sanatorium treatment gave such excellent results (80 per cent. to 90 per cent.), in such cases it was not likely that tuberculin would be much used for treatment.

Dr. Springthorpe congratulated Doctors Stoney and Gault on the practical papers which they had brought before the Section. Sanatoria met many of the constitutional requirements of tuberculosis in a certain number of cases, but only in a general and not in a specific manner. The underlying principles could be applied without sanatoria, and the specific antagonist was still wanting. Tuberculin was useful not only for diagnostic purposes, but also as a specific to help in building up a systemic resistance, if properly applied, in suitable cases, and many sanatoria cases were so suitable. The mixed infections, however, were probably so varied that the secret of individual immunity might depend upon antitoxins produced after manipulation

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of the individual sputum. It was the constitutional factor that required strengthening, rather than the bacterial that required destruction. The healthy were practically immune so long as they remained healthy: hence the value of everything that promoted healthy existence. Even if the tubercle bacillus were exterminated, there was probably some related organism that would develop some new disease under the new conditions.

THE PROGNOSIS OF PULMONARY TUBERCULOSIS.

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I propose dealing shortly in this paper with some points in the prognosis of pulmonary tuberculosis. Someone has said that the wise physician will not give a prognosis in this disease; but, unfortunately, it is impossible to deal with a sanatorium full of consumptives without attempting a forecast of each case for one's own satisfaction, if not for that of the patient and his friends. The disease is so variable in its course, so full of surprises, and the symptoms and complications open to so many interpretations, that one might well hesitate on the threshold of such a paper. I need make no apology if I do not attempt to treat the subject exhaustively. I think it preferable to pass over altogether, or in a few words, points on which we are more or less agreed, and to refer more particularly to other factors in prognosis regarding whose influence more or less difference of opinion may be expressed.

The fact that tuberculosis is absolutely curable has been fully established by *post-mortem* evidence. Pathologists daily find healed tubercular lesions in persons dying from diseases other than phthisis, and the records vary from 7.5 per cent. to 38.8 per cent. Osler has recently given it as his opinion that no one reaches old age without having a focus of tuberculosis somewhere, and he quotes the researches of Naegeli as establishing the truth of this contention. The latter observer made a special study of this question in a series of 500 *post-mortem* examinations. Every organ of the body was investigated, sections made of each part, and particular attention devoted to an inspection of the individual lymph glands. Tuberculous lesions were found in 50 per 100 of the bodies examined to the age of 15, 96 per 100 at the age of 18, and by the time the fortieth year was reached a tuberculous focus was found in every body. It is evident from this and other researches that tuberculosis is recovered from—or at least remains latent—in a very large percentage of the population, and that only a comparatively small proportion of those affected actually die from this disease. These records prove that the majority of the human race possess sufficient powers of resistance to cope successfully with the bacillus when once it has gained access to the tissues. In many of the cases under consideration the lesion remains practically insignificant, and the person attacked does not arrive at that stage in which definite symptoms of disease are presented. The remarks in the remainder of my paper will, for the most part, be directed towards that other less fortunate section in whom the bacillus has for the time being gained the ascendancy, and has produced sufficient constitutional or local signs of disturbance to bring the case under the notice of the physician. Unfortunately, in this group the prognosis is not so roseate as one might be led to infer from the more resistant group of Naegeli, to which I have just drawn your attention.

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Generally speaking, we may assume at the outset that two factors determine the fate of the victim attacked—the virulence of the bacillus, and the natural resistance of the individual. These two factors are so closely related that it is somewhat difficult to dissociate the influence of the one from the other. The development of virulence in a particular bacillus will, I think, be to a large extent determined by the nature of the soil on which that bacillus is implanted. The resisting power to tubercle markedly varies in animals of different species; it also varies, though to a lesser degree, in animals of the same species, and even of the same family. It seems somewhat strange that the influence of family hsitory on the prognosis of pulmonary tuberculosis still remains a most debatable factor. Most authorities affirm that family history has an unfavorable influence, not only on the incidence of the disease, but also on the prognosis of any particular case once the disease has developed. Others use the same series of figures to prove the opposite contention, that children of the tuberculous inherit an immunity against the disease. As a supporter of inherited predisposition, Turban goes further than most writers, and argues not only for inheritance of a general predisposition but for the inheritance of a specially vulnerable spot, a *locus minoris resistentiæ*, as it is termed. For instance, he relates a series of cases which have been under his observation where, in certain families, there was a tendency for the disease to begin at one particular apex, and to run a very similar course; and, in support of his theory, mentions the somewhat analagous case of Brehmer's, in which a mother and her four eldest children all developed lupus on the same cheek.

Since reading this paper a few years ago, I have particularly watched a number of cases of pulmonary tuberculosis in members of the same families, and, while I have seen occasional cases to support the above theory, I have been unable to convince myself that it is anything like a common occurrence. After all, it is the usual thing for pulmonary tubercule to begin at one or other apex, and it should be no difficult matter to find cases in one family with the disease commencing at a similar spot. Not long ago I had a patient under my care with ulceration of the epiglottis, and tubercular infiltration of both cords; his father and an elder brother both died from tuberculosis of the larynx, and in all three cases the lung symptoms played a subordinate part. This group is the most definite one I have observed to support the theory of *locus minoris resistentiæ*, but such cases are not seen every day. If I might express an opinion on a very much disputed point, I should say that I think hereditary predisposition has in the past been considerably over-rated, and that it has often led to gloomy prognostications not always warranted by the facts of the case. I think we must all admit having seen families where one member after another developed a virulent type of the disease, and rapidly died out. Such cases leave a strong impress on one's mind; but, in looking over a large number of cases treated in sanatoria, I have found my percentage of arrests almost as good in those with as in those without family history. Conversely, I have looked up the records of the most virulent cases I have treated during the last few years—cases which progressed rapidly to a fatal issue without the slightest response to treatment—and I have been interested to find that in a number of these no trace of tuberculosis could be found on either side of the family tree. Provided the other features are satisfactory, my own rule is to let family history take a very subsidiary position in estimating the prognosis of any given case.

Reverting to the subject of the tubercule bacillus and its virulence, it would be interesting if one could connect a particular type of bacillus with a certain type of pulmonary disease. So far as I am aware, not much attention has been given to this aspect of the subject, though no one can make a routine examination of the sputum without frequently noting morphological variations

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in the tubercle bacilli present. I believe it was Fränkel who first expressed the opinion that the small type of bacillus was more frequently associated with the more acute processes of tubercular lung disease; and I am inclined to agree with this observation. The length of the tubercle bacillus may be generally stated at 1.5 to 3.5, or 4u.; but considerable variations are not infrequently found. A year or two ago I made observations in a series of cases, and found the bacilli varying from short slender uniformly stained rods of 1.5u. or even less in length to the larger thread-like beaded bacillus of 8 or even 9u. in length. The former short variety I have usually found in more acute processes, and, when present, is frequently in large numbers—suggestive of rapid proliferation. I am also of opinion that congestive attacks, or lobular or even lobar extensions, of the disease are more commonly associated with the short slender type of bacillus than with its long thread-like prototype. The latter form is generally associated with the more chronic or fibroid types of the disease, and, from a prognostic standpoint, may, I think, be regarded less unfavorably than the short slender variety. Occasionally one finds branched and clubbed forms, sometimes regarded as involution or retrograde types of the bacillus; these are less acid-fast than the ordinary rods, and are therefore less seldom noticed in routine examinations of the spit. Some years ago Ransome asserted that tubercle bacillus could grow outside the body on sterilised filter or wall-paper, and that under those conditions it had a tendency to take on a thread-like or branched appearance, suggestive of the mycelial formation. Recent researches tend to place the tubercle bacillus among the streptothrices, and the involution forms met with in sputum may be regarded as a reversion to a saphrophytic or alternate type of existence. Rabinowitsch, Moeller, and others have demonstrated the fact that acid-fast organisms are widely distributed in nature—for instance in butter, in timothy and other grasses—and it is a probable speculation that the first case of pulmonary tuberculosis arose from one of these acid-fast saphrophytes becoming grafted on a weakened lung and assuming the parasitic type.

The statement that the long thread-like bacillus of pulmonary tuberculosis is of less grave import than the short slender variety must, however, be taken with certain reservations. Thus I have seen cases where the large type bacillus was present, and the lung apparently making good progress to recovery, when the development of a renal, meningeal, or other tubercular complication hastened the terminal issue. These observations appear interesting in view of the recent research by Moeller into the virulence of cultures derived from various human sources of tuberculosis. I need not refer to his work in detail further than to say that of the organisms examined he placed the tubercle bacilli from lupus as of greatest virulence, and next in order of virulence those from miliary tuberculosis, sub-acute pulmonary tuberculosis, chronic pulmonary tuberculosis, and fistula in ano. Taking these researches in association with the clinical observations I have just mentioned, I think it possible that a bacillus of retrograde type in the lung may develop a much higher grade of virulence when mobilised and translated to some other organ in the body. Such accidents would, of course, upset any prognostic calculations based on the type of bacillus present in the lung discharges. They also illustrate the difficulty of arriving at any certain prognosis from any one single factor, or even combination of factors, in an insidious treacherous disease like pulmonary tuberculosis.

I do not propose dwelling on the prognostic significance of the sputum further than to say that the number of bacilli present is largely a matter of discharge, and forms no certain criterion of the ultimate issue of any case. In the earlier stages of the disease the discovery of large numbers of tubercle bacilli on repeated examination must be regarded more seriously than where the numbers are few; but, in the later stages, large numbers of bacilli may

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be discharged from a cavity which is fairly quiescent and circumscribed: and, indeed, these organisms frequently continue to be discharged for years in cases which are otherwise apparently doing well. In cases approaching quiescence a diminution in the quantity of sputum discharged is also worthy of note as of very favorable import.

Probably no factor is of greater prognostic significance in this disease than the condition of the circulatory system. A good deal of discussion has centred round the question whether the average subject of pulmonary tubercle has a small heart. Whether this be so or not, it is at any rate certain that a low blood pressure is a frequent concomitant of the disease, and that the rapidity and tension of the pulse may be taken as a most valuable aid in estimating the probable results of treatment. I always feel hopeful about a case whose pulse at rest is under 100 and of fair strength. Even where the disease has proceeded to localised cavitation, a pulse rate of 70 to 80 may determine a favorable issue. On the other hand, cases of even early disease with a rapid dicrotic—or, still worse, hyperdicrotic—pulse of 120 or over will as a rule go progressively downhill. I have never known a case of pulmonary tuberculosis, with a pulse rate of 140 regularly at rest, to recover: I know such recoveries are recorded, but I am satisfied they must be extremely rare. In the malignant type of case the pulse is generally of a soft rapid character. Such a pulse may represent a naturally feeble circulation in the subject of attack, or it may be an indication of extreme toxæmia resulting from a virulent infection. In either case the outlook is serious. Naumann studied 100 cases of tuberculosis, uncomplicated and without fever, in order to determine the blood pressure of such subjects. In sixty-nine the blood pressure was over 130 m.m., in thirteen cases from 115 to 130 m.m., and in eighteen under 115 m.m. These figures may be considered high, normal, and subnormal values. I have been unable to make an exact comparison with Naumann's figures, since most of the cases admitted to King's Tableland have some slight pyrexia; but, so far as my observations go, I think Naumann's records in the tuberculous subject distinctly too high. Our cases, which are mostly in the early and intermediate stages, are selected by the medical references on the grounds of their possessing a reasonable hope of recovery; and, as a matter of fact, 54 per cent. have proceeded to complete arrest of the disease, while all but a small percentage have improved to a greater or less extent. With such patients I have found it very much the exception to have blood pressures exceeding normal, and most of them are the very reverse. Of the last 100 consecutive cases the blood pressures on admission worked out at seventy cases subnormal, twenty-six normal, and only four above normal. The figures varied from 75 Hg. m.m. to 136 Hg. m.m., and in the 100 cases the average blood pressure was only 107 Hg. m.m. In twenty-five uncomplicated cases of moderately early disease, and with evening pyrexia not exceeding 99°.6, and in all of whom the disease proceeded to apparent recovery or arrest, the blood pressure averaged 110 Hg. m.m., or of a slightly subnormal value. My results are therefore contradictory of Naumann's, and are more in accordance with the general accepted clinical observation, that low tension is one of the commonest symptoms in pulmonary tuberculosis. The observations in my series of cases were taken on a Martin's mercurial hæmo-dynamometer. I look upon blood pressures under 100 Hg. m.m. as generally bad from the prognostic standpoint, with, of course, a corresponding improvement in the outlook as the values approach normal. I need not pursue the subject at present further than to say I have not observed any essential relationship between the recorded blood pressures and the extent of lung involved.

The temperature in pulmonary tuberculosis is a valuable indication of progress, and of considerable prognostic significance. Like other factors,

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the temperature chart is open to different interpretations, and I shall only attempt to deal with a few outstanding points. Generally speaking, apyrexial cases are more favorable than pyrexial, and the more marked the pyrexia the less favorable the outlook. Occasionally, however, one sees exceptional cases of advanced progressive disease in which the temperature remains persistently subnormal. In these cases there appear to be deficient powers of reaction, and the prognosis must be founded on the physical signs and general condition, rather than on the degree of temperature present. In cases of marked pyrexia one should carefully watch the patient for a time before pronouncing an adverse opinion; under proper treatment such temperatures may subside in the course of a few weeks. In other cases the pyrexia is the indication of a fulminating type of disease, which progresses rapidly to a fatal termination. Koch and his pupils have taught that temperatures in pulmonary tuberculosis exceeding 100°.4 (38° C.) are due to the presence of mixed infection, the more usual complicating organisms being the streptococci, pyocyaneus, tetragonus, staphylococci, and the bacteria of influenza. The steep hectic fever, chills, night sweats, and rapid wasting characteristic of the later and terminal stages of pulmonary phthisis are attributed to the action of these organisms. The presence of pyrexia due to such infection would of course add gravity to the prognosis of the case involved. The subject of mixed infection is one on which considerable diversity of opinion exists, and it would be beyond the province of this paper to enter on a consideration of the various conflicting views: I would only say that, while acknowledging the occurrence of mixed infections and their gravity from the prognostic standpoint, I am also satisfied that pyrexia exceeding 100°.4 may often be due to processes of a purely tubercular character. Conversely, one will sometimes find cases in which foreign pathogenic organisms are present, but which nevertheless continue to run an apyrexial course from beginning to end. In the latter event the organisms are no doubt in a latent condition, and, comparing my experience with that of others, I think it probable that such latency is a more common occurrence in an open-air sanatorium than under the ordinary conditions of practice. No doubt the subsidence of temperature resulting from sanatorium treatment is in many cases due to the fact that a mixed infection has been reduced to a purely tuberculous process, with a corresponding improvement in the prognostic outlook.

There are other studies in connection with the temperature chart—such as the relative values of oral, axillary, and rectal temperatures; the significance of "exercise" as compared with "rest" temperatures; and the interpretation of subnormal, inverse, and other types. It would take up too much time to consider these fully, though they must all be of interest to those engaged in this class of work. I would only say that a careful record of the temperature chart enables one with considerable certainty to follow, day by day, the progress of the disease, and to anticipate, or possibly avoid, extensions and complications of various kinds. In the event of intercurrent complications, resiliency of temperature is a point of good import—the term "resiliency" being applied to the degree of ease with which an elevated temperature curve subsides to normal after the presence of some disturbing influence.

One of the most promising fields for research on prognosis and treatment is opened up by the recent researches of Leishman, Wright, and Douglas on the opsonic indices of blood serum. Modern studies on immunity show that the phagocytic effect obtained when bacteria are introduced into the blood is dependent upon an action exerted by the blood fluids directly upon the micro-organisms. This action, which prepares the micro-organisms for phagocytosis, has been termed by Wright an "opsonic" action, and a method has been devised whereby the opsonic index of the blood serum of any indi-

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vidual may be expressed in terms of almost mathematical precision. Taking the normal index as unity, Wright and Douglas have found that the indices in cases of pulmonary tuberculosis may be as low as .3, or even less, and that generally speaking the opsonic indices in the various forms of tuberculosis are considerably subnormal. It has also been found that the injection of certain vaccines may be followed by an increase in the protective substances in the blood with increased phagocytosis, and, of course, a more favorable prognostic outlook. The vaccines used in Wright's research on tuberculosis were the old and new tuberculins (T.R.) of Koch. The injection of an overdose of vaccine will be followed by a negative phase, in which the protective substances in the blood are decreased after inoculation; and this untoward result is said to be due to an overtaxing of the machinery of immunisation. In the course of treatment the endeavor would of course be to produce a positive or favorable phase, and to avoid the production of a negative phase. By a series of successive inoculations, Wright has shown that one positive phase may accumulate on top of another till finally complete immunisation is the result.

Bulloch has followed up Wright's work on similar lines, and agrees that the opsonic indices are, as a rule, low in tuberculosis. He found 65 per cent. of lupus cases had an index below .8, and, in a general way, there was a correspondence between low opsonic index and a failure to cure by means of the Finsen light. In active tuberculous bone disease the index was also low. In pulmonary tuberculosis the results appear somewhat confusing. In this disease Bulloch found the indices very variable, figures being often obtained considerably above the normal, while a large number of cases of "arrest" or "cure" of early pulmonary tuberculosis showed an opsonic index considerably below the normal. The importance of these researches from the points of view of treatment and prognosis is very apparent, and further work on the same lines by bacteriologists in association with the clinical physician would appear to promise results of much value in the near future.

I feel that this paper has already exceeded the limits I intended at the outset. You will notice I have not referred to many material points in prognosis—such as the factors of nutrition and assimilation; to the physical signs and area of lung involved; to the graver prognostic outlook presented by such complications as diabetes, alcoholism, syphilis, tuberculosis of the larynx, meninges, intestine, genito-urinary tract, or evidence of disseminated tubercular disease. If I have not considered these, and other factors, it is not from any want of appreciation of their significance. In forming a prognosis in pulmonary tuberculosis one has to consider not only each individual factor present, but to take a comprehensive view of the whole case. In the present paper I have simply attempted to outline a few outstanding points, some of more or less debatable value; and I trust the consideration of these has been not altogether without interest to the members present.

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RADIUM AND ELECTRO-THERAPEUTICS IN THE TREATMENT OF DISEASES OF THE SKIN.

By Herman Lawrence, M.R.C.P., &c.,

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Ladies and Gentlemen—Since we met at the last Medical Congress, radium has, so to speak, come into the world, and with its discovery science has received many shocks. This new element has proved itself to be of no small importance in the matter of the treatment of certain skin diseases. Likewise during this time the wonderful powers of radio-therapy, actino-therapy, and the high frequency currents as applied to diseases of the skin, have become recognised; and as I have had unusual opportunities for the application of these methods, I have chosen this subject for my paper. When radium was discovered it was stated that there could be very little of this new element in the substance of the earth. Certainly this prediction seems to still hold true, for specimens of radium—at least, of any therapeutical value—are still practically unattainable, out here. You are aware that pure radium, being so great an oxidisable element, has not yet been obtained in a pure state. The radium salts, the chlorides and bromides, which are used therapeutically are constantly emitting three varieties of rays, called the A, B, and G rays; these rays are considered to be produced by the spontaneous disintegration of its molecular parts. In my specimens the radium is encased in little aluminium boxes, the aluminium being one hundredth of an inch in thickness; this covering cuts off the A-rays. The A-rays are charged with positive electricity and have very little penetrative power, being stopped by a sheet of paper, they are of little value therapeutically. The Beta rays, charged negatively, have much greater penetrative power; and the G-rays, which are supposed to resemble the X-rays, have still greater powers of penetration. The therapeutical value of a specimen of radium depends upon the penetrative power of its Beta and Gama rays. I have here thirty-five milligrammes of radium, and will now exhibit a radiograph taken through a partition wall separating one bedroom from another. The radium was being placed near the wall in one bedroom and some steel instruments placed upon the other side of the wall—that is, in the next room—then a photographic plate was suitably supported behind the instruments. The rays from the radium, passing through the wall, produced a shadowgraph of the steel instruments upon the photographic plate. As I have an electroscope handy, I will also demonstrate the power the radium has of rendering the air near to it conductive of electricity, that is to say, when I charge the electroscope—whether it be with positive or negative electricity—the radium has the power of rendering the air capable of conducting away the electrical charge from the electroscope.

I now charge the electroscope with positive electricity, by rubbing glass-rod and silk together, and you notice the radium, even at the distance of several feet, quickly causes the charge to escape; and, likewise, when I charge it with negative electricity, the radium still causes the charge to escape. This is a very delicate test for radium, and was used by Mdme. Curie for separating out the radio-active substance from crude pitch-blend.

The Effect of Radium upon Micro-organisms.—Some write positively as regards the bactericidal effects of radium; others, however, have not obtained any definite results. I exposed a mould, taken from a decaying strawberry

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and grown upon agar for the purpose of this experiment to radium for two hours; this specimen was then placed in a fresh sterile tube of agar, and it grew apparently without having suffered any injury.

The Therapeutical Effect of Radium.—When radium was discovered it was thought, on account of the wonderful penetrating powers of its rays, it might prove of value in the treatment of cancer. This hope, however, was not supported by practical results, and the value of radium as an agent in treatment was then altogether belittled, for it has since proved itself to be one of the most potent therapeutical agents ever yet discovered. Your radium specimen is always ready for use—it cannot get out of order; whereas, with the X-rays the amount of attention necessary for their successful application is never ended. Personally, I combine the treatment by radium with radio-therapy or actino-therapy, as I find it convenient or necessary to do so. In extensive or ulcerative cases of lupus I apply the X-rays, and when the case is, so to speak, toned down, but with the so-called yellow spots markedly in evidence—especially so when the part is pressed upon with the dermoscope—I then apply the radium to these spots, and it has proved itself of veritable use just when the X-rays seem to have ceased to be effective. Many rodent ulcers have healed fairly quickly under the influence of radium alone. Warts, which have proved intractable with the usual applications, have disappeared when the radium has been applied to them. Lupus-erythematosus, more particularly the chronic discoid patches, have cleared up, leaving a scar such as the disease itself would probably have left after many years duration. Some patients describe the sensation they feel while holding the radium in contact with the diseased part as burning and drawing; others, apparently, are unaware of any sensation whatever. Radium will cause necrosis of the skin, and one has to use judgment as to whether the case has had sufficient treatment. One must be careful that the radium is not itself producing destruction of the tissue, as I have seen the disease soon heal up when the applications of the radium were discontinued. The rays from the specimen of radium marked No. 1 can be detected in a dark room penetrating ten pennies placed one upon another. The aluminium covering is likely to be eaten away by the free bromide, and radium contained in these aluminium cases should be carefully watched. Rays from the radium can be detected in the dark with the eyes shut by placing it upon the eyelids; and this fact is made use of in cases of cataract, and as regards the question of the advisability of operation.

X-rays.—Although the Röntgen rays were discovered some ten years ago, and were later on used in the treatment of lupus, rodent ulcer, hypertrychosis, it was not until the last two years that the marvellous effect of radio-therapy upon that large group of skin diseases which might be brought under the heading of seborrhœa, eczema, psoriasis, was recognised. Norman Walker, a student of dermatology of marked ability, who, having had the opportunity of seeing the treatment of skin diseases as carried out by the authorities of the world, used the following words in expressing his opinion at that time upon the treatment of skin affections by radio-therapy, viz.—"The most potent of the recent additions to the dermatologists' armamentarium are those mysterious electrical vibrations known as the Röntgen or X-rays.": and that opinion was formed before the actual wonders of radio-therapy in the treatment of skin affections was realised. In the treatment of intractable cases of acne vulgaris I consider radio-therapy plays a most important part. In tinea tonsurans, resisting all other treatment, radio-therapy can be relied on to cure the disease; but of course care is necessary here in the degree of application, as well as in a great many other cases. Sycosis, whether it is a case of tinea barbæ or so-called non-parasitic variety, you can almost name the number of applications for successful treatment.

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The previous duration of disease, even many years, does not alter the prognosis. Alopecia areata frequently improves rapidly under this treatment. I have had many cases of eczema, psoriasis, and seborrhœa apparently cured by radio-therapy. And if radio-therapy could be relied upon to successfully treat all the skin troubles which may be brought under the headings of eczema, psoriasis, and seborrhœa, the question would arise, what are the skin troubles which radio-therapy could not cure? One must, of course, at once recognise the fact that there are many skin affections which do not require treatment by radio-therapy—for instance, impetigo contagiosa is generally so readily cured by suitably applied parasiticide applications, dermatitis traumatica, drug erruptions, feigned eruptions, eruptions due to plants, and other recognisable external influences; all such diseases, of course, require such treatment as is obviously suggested in each particular case.

The question of dosage, or efficient application of the rays, must necessarily depend upon the nature of the case, that is to say, one is compelled to use extra care in treating acne of the face; but if the case be one of rodent ulcer or lupus, a stronger exposure—producing reaction—may be necessary. As a rule I seldom find a reaction necessary, and believe that the pathological alterations in the skin—such as atrophy of sebaceous and sweat glands—can be produced without ever having applied the rays to the extent of producing any apparent reaction in the tissues.

Dermametropathism in the Light of Radio-therapy.—At the Congress held at Hobart three years ago I read a paper called "Skin Markings," in which I contended that "the irritability of tissues in general may possibly, in some diseases, be calculated by the irritability of the tissues of the skin; at any rate, that certain markings prognosed the chronicity of certain diseases, and advise, in some cases, the near onset of a relapse of the disease, and that certain markings explain the exaggeration of the symptoms in some individuals, and that probably suitable treatment may be worked out by observing the effect of treatment upon the markings of the skin."

Twelve months afterwards I read a paper called "Dermametropathism," which was a further contribution upon the subject of skin markings. In this paper I contended that, by the markings, one could calculate or gauge the intensity or chronicity of the disease, and that the markings could be made use of for prognosticating the value of treatment. Although I used the commoner skin troubles—as acne vulgaris—for demonstration purposes, I laid special attention upon the marking of Urticaria pigmentosum, which marking was so unique and so plainly evidenced upon the application of pressure; but as the disease was practically incurable by treatment, it was impossible to demonstrate that the marking would gradually become normal, and its tendency to become normal would precede the cure of the disease. However, under the influence of radio-therapy we are now able to cure the disease in a reasonable time, and the return of the marking to normal has preceded the disappearance of the disease. Likewise with many other conditions, where there is now a comparatively quick change from the diseased state to normal, under treatment by radio-therapy the marking has similarly changed quickly to normal.

The Effect of X-rays upon Micro-organisms.—W. Allan, of New York, quotes twelve authorities who positively state that the X-rays have no harmful effects upon micro-organisms! One authority having exposed certain micro-organisms to the X-rays for twenty-four hours without doing them any apparent harm! On the other hand, he also quotes twelve authorities who positively state that the X-rays have the power of destroying micro-organisms! I now exhibit a few growths of micro-organisms, which have taken place in spite of some fairly severe exposures to the X-rays. In fact, in the case of the mould which grows so readily upon strawberries, the X-ray exposures were apparently followed by a decidedly exaggerated growth of the mycelium as compared with

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a control growth. However, whether the X-rays destroy micro-organisms directly or not, they must certainly have the power of rendering the soil unsuitable for the continued growth of the micro-organism, as the micro-organisms disappear with the cure of the disease. In fact, radio-therapy opens up the question as to the actual attention the micro-organisms should receive as regards treatment of some diseases. Take, for instance, acne vulgaris; the primary etiological factor is credited by some to the bacillus acnes, and I believe that Koch's four postulates have been bacteriologically obtained in connection with this disease. Even so, I would not be prepared to look upon the bacillus acnes as the primary etiological factor. We must remember that, practically, acne vulgaris is a disease of age—roughly from 13 years to 20 years; it is likewise a disease of region—face and shoulders. The bacillus acnes is evidently only able to invade the skin at a certain age, and in certain regions. I am inclined to consider that the success of radio-therapy points to the primary etiological factor being, as I pointed out in my paper, "Skin Markings," viz., that influence which causes instability of the vaso-motor system, as evidenced by skin marking, and which is constantly present in the acne regions during the acne age; and that the success of radio-therapy in this disease is due to its power of controlling or remedying the altered conditions of the skin following upon an increased irritability of the vaso-motor supply of these regions. These altered conditions are hypertrophy of the sebaceous and sudoriporous glands, thickening and stiffening of the corneal layer, retention of sebaceous matter, greasiness of the skin. This condition gives rise to retention of sebaceous matter, formation of papules, exudation of serum, and then the staphylococci; and the bacillus acnes finds the soil suitable for its growth. Radio-therapy seems to me to be the last step before we discover the ideal treatment, namely, the prevention of the instability of the vaso-motor system which makes it possible for the disease we term acne vulgaris to occur.

The X-ray Bath.—What I have termed an X-ray bath is a method of applying the X-rays so that the whole skin of the patient may be exposed to the rays at one time. The necessity for such a procedure may be gathered from the following:—Pusey, of Chicago, describes a case of mycosis fungoides which was improving under the X-rays treatment, but, owing to the tediousness of the exposures and the generalisation of the tumors, the disease, so to speak, kept ahead of the treatment. So that with a case of granuloma fungoides, if the disease is well advanced, as pictured in the photograph I now exhibit of a case which occurred in my own practice some years ago, then the prognosis is—excluding X-rays treatment—practically hopeless, the patient probably dying within a few weeks. Applying the X-rays from one tube at a distance suitable to be effective would mean a great number of applications to get over the whole body, and one can hardly devote the whole of one day to the X-rays treatment of one patient; in fact, the patients themselves often tire of this method of treatment. With six tubes working at once, however, you can see that the disease—though universal—can be treated in a reasonable time, daily exposures being given if considered necessary. I generally move the patient around so that his skin is exposed to each set of tubes for about three minutes, that is, twelve minutes in all. I have also found this method of great service in treating general eczema, psoriasis, and also cases of urticaria. It could be tried in a case of generalised sarcoma. In children one need not use more than three or four tubes at a time, the surface of the body being so much less than in an adult. In cases of pruritus, where there has been no clinical evidence of skin disease, I have found this general exposure to X-rays followed by good results. I have used general exposures in localised troubles —as cancer of the breast. I believe this general exposure has an increased

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action upon a localised disease. as compared with the application from a single tube to the part. This photograph is taken of the X-ray bath with the six tubes at work; three coils, you will notice, are worked with mercury motor breaks, whilst the others are worked with electrolitic breaks. The current supply to coils and breaks is described in accompanying chart. In the photograph I am working two tubes from one coil, as one of my coils for this work was out of order at that time. I have not found this method of working two tubes from one coil satisfactory, as it is impossible to keep the tubes at the same vacuum. Of course there is plenty of room for danger with this so-called X-ray bath, and one can easily understand that a generalised reaction from over-exposure could be followed by a fatal result. The same remark would hold good as regards the application of chloroform; and I advise anyone trying this method of treatment to use the greatest care in its application. In front of each tube will be noticed little radiometer discs for gauging the effects of the X-rays from each tube. The patient should stand in a cage made of cane, and the head protected with a lead foil mask. A trained nurse should be in attendance to guide the movements of the patient. A linen cover-all should be worn.

Treatment of Hypertrichons by Electrolysis and Radio-therapy.—If necessary, I anæsthetise the part by cataphoresis, using solution of adrenalin and cocaine.

Three needles are connected with fine wire, same as used in winding secondary of X-ray coil; these are connected with negative pole of suitable number of dry cells.

After removing the hair by electrolysis, I apply the X-rays, and have found that the X-ray exposures seem to considerably lessen, or even prevent, the electrolytic action of the needles being followed by any noticeable reaction in the skin.

This action of the X-rays seems to suggest that their efficient application in the form of an X-ray bath might lessen, if not prevent, the pitting of smallpox.

EPILEPSY: IS IT INCURABLE?

By George E. Rennie, M.D., M.R.C.P., Lond., Sydney.

Although epilepsy has had a place in medical nosology from the earliest times, and much speculation has been indulged in as to its true nature and pathology, we must confess that even at the present day the extent of our exact knowledge of its pathology and etiology is limited. There is much diversity of opinion as to what should be included under the term "epilepsy," and several definitions have been given which are considered by one and another to cover the whole ground of the symptomatology and pathology of the disease.

Now, before we can discuss the question of the curability of this disease, we must have some clear idea as to what we mean by epilepsy and what we mean by "cure." When we attempt to give a clear and comprehensive definition of this disease we find at once that we are called to a most difficult problem. Our difficulty arises from the fact that the chief symptom of the disease we commonly call epilepsy—namely, the convulsive attack, with more or less loss of consciousness, and the sequence of clonic and tonic spasms—is also a symptom which may occur under a variety of conditions, but in all of which we find, first of all, a state of irritability of the brain (to use a

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rather coarse term), and some reflex exciting cause, either peripheral or central. For example, we know, as a clinical fact, that the brain of the infant or growing child is irritable, or reflexly excitable (whatever interpretation we may be able to put on this somewhat crude term), and that convulsive attacks, which are manifestations of uncontrolled cerebral motor discharges, are easily induced in a child. The convulsive attacks which we meet with in children who are teething, and in which the reflex exciting cause is considered to be the condition of inflammatory irritation at the terminations of the sensory branches of the fifth nerve, or who are the subjects of some gastro-intestinal irritation, owing to the invasion of some specific disease or to the presence of some undigested food or irritant, are manifestations of this uncontrolled excitability of the cerebral cortex. [But we know, also, that in some children, when the peripheral exciting causes are removed, no further convulsions may occur, and the child grow up perfectly strong and healthy, both in body and mind; but we also know that in some children, in whom convulsive attacks have been excited in the first instance—apparently as the result of some simple reflex irritation—after the removal of the specific irritation, the convulsive attacks recur, and the child presents the recurrence of these attacks without any apparent cause, and we then pronounce the case one of "epilepsy."] We know, also, that convulsive attacks accompanied by an aura and loss of consciousness, with clonic and tonic spasms, occur at intervals in patients who are the subjects of gross organic cerebral disease—such as cerebral tumor—or who are the subjects of some congenital brain defect, in whom there are not only defective cerebral development (a negative condition, which, according to Hughlings Jackson, cannot produce positive symptoms), but also some irritation—a positive condition—which causes the positive symptom of convulsions. These cases are sometimes spoken of as symptomatic or Jacksonian epilepsy, if the convulsive attacks are confined to one side of the body or part of one side of the body, and not necessarily accompanied by loss of consciousness. It is clear that here we have a definite organic irritation of the cerebral cortex, and cases of this nature are supposed to be distinct from those of so-called "idiopathic" epilepsy, in which frequently no gross macroscopic lesion of the brain can be detected.

One of the chief difficulties which have existed, and will continue to exist, in the way of elucidation of the true pathology of the idiopathic epileptic convulsive attack is the fact that there is no tendency in the disease to lead to a fatal termination—at any rate in the early stages—that if a fatal termination does take place owing to the disease itself, it is generally after a series of attacks, or in a condition of status epilepticus, which itself gives rise to a state of congestion of the cerebral veins, accompanied by minute hæmorrhages, pathological conditions which tend to obscure rather than to help towards a true understanding of the pathology of the disease. Moreover, if the patient survive for some years and present recurring attacks of convulsions, it is not always easy to definitely state how much of the pathological conditions which may be found in the brain is due to the recurrent convulsions, with their accompanying cerebral congestion, and how much to the original—and what we might call the fundamental—cause of the disease. And here I would remark that it appears to me that a great deal of the experimental work which has been done towards solving the problem of the epileptic convulsion is beside the mark, and only tends to show—what we already know from our clinical and *post-mortem* experience—that uncontrolled motor cerebral discharges can be excited by disturbances of the cerebral circulation, or as the result of some irritant poison acting apparently upon the cerebral cortical cells themselves. We know, for example, that in uræmia, the condition in which the kidneys are failing in their functions of excretion, we get convulsive

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attacks which are indistinguishable from epileptic convulsions; but this does not help us much towards solving the question of the pathology of idiopathic epilepsy, except in so far as we may reason from analogy that in idiopathic epilepsy we may have a similar kind of irritant, or toxin, or poison of some kind which irritates the brain and causes the discharge of uncontrolled motor activity. Clinical pathological investigations of cases of idiopathic epilepsy have shown that some change in the metabolism of the body takes place immediately antecedent to the occurrence of the fit, manifested by an increased toxicity of the blood and the excretions; and we are justified in concluding that there is probably a development of one or more complex chemical bodies which act as irritants on the cerebral cortical cells, and are the immediate cause of the convulsive attacks: in other words, the irritant is the result of auto-intoxication; and the balance of all modern investigations goes to support this view. But this alone is not sufficient to determine all the clinical phenomena of the disease. The microscopic examination of the cerebral cortex tends to show that, at any rate, in all long-standing cases of the disease, there is a wasting of the sensory cells of the second cortical layer with neuroglia hyperplasia. We have thus evidence that in true idiopathic epilepsy there is organic disease of the cerebral cortex, plus irritation from auto-intoxication.

Now, if we take a survey of the various morbid conditions which are commonly associated with the occurrence of convulsive attacks, we cannot shut our eyes to the fact that for the production of the typical convulsions with loss of consciousness and spasms, such as are met with in idiopathic epilepsy, some special condition of irritability of the cerebral cortical cells must be present. Why do we sometimes get typical convulsions of this nature for years before the symptoms specially characteristic of cerebral tumor manifest themselves, and in other cases of cerebral tumor no convulsions occur? The situation of the tumor in the brain alone will not explain it. Or, again, why do we get some cases of meningitis in which there is generally some encephalitis as well, with no convulsions, or at any rate only in the early period of the disease—a condition in which we have, *par excellence*, one would say, an irritation of the cerebral cortical cells by inflammatory products? Or, again, in some cases of uræmia we get various nervous symptoms, such as mania, or some paralytic conditions, and yet no convulsions? Surely these facts point to the conclusion that, for the development of a typical convulsion, there must be some special condition of the cerebral cortical cell, or else some special kind of irritant, whether that be toxic or vascular. We must, of course, recognise that for the production of the convulsive attack —whether this be what we may call a simple convulsion or a convulsion which is repeated at intervals in a patient who is pronounced to be an epileptic—we must always have an irritant, whether we can recognise its exact nature or not, that is, an irritant which is conveyed by a peripheral sensory nerve, or the nerves of the special senses, or a chemical irritant conveyed by the circulation to the brain; and we must have the cerebral motor cells in a condition capable of being reflexly stimulated to discharge; and there must also be an intact motor path. These conditions are necessary for the production of a typical convulsive attack; and just as under these conditions a convulsion may be excited by some peripheral sensory stimulus, so we also know that the motor discharge may be inhibited by a powerful peripheral stimulus.

We have, however, further to note that in some patients we do not get the fully developed convulsive attack, but only some slight loss of consciousness, with or without some very slight motor manifestation, either of the nature of a spasm or a relaxation of muscles. These so called attacks of petit mal may occur alone as the only symptom of "epilepsy," or they may occur associated with the fully developed convulsions.

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Another fact which we must bear in mind is the frequent mental impairment which is associated with the recurrence of the convulsive attacks. This is commonly stated to be due to the recurrence of the fits; but this appears to me to be an erroneous view, for, if it were correct, we ought to find every patient who is the subject of recurrent convulsions to be the subject of some degree of mental impairment. This is, however, hardly in accordance with facts.

I think you will see the bearing of these remarks on the question raised in the title of this paper. We must, I think, regard convulsive attacks as of two kinds: first, those which are manifestations of an undue excitability of the cerebral cortex, which is aroused as a result of some peripheral sensory irritation, the main factor being the reflex irritation; and second, those in which there is a definite morbid condition of the cerebral cortex, that is, an organic structural change, a degeneration of the cells in the sensori-motor cortex. The former condition may possibly pass into the latter, but not necessarily so, and, under appropriate treatment, by the removal of the source or sources of peripheral irritation, the undue excitability of the cerebral cortex may be subdued, and the patient be cured of his convulsive attacks. To this class belong, I think, those cases of recurrent convulsions which are not accompanied by serious deterioration of mental or physical health. To the latter class belong the large proportion of cases of recurrent convulsions which are accompanied by signs of mental enfeeblement, and by various abnormal psychical manifestations—such as epileptic amnesia, the unconscious committing of crimes, &c.

We are thus driven back to review our position in regard to the definition of what we mean by "epilepsy." Are we to include all these different groups of cases under the one name "epilepsy," modified possibly by some adjective—such as "major," "minor," "psychical," &c.? I should consider that a patient who is suffering from recurrent convulsive attacks, which cease as a result of the removal of some peripheral irritation, *e.g.*, the removal of worms from the intestine, the correction of some refractive error, or the abstention from some chemical irritant—such as alcohol or absinthe—is not suffering from true epilepsy, but is the subject of reflex convulsions. But if, after the removal of all possible sources of peripheral irritation, the attacks occur, and specially if they recur in spite of an improvement in hygienic surroundings, and in spite of the depressant effects of the bromide or other drugs on the excitability of the cerebral cortex, then that patient is suffering from true idiopathic epilepsy; and such patients, I believe, invariably present some mental deterioration, whether this be a mere loss of control of temper, or increased irritability, or the more definite signs of dementia.

In my opinion the definitions usually given fail, in that they do not differentiate between these two types of convulsive attacks; namely, those depending on some simple reflex irritation, which can be recognised on careful investigation, and recurrent attacks for which no obvious cause can be found.

Within recent years several groups of statistics have been compiled, by different physicians, bearing on the question of the curability of epilepsy, and the conclusions arrived at have been that in from 5 to 15 per cent. of cases a cure is effected. But statistics are notoriously unreliable; and yet they are of some service, in enabling us to take a broad view of the general progress of patients suffering from recurrent convulsive attacks under different methods of treatment.

Now, before we can arrive at an answer to the question of the curability of a disease we must clearly understand what we mean by "cure." And here we are at once brought face to face with a difficulty. Some years ago the

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American physicians discussed the question of what should be regarded as a cure of epilepsy. Some were disposed to consider a case as cured in which there was no recurrence of the convulsions for ten years, this being considered a sufficiently long period for the brain to have recovered a normal degree of stability. We know that it does take a very long time for the brain to lose its condition of reflex hyper-excitability, for patients who may have had no fits for perhaps two or three years while taking bromides will experience a return of the symptoms almost immediately on leaving off the medicine, the fits having been merely kept in abeyance, the disease not having been cured.

If we consider for a moment other kinds of disease, and ask ourselves are patients cured of them, we shall be obliged to answer that in many cases the disease is not really cured; the patient is relieved, from time to time, of the symptoms, and may be free for years, and yet the tendency is towards a recurrence of the symptoms. Take, for example, the common disease rheumatism. A patient may have an attack of rheumatism. After some time the symptoms subside, and he returns to a normal condition of health, and he is considered to be cured. But we know that he is liable to a recurrence of the disease from time to time, although he may never have a recurrence of the symptoms for the rest of his life. Or take bronchitis: we know that a patient may be cured of an attack of bronchitis; we also know that the bronchial mucous membrane has been damaged by the attack in such a way that the patient is more susceptible to a recurrence of the disease, and as he grows older the attacks may become more and more frequent, until ultimately be becomes a chronic bronchitic, and is incurable. Although he may have been relieved of the symptoms of the previous attacks, he cannot be considered to have been cured of his disease.

Now, I think a similar condition of things holds with regard to epilepsy. A child who inherits no tendency to epilepsy may have convulsions during teething, that is, convulsions due to some reflex peripheral irritation, and after the subsidence of the irritation no further convulsions may occur. But is it not true that the occurrence of these convulsions predisposes to the recurrence of convulsive attacks later in life? The rapid development of the brain and nervous system coincident with puberty may favor the recurrence of the attacks, which, however, may subside under treatment. The patient may thenceforward have no further attacks. Or, on the other hand, later in life the convulsive attacks may recur, and the patient present the clinical phenomena of a case of so-called idiopathic epilepsy, and he may become a confirmed epileptic. One cannot predict the exact course which any individual case will pursue; one can only say that the patient has been cured of his epilepsy when he remains free from the recurrence of the attacks up till the time of his death. The mere fact of a patient remaining free from the convulsive attacks for many years does not, in my opinion, mean that he has been cured of his disease; and when statistics are adduced to show that epilepsy is cured in a certain proportion of cases under one or another line of treatment, I maintain that such are fallacious. All that they show is that, in a certain percentage of cases, as a result of treatment, the conditions favorable for the development of the convulsions do not recur; but the potential state still remains. I would not be taken to mean that I do not believe in any method of treatment as being efficacious. On the contrary, I believe that, by a judicious use of drugs or other method of treatment, we may be able to prevent a recurrence of the fits to a greater or less degree. But drugs cannot cure a condition of organic degeneration of the cerebral cortex, such as underlies all cases of true organic idiopathic epilepsy; all that we do is to lessen the reflex excitability or remove causes of peripheral irritation, but we do not cure epilepsy.

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To recapitulate, then, in conclusion I would say that the answer to the question I have raised in this paper depends upon the view we take as to what we include under the term "epilepsy." If under this term we include all cases of recurrent convulsions, whether these be due to some recognisable reflex irritation or not, then we may agree with those physicians who maintain that in a certain proportion of cases a cure of the disease can be effected. If, however, we restrict the term in the way I have indicated, we must regard it as an organic disease of the cerebral cortex and, as such, as incurable.

DISCUSSION.

Dr. JAMIESON said that before attempting to answer the question it was necessary to ask and give some reply to another: "What is epilepsy?" There must always be a distinction made, if possible, between true epilepsy—called idiopathic—and all kinds of eclamptic or epileptiform attacks. This, of course, might be difficult enough, since eclampsia had so many causes; many of them very obscure. They might be poisons, as in chronic renal disease, or irritations—peripheral or central. And it had to be remembered that the proneness to the occurrence of convulsions differed greatly in different persons, and at different ages. All young children had a considerable, though unequal, liability—what he had been in the habit of describing as *convulsibility*. This tended to pass off with advancing age, and the fact of an infant having had one or more attacks of general convulsions did not practically show that there was risk of the later development of true epilepsy. But the "convulsibility" might be so marked and persistent that the fear of such development was naturally and properly excited. One of the commonest causes of infantile convulsions, in his opinion, was acute indigestion; pieces of meat, cheese, raisins, &c., might readily produce them in an infant, but would not do so in an ordinary healthy schoolboy. He once knew a case where such a cause as eating green fruit brought on convulsions in a boy of about 13, ordinarily in good health. In such a case it was natural to look forward with considerable misgivings to the boy's future as a probable epileptic. Where attacks began without obvious cause, recurred at intervals without any obvious excitant, and were not associated with any progressive signs of disease, either of the brain or other organ, the condition was probably epilepsy; and, though the attacks might be suppressed for a time, cure was, to say the least, extremely unlikely. In his opinion true epilepsy, when once established, was practically an incurable condition. And perhaps the hopelessness was best seen where the petit mal attacks were a prominent feature. The bromides would often suppress the fully-developed convulsive attacks, but seemed almost always to be without effect against these less violent but really more maleficent manifestations. His own opinion, therefore, was that Dr. Rennie's question must be answered in the negative, in the present state of our knowledge.

DIETING IN RENAL DISEASE.

By GEORGE E. RENNIE, M.D., M.R.C.P., LOND., Sydney.

In introducing this subject I wish, at the outset, to state that I intend to confine my remarks to the subject of dieting in the various forms of acute and chronic nephritis.

Some preliminary considerations are essential. In the structure of the kidney we have two important elements—(1) The epithelial lining of the

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glomeruli and of the tubules, and (2) the blood vessels, both of which take an important share in the physiological function of excretion, and both of which may be involved to a greater or less extent in the morbid conditions of the kidney which determine the clinical manifestations of acute and chronic nephritis.

It is pretty certain that the water of the urine is derived from the blood, partly by a process of osmosis and partly by an active secretion on the part of the glomerular epithelium; and we know that the quantity of water excreted is directly related to the amount of blood which circulates through the kidneys, and also—though to a much less extent—to the blood pressure. The mineral salts and pigments in the urine are excreted from the blood by the secretory activity of the glomerular epithelium. The organic constituents, namely, urea, uric acid, and the extractives, are excreted by the tubular epithelium. In the case of one substance, hippuric acid, there is good reason to believe that it is formed in the kidney from benzoic acid. This shows that the kidney is capable of synthesising complex organic substances, and this function of the kidney will consequently suffer as well as the others.

In the case of acute parenchymatous nephritis we must remember that the kidneys are in a condition of acute inflammation, that is, they are responding to the irritation excited by the presence in the blood of a toxic substance or substances which are being excreted, and, in the process of excretion, are irritating the renal structures: the blood vessels are dilated, the circulation is slowed, there is exudation of serum into the surrounding tissues, and the tubular and glomerular epithelium is impaired in its nutrition, and consequently also in its selective excretory functions. Further, where there is general renal dropsy we know that the nutrition of the capillaries is impaired, so that exudation of serum into the connective tissue spaces is taking place. When such a condition passes into chronic parenchymatous nephritis we have a failure of the kidney to excrete the normal waste products of the body—the result of the ordinary metabolism, plus the waste products resulting from the food which has been ingested—and these excretory products in their turn irritate the kidney and also react upon the whole system, producing a condition of chronic toxæmia, or uræmia, as it is called. In this condition the stress of the disease falls upon the epithelial structures, which are impaired in their nutrition and manifest various degrees of degeneration; consequently they are unable either to synthesise or excrete as they should.

In chronic interstitial nephritis, or granular kidney, the primary lesion is probably in the blood vessels, this leading to overgrowth of the connective tissue and subsequent impairment of function and ultimate destruction of the epithelium of the glomeruli and of the tubules. This process is much more gradual in its development, but ultimately leads to a considerable reduction in the actual amount of actively functionating kidney substance. When we get an attack of acute inflammation superadded, or when the heart begins to fail and a condition of passive congestion in the kidney is set up, the excretory powers of the epithelium are almost in abeyance, and we get evidence of uræmia.

In considering the diet which is most suitable to be adopted in the treatment of these various conditions, it is necessary to bear in mind that the digestive powers of the stomach are impaired, the power of retaining and digesting different articles of diet varies, and this, to a certain extent, independent of the effect on the kidney of the metabolic products of the particular articles of food. We must remember that the nutrition of the body must be maintained somehow or other, and that, to effect this, a certain amount of the staple food constituents—namely, proteids, carbohydrates, and fat—

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must be administered. We have further to bear in mind the end products which result from the digestion of the different articles of diet, and to consider how these end products are excreted. Hence we have the problem before us—how are we to so adjust our diet scale for patients suffering from acute or chronic renal disease so that a sufficient number of calories will be produced to maintain the body nutrition and, at the same time, while giving the kidney as little work to do as possible, the waste end products of the food shall be excreted as completely as possible? The ordinary constituents of the urine, as you know, are water, mineral salts, nitrogenous matters, and extractives; and these various constituents are normally not excreted with the same degree of facility. Von Noorden gives the following list of the relative facility of excretion of these substances:—Those excreted with difficulty: urea, creatinin pigments, hippuric acid, phosphates, inorganic sulphates, potassium salt (?), water; those well excreted: uric acid, xanthin bases, aromatic substances, ammonia, amido acids, chlorides, carbonates, water. This of course applies to kidneys in normal conditions; and the difficulties will be still greater if the kidneys are in a state of disease. We must, therefore, in selecting our diet list, avoid, as far as possible, using those articles of food which lead to the formation of such end products as are difficult of excretion.

We shall consider first acute parenchymatous nephritis. The pathological condition of the kidney varies somewhat in different forms of this disease; for example, in the nephritis which follows scarlet fever the stress of the disease falls specially upon the glomeruli, while, in other forms of the disease, the tubular epithelium is the seat of an irritant proliferation with congestion of the blood vessels. Hence, in scarlatinal glomerular nephritis we must endeavor so to adjust the diet that the glomeruli are relieved as far as possible from the function of excretion. Remember, therefore, that we must specially limit the amount of water and salts. In other forms of acute nephritis, where there is a diminished excretion of urine, partly, perhaps, owing to mechanical blocking of the tubules with epithelial debris and blood exudates, in which the glomerular epithelium is not specially involved, a more liberal supply of water might be indicated with a view of flushing the kidney, provided we can be sure that the kidneys are able to deal effectively with the hydræmic blood. It is this very point which has led to some diversity of opinion as to whether it is advisable or not to give large quantities of water to a patient suffering from acute nephritis. It is generally taught and believed that in acute nephritis a diet restricted to milk only is the best. Milk, of course, contains a large percentage of water, and in restricting the diet to milk only we are giving a large amount of water along with a relatively large amount of phosphoric acid, fat, and a smaller amount of proteid carbohydrate. Milk contains so much phosphoric acid that adults who are living on a pure milk diet show a daily increase in the urinary phosphorus excretion amounting to three or four grammes. We have seen above that phosphates are excreted with difficulty; consequently, when giving a pure milk diet, we are giving a fairly large amount of a substance which is excreted with difficulty by the kidney. To counteract this ill effect, von Noorden, some years ago, advised that patients suffering from acute nephritis should take a small quantity of carbonate of calcium several times a day along with their milk. By this means a large amount of the phosphoric acid is combined with the calcium, and is excreted by the bowel. Milk also contains a relatively large proportion of proteid, and a patient who is taking four or five pints of milk a day will receive from 110 to 120 grammes of proteid; thirty to thirty-five grammes of this will be converted into urea and require excretion by the kidneys, an amount which the kidney in a condition of acute inflammation

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will be unable to deal with satisfactorily. Moreover, if the patient is able to take three or four pints of milk a day without vomiting, a considerable amount of water must be absorbed and the blood rendered hydræmic; when we keep the skin and bowels active a certain amount of this superfluous water is removed, and a comparatively speaking small amount of water remains to be excreted by the kidneys, and consequently very little flushing effect will be produced. If, in addition to the milk, we give our patients several tumblerfuls of distilled water per diem, we still further increase the hydræmic condition of the blood, we increase the amount of blood which is conveyed to the kidney, but unless the glomerular capillaries and the glomerular epithelium are in a fairly good condition so far as their excretory power is concerned, we do not get a flushing effect, and the hydræmic condition of the blood tends rather to increase the amount of dropsy, even if it have no other ill effect. If, however, the stress of the disease has fallen upon the tubular epithelium, then a flushing of the tubules may be effected; and this method of treatment, and the removal of the epithelial debris and blood exudate will tend to relieve the congestion of the intertubular blood vessels and favor the return of the normal excretory power of the tubular epithelium. Hence, in my opinion, if we know the case to be one of scarlatinal nephritis, we should limit the amount of water and only give a comparatively small quantity of milk, with the addition of some carbonate of lime. If, on the other hand, we know the nephritis to be due to exposure to cold or some toxic influence other than scarlet fever, then the administration of a larger amount of prepared milk, together with large draughts of distilled water, may prove of considerable benefit; but should only be persevered in if there be a definite increase in the amount of urinary water, thus proving that the kidney is in a condition to deal with a large amount of hydræmic blood. If, on the other hand, there be no increase in the quantity of urine and the œdema increases rather than diminishes, then there should be a limitation in the amount of fluid ingested. We have seen that milk is not, in every case, at any rate, so perfectly harmless and beneficial as an article of diet in acute nephritis, and we have to consider what other articles of diet may be given either in addition to or in substitution for milk, since we have further to bear in mind that we are occasionally brought face to face with a patient who is unable to retain or digest the necessary amount of milk. It is well to remember that it has been ascertained that it is possible to administer very small quantities of proteid for a short time without impairing the health of the individual, provided a corresponding amount of carbohydrates is administered at the same time; consequently in the early stages of acute nephritis we can, if necessary, be content with administering only a small quantity of milk and increase the amount of fats by adding cream, and give carbohydrates in the form of oatmeal, groats, or maizena, with the addition of butter, sugar, honey, or golden syrup. By this varied diet we can give the desired quantity of calories with only a small percentage of proteid. With the increase in the urinary water, and the diminution in the amount of œdema, and the subsidence of other symptoms, we may gradually add other articles of food—such as bread, vegetable soups, and later on, fish, chicken, or meat.

In chronic parenchymatous nephritis the pathological conditions are different from those in acute nephritis. We have not the condition of acute congestion of the blood vessels with sluggishness of the circulation, but a condition, as I have already said, of defective excretory powers in the renal epithelium. Hence we should avoid giving those articles of food whose end products are only excreted with some difficulty. This would necessarily lead us to restrict the amount of animal proteid and to increase the amounts of carbohydrates and fats. On these indications we should withhold meat

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and meat extracts, both red and white, and supply the necessary amount of proteid in the form of milk albumen, eggs, or vegetable proteid. With regard to the administration of water, we must be guided by the same indications as in acute nephritis.

With regard to the dieting in chronic interstitial nephritis, or granular kidney, we are brought face to face with more difficult problems. This is essentially a chronic disease, and patients suffering from it may live for many years; it is, consequently, difficult to lay down a diet which will be suitable at all periods of the disease, and which will be sufficiently varied to prevent patients from becoming nauseated and disgusted. We have, moreover, in this condition to pay particular attention not only to the kidney but also to the associated cardio-vascular disease. In consequence of the general high blood pressure, and the large amount of blood circulating through the kidney, there is always a large excretion of water; the amount of water tending to increase, as Bradford has shown, as the amount of functional kidney substance tends to diminish. The only exception to this being during periods of intercurrent acute nephritis, or when the heart begins to fail and we get a condition of passive congestion of the kidney. As a general rule, too, at any rate in the early stages of the disease, the kidneys appear capable of excreting an almost normal amount of urinary solids. Bearing in mind these facts, there can surely be, not only no special advantage, but, on the contrary, positive harm in binding down patients suffering from this disease to a strict milk diet.

In view of the fact that milk contains a large amount of water, we should rather restrict the amount of milk which is drunk, and also restrict the amount of fluid of any kind. The digestive powers of the stomach are, to some extent, impaired, and we must give food in such a state and of such a nature as can be easily digested and assimilated. The administration of much fluid of any kind, whether it be plain water, or broths or soups, should be avoided, and the meals should be as dry as possible, and ample time allowed between each meal for its full digestion.

Now let us consider the special articles of diet as regards their suitability in this disease. First, as regards proteids, in what form should they be given? It has been commonly taught that all red meats are injurious, and that so-called white meats alone should be allowed. This opinion is based upon the supposition that red meats contain considerably more of the nitrogenous extractives that are injurious to the kidneys than white meats. Some investigations on this point were undertaken by Offer and Rosenquist; and their experiments tended to show that the nitrogenous extractives were found to be sometimes more abundant in red meats and sometimes in white meats; but greater differences were found in the same class of animals, and between several samples from the group of white meats on the one hand and of red meats on the other, than between the average amount contained in white and red meats. Certain kinds of white meat—such as poultry and hare—were even found to be richer in extractives, on an average, than beef. Two of von Noorden's assistants (Kaufmann and Mohr) studied the elimination of urinary excretion after the ingestion of equal amounts of white and red meats. Their results were ambiguous, but certainly tended to show that the amount of albumen in the urine was not influenced by the form in which proteid is taken. After all, clinical experience furnishes the best answer to this question. My own experience, so far as it goes, agrees with the statement made many years ago by Goodhart, that an occasional administration of red meat is not only harmless but is actually beneficial, in enabling us to give a more varied diet and relieving the monotony of a diet of fish and chicken. Von Noorden has arrived at the same opinion. We may say that red meat, in a form easily

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digestible, may be allowed daily, provided no complication is present to contra-indicate it. There can be no objection either to the administration of albumen, in the form of eggs, either boiled or poached, or in the form of custard, in moderation. But the total amount of animal proteid, whether it be in the form of fish, fowl, meat, or eggs, must be restricted—but not to such an extent as to impair nutrition or physical strength; and these must be presented in such a form as to be easily digested. It is important to remember that the functional activity of the kidneys depends upon the maintenance of a free flow of blood through them, and the cardiac hypertrophy which we find in cases of granular kidney secures this result. But the hypertrophied heart muscle demands an extra supply of nourishment in the shape of proteids and fats, and we must see that our dietary scale includes these in fair amount.

As regards vegetables, while, in general terms, one may say that these are harmless, we must remember that some—such as green peas, cabbage, &c.—are difficult of digestion, and tend to cause flatulence and thus to embarrass the heart. Some, on the other hand—such as radishes, celery, and asparagus, &c.—tend to directly irritate the kidney, and may lead to the occurrence of hæmaturia, or at any rate increase the amount of albumen. For the same reason, spices and condiments should be avoided by the subjects of granular kidney.

As regards the use of the ordinary beverages—such as tea, coffee, and cocoa—we have to consider their action from different points of view. As I have already remarked, the digestive processes in these patients are somewhat impaired, and anything that will tend to still further delay digestion and cause flatulence is to be avoided. The infusions of tea and coffee appear somewhat to delay digestion; and, by reason of the theine and caffeine which they contain, they act as stimulants to the heart, possibly by direct action or through the cardiac ganglia. This action is not a powerful one in healthy persons, but when we have the heart hypertrophied, and the muscular tissue perhaps imperfectly nourished, by reason of the degeneration of the coronary arteries, the effect of these infusions may be detrimental unless taken in moderation. They also tend to raise blood pressure, and so increase the predisposition in these patients to the occurrence of cerebral hæmorrhage. There is reason to believe that caffeine and, probably, theine, have also a direct stimulating effect upon the renal cells, which leads to an increase in the ordinary excretion. But such stimulation, while it may be of therapeutic value under other pathological conditions, can hardly be considered beneficial when the renal epithelium is impaired in its nutrition, or actually degenerated. On the contrary, this action must be injurious, and accordingly tea and coffee should only be used sparingly. The same objection cannot be taken to cocoa, but, by reason of the large amount of fat which it contains, it may be digested only with difficulty.

As regards other beverages, which contain more or less alcohol, from the point of view of its poisonous influence on the kidney we should advise complete abstention from their use in any form. But here again other organs have to be considered besides the kidneys, specially the heart and circulation and the stomach. The use of alcohol may be indicated when the heart is flagging, and a cardiac stimulant is needed; as, for example, when some complication—such as pneumonia or bronchitis—has developed. Then, again, the appetite may be more or less lost, and, according to some physicians, the use of a little wine or champagne with dinner may promote an appetite and aid digestion. But it is doubtful if the use of alcohol for these specific purposes can be defended, in view of its otherwise detrimental effects, especially as other drugs can be used to secure these ends; and our safest course is to interdict its use absolutely. Some of the other so-called temperance

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beverages, which contain only a minute quantity of alcohol, are injurious by reason of their other constituents; for example, ginger beer and ginger ale not infrequently contain capsicum, and this is a direct irritant to the kidney.

One further word on the use of water. Opinions differ as to the amount of water which should be allowed these patients. Where the amount of urine is largely increased, thirst may be a troublesome symptom, and a certain amount of water may be allowed to relieve it; but this is the only condition in which the administration of large quantities of water would be justifiable.

Tobacco has a more powerful action upon the heart than either tea, coffee, or alcohol, and the heart of the patient who is suffering from chronic granular kidney will not tolerate, as a general rule, a cardiac poison like tobacco. But here again circumstances and idiosyncrasies must be considered. Cigarettes must be unhesitatingly condemned; but a moderate use of the pipe or cigars—provided no cardiac irregularity, palpitation, or depression is produced—may be allowed if the patient has been an habitual smoker; for we must not attempt to deprive our nephritics of anything more than is absolutely essential. By attempting too much restriction we dishearten the patient, and, instead of enjoying a fair measure of good health for perhaps many years, he will probably become a confirmed hypochondriac.

DISCUSSION.

DR. JAMIESON said that, on such a large subject, it was difficult to make other than general statements. In acute nephritis, when the kidney had almost completely lost function, there should clearly be as little work as possible put on it. In the glomerular form there might almost be complete suppression, and it was manifestly undesirable to introduce more fluid into the blood and tissues when the outlet was practically closed. Even milk, therefore, desirable as it might be on other grounds, should be given only in small quantities; and of solid food none. On the other hand, when secretion had again begun, it might be eminently beneficial to prescribe milk and other mild fluids liberally. It was doubtful if at any stage of renal disease the stronger animal broths were even desirable. When the state of chronic disease was fairly established, he thought the most important thing to keep in mind was that the capacity of secreting the organic nitrogenous constituents of the urine was more or less, and often greatly, diminished. And it was perhaps specially important in the granular kidney. The patient may long be in comparatively vigorous health, able to enjoy, and perhaps digest, solid food. And all the while there might be a growing incapacity to throw off the waste products of metabolism, with consequent standing risk of accumulation and auto-intoxication. A liability to a uræmic attack in some form might thus arise, almost unexpectedly. As regards the prescription of a diet, under these circumstances, the important thing seemed to be to have regard to quantity quite as much as quality. Red meat might, in practice, be better than white meat, if less satisfied the appetite; and the same was true of the least stimulating articles, be it fish or eggs. When it is a question of satisfying the mere needs of the body, it had been clearly shown by the experiments of Chittenden, following up the teaching of Fletcher, that the average person could live comfortably on one-half or one-third of the quantity habitually consumed. Diet, to put it shortly, was more a question of quantity than of kind, on the supposition that there was a proportioning of its elements in respect of proteids and carbohydrates.

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TWO CASES OF LEAD-POISONING IN CHILDREN.

By J. Macdonald Gill, M.D., Lond., Sydney.

Case I.—Robert H., 5 years of age, was admitted to the Children's Hospital, Sydney, on January 26th, 1905, and discharged on March 24th.

The history was as follows:—He had always been a healthy child. The present illness had lasted seventeen days, with pain and weakness in the back and limbs; he could not bear to be touched, and lay in bed with his knees propped up. There was also a history of severe pain in the abdomen in the early part of the illness, probably colic, which had passed off. No history of lead-poisoning was obtained at first, but the mother afterwards found out how the poisoning took place. A wall at the back of their premises was being painted, when this boy was seen by the older children to suck his fingers after getting them covered with paint. The mother said it was brown, not green, paint. About four days after this took place he became ill with pains and weakness in the legs. On admission to the hospital the child looks dull and heavy, but does not cry out much unless touched. He lies in bed with his knees drawn up. The mental condition is good. He has had headaches, but none since admission.

Eyes.—The pupils are equal, and react to light and convergence. Has weakness in both external recti, but no ptosis. The optic discs are normal. Eyesight good. There is slight facial paresis on the left side, especially of the lower face muscles. The tongue is protruded in the middle line. The senses of smell, taste, and hearing are apparently normal. Speech is nasal in character, and articulation is defective, but deglutition is unimpaired; the soft palate contracts fairly well when tickled.

Upper Limb.—There is partial wrist drop and inability to extend the fingers. The reflexes cannot be obtained. There is no special tenderness on pressure on the posterior interosseous nerve.

Lower Limb.—There is almost complete loss of power in the extensor muscles of the thigh and leg, so that the child lies in bed with the knees flexed, the ankle joints extended, and the toes flexed. The muscles are wasted and flabby. There was some anæsthesia up to about the middle of the thigh. The knee jerks are both absent; the plantar reflexes are present, of the flexor type, though feeble. There is no tenderness on pressure on the muscles themselves; but intense pain is caused by pressure on the great sciatic and popliteal nerves in each leg, behind the hip and knee joints, where the nerves are not covered by muscle. Similarly, when the nerves are put on the stretch, by fully extending the knee joint, or by flexing the hip with the knee extended, the child screams with pain. Such tenderness is not observed in the arms, probably owing partly to the deep course of the posterior interosseous nerve, and partly to the mildness of the affection there.

Bladder and Rectum.—There is almost complete loss of control over the bladder; he has absolutely no control over the rectum, the sphincter ani being completely paralysed. The muscles of the back are very weak. The abdominal walls are retracted; the diaphragm contracts well. Respiration is easy. The abdominal and cremasteric reflexes are present. There is no intestinal colic, the bowels being open daily without difficulty. The urine contains a trace of albumen. There are no diphtheria bacilli in the throat. There is no blue line on the gums, and his teeth are quite clean and free from tartar. On February 2nd it is noted that he is improving slowly, but that there is complete paralysis of the left external rectus, whereas the right is normal. The bladder reflex is normal, while the anal sphincter is still completely paralysed. While in the hospital he improved rapidly and continuously. On discharge it is noted that he can walk well, and has regained

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most of the power in his arms and back. The muscles of the legs are much wasted, and the knee jerks still absent. The temperature remained normal throughout his stay in the hospital.

Case II.—Samuel Henry H., 10 years of age, was admitted to the Children's Hospital on April 3rd and discharged on April 17th, 1905.

History.—The boy was brought to Sydney for treatment from his home at Kempsey, on the Macleay River. Four years ago he had a fall on to the back of his head, which was followed by convulsions. Eight months later he had a severe febrile attack, with constipation: he then had convulsions for thirty-six hours, and recovered completely in four weeks. Two years ago he lost the use of all four limbs, and was in bed for six weeks; he gradually recovered till he could run about. Four weeks ago the weakness appeared—in the hands especially. He has been pallid for some months. Their house was painted before the attack which took place two years ago. The bowels are regular. Has no headaches.

On Admission.—The boy is small for his age, and, intellectually, is very backward; he is quite childish in his habits. The teeth are very dirty, and have a considerable deposit of tartar on them. There is a definite blue line on the gums. There is well-marked foot and wrist drop: the muscles are not completely paralysed, but are very weak. He can walk with his feet in position of talpes equinus. The knee jerks and other reflexes are present. The nervous system is otherwise normal.

The Urine.—Sp. gr. 1014—acid, contains a trace of albumen. During his short stay in the hospital, the boy remained in about the same condition; he was discharged with directions to attend the out-door department, but did not come again.

Remarks.—Peripheral neuritis, due to lead-poisoning, in children, is of particular interest to us in Australia, and has become familiar to the readers of the *Gazette* from the writings of Drs. Lochart Gibson and Jefferies Turner, of Brisbane. There has been no satisfactory reason given for its rarity of occurrence in Sydney, as compared with its frequency in Brisbane. As far as I know, these cases are the first recorded in Sydney. Of course it might be thought that cases do occur, but pass unrecognised: that can hardly be the case, as peripheral neuritis (apart from post-diptheritic paralysis) is so unusual in children as to at once compel attention.

Taking the cases in detail, the diagnosis in Case I. was finally established by the detection of lead in the urine. When the question of lead-poisoning was raised, I asked Dr. Cooksey, of the Public Analyst's Department, Board of Health (Sydney), to examine the urine: this he very kindly consented to do, and found distinct traces of lead and copper, the copper being, perhaps, due to some green paint being mixed with the brown. However, that is a matter of no importance, as small quantities of copper are not poisonous. It was after this that we obtained the history from the mother. The rapid improvement in the hospital was very satisfactory, and has been noticed before in similar cases. Case II. was quite easy of diagnosis, and belongs to the more familiar type of this disease. In this case no lead was found in the urine; probably owing to the case being a chronic one. How this boy got the lead was uncertain: perhaps the illness he suffered from two years before was lead neuritis; if so, a similar explanation to that in the first case would suffice, more especially as the boy is but half-witted.

I have brought these cases before the profession, as it is of the highest importance to the little patients that the true nature of their complaint should be recognised. My thanks are due to Dr. Blackburn for his assistance in the examination of the first case, and to Dr. Plomley, Resident Medical Officer at the Children's Hospital, for the notes of both cases.

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NOTES OF A CASE ILLUSTRATING THE SYMPTOMS OF TUMOR OF THE OCCIPITAL LOBES OF THE BRAIN.

By James Jamieson, M.D.

Of late years there has been a growing dissatisfaction with the doctrine of the existence of tolerant, or silent, areas in the brain. With that has come an increasing unwillingness to accept the notion of latent disease of structural or destructive kind affecting it. That there are regions comparatively tolerant of injury may be admitted, and that even large abscesses may form in the substance of the brain, with the production merely of symptoms of a very general kind, must of course also be admitted.

The white matter of the frontal, temporal, and occipital lobes has particularly been looked on as comparatively tolerant of disease or injury. But, with increasing exactness in clinical methods, and more careful observation of the grouping of symptoms, localising signs as regards even these areas have been formulated. There will probably never be such definiteness of knowledge about the exact location of destructive or irritative lesions of these regions as we have about those of the mid-brain, with its mapping out of motor areas. Experiments on animals, which have added so much to our knowledge of this part, cannot be expected to help us in a similar way with them. Helpful signs and symptoms there are—progressive changes of character or lessened power of judgment in tumors of the pro-frontal lobes, with perhaps development of aphasic conditions. Visual defects of a special kind may be very significant in those of the occipital lobe; notably word-blindness, and hemianopsia. But these by themselves can only point to some great region being involved, failing perhaps to tell us, in the latter case, whether the lesion is superficial or deep-seated.

It must be by means of clinical records, supplemented by *post-mortem* examinations—and these in large accumulation—that more exact differentiation will be made possible.

The following case—though, unfortunately, defective in details of early symptoms—has considerable interest, and so suggestiveness.

M. R., female, single, aged 30, was admitted to the Alfred Hospital on July 19th, 1904, with a rather vague history of increasing impairment of vision and loss of power, especially in the legs. She had been ill for three or four months, but had been confined to bed for about one month only. She was very ignorant and unobservant, and the same was true of any of her friends who could be questioned, so that details about the onset and early symptoms could not be got. There had not been vomiting, but she had been losing flesh. She could only say that for a month her sight had been very bad, and when examined it was found that she could not with either eye see fingers held up before her. When asked about headache she put her hand on the post parietal region on the right side as the seat of it.

Nothing of importance could be learned of the family or personal history. She gave no account of any previous illness, and there was no evidence of syphilis. There had been amenorrhœa for three months; the pulse was 99, respiration normal, temperature 97. The urine was acid, and free from albumen. The apex beat was in the normal position, and there was no sign of valvular disease or arterial change. The pulse tension was 124, and the red blood count and hæmoglobin measurement were but slightly below normal. There was no sign of lung disease, and the abdominal organs seemed to be healthy. The knee jerks were both exaggerated, but there was no ankle clonus, and the plantar reflex was active, but of normal character.

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Her eyes were examined by Dr. Gault, ophthalmic surgeon to the hospital, who reported that the media were clear and the discs normal. Previously to this it had been found that the pupils did not react to light, and it was also shown that there was external ophthalmoplegia, considerable in range though not very complete. Notably there was paresis of the external rectus, double, but most distinct on the right; and there was loss of power to roll the eyeballs up, especially on the left side. There was also apparent drooping of the upper lids; but these facts were ascertained with great labor and difficulty owing in part to the blindness and in part to the mental condition.

The loss of power in the limbs became steadily greater, and the leg reflexes became gradually less distinct till the knee jerk on the right side was lost, and that on the left obtainable only on reinforcement. The plantar reflex remained normal in character but became less active, especially on the left side. The loss of power also became steadily greater, and most in the legs—though the hand-grip was much weakened, and most on the right. Towards the end there came to be some right facial paresis, and Kernig's sign was found to be present on both sides. Her eyes were again examined on the 8th of August, with the result that both discs were still normal.

She died on August 10th, having become deeply unconscious on to coma.

An attempt to interpret the connection of these multiform symptoms, and to decide on the nature and seat of the lesion producing them, led to much discussion. The widespread paralytic condition associated with complete blindness—and that blindness not due to any retinal change, no atrophy or retinitis, no choked disc or œdema—made a somewhat unusual combination. And there were also the important features of absence of vomiting and fever, and of any sign or history of syphilis or of circulatory disease.

The steady progression of symptoms, without any sudden onset or rapid aggravation, corresponded best with the growth of some tumor, though the course was somewhat short, and two out of the three so-called cardinal signs of cerebral tumor were lacking. Blindness, of course, took the place of the usual retinitis, but it was manifestly blindness of some unusual kind. This blindness had to find some reasonable explanation on any supposition as to the pathological condition. It might be accounted for by some destructive pressure at the chiasma; by some bilateral lesion affecting the visual centres in the occipital cortex; or by some lesion cutting both optic tracts in their course between these centres and the optic nerves. For any lesion seated at the base and pressing on both optic nerves, and also implicating both pyramidal tracts and various cranial nerves, to be accepted as the cause was almost inconceivable in the absence of retinal congestion and other changes, and of bulbar or severe neuralgic symptoms—so apt to mark the growth of basal tumors.

The possibility of meningitis exerting destructive pressure on both visual centres and extending forward along the base was suggested by the presence of Kernig's sign. But useful as that sign sometimes is, it is not all pathognomonic of meningitis—and, indeed, is met with in cases of cerebral hemiplegia with various modes of causation. But a meningitis going on steadily, as this condition did, without rise of temperature, rigidity of the neck, or almost any of the ordinary signs of the disease, was not an acceptable solution of the problem.

The existence of a very large abscess, or of an abscess in each hemisphere, was excluded by the absence of fever or any sign of sepsis, even if there had been any cause discovered from which cerebral suppuration could have been supposed to take origin. No abscess could have lasted so long while also growing steadily—almost rapidly. We seemed to be left with a tumor of

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some kind, as best in accord with all the conditions. It almost certainly must have begun on one side—the left, near the middle line, growing both laterally and forward so as to cut the optic tracts and gradually involve the pyramidal tracts. On this supposition the blindness was regarded as being really a double hemianopsia.

The necessarily large size of the tumor thus postulated made the absence of retinal changes difficult to understand, though their very absence was in a way helpful in suggestion as to the nature of the blindness. The range of implication of cranial nerves, if this tumor was the sole cause, was also a difficulty. But brain pathology has not yet been so simplified that every symptom, in a complex group of this kind, can easily find full explanation.

Post-mortem examination revealed the substantial correctness of the diagnosis. There was difficulty in extracting the brain, the anterior aspect of the basal portion of which was firmly adherent to the dura mater, and the dura to the bone. There was found to be a soft, almost fluctuant, mass of gliomatous nature, involving the origin of the Sylvian fissure and extending backwards along the basal aspect of the temporo-sphenoidal lobe. It involved the left optic tract, and stretched across so as to impinge also on the right tract. The pons presented a curious white gelatinous layer immediately under the pia and firmly adherent to the pontine substance. On section the soft tumor was seen to extend back into the left lateral ventricle, not involving the internal capsule or the great basal ganglia, but just impinging on the external capsule and the claustrum. The ascending parietal region was slightly encroached on.

The condition found on the surface of the pons may be taken as accounting for the bilateral paresis of the external muscles of the eyeball, and possibly for the appearance of Kernig's sign. The reason for the absence of retinal signs with such a large tumor most probably was to be found in its softness, and in the fact that it grew rather by incorporation than by pressure displacement of the structures in its path.

A case of tumor of the centrum ovale, presenting some likeness to that here recorded, was reported in the *British Medical Journal*, May 21st, 1904. The special points made were the wide range of symptoms produced and the absence of optic neuritis.

PRELIMINARY COMMUNICATION ON THE NO-FOOD TREATMENT OF TYPHOID FEVER.

By O. L. M. Abramowski, M.D., Berlin, Mildura, Victoria.

Mildura, situated on the River Murray, in Victoria, is well known over the whole Commonwealth as an irrigation settlement, producing the finest crops of fruit in Australia. But there is one crop in Mildura produced annually, from November to May, which is only known to the local residents and to the Department of Public Health in Victoria: I mean the crop of Typhoid Fever. This made its appearance soon after the first people erected their tents on the bank of the river in 1888, and has been with us ever since. Up to January, 1903, 270 cases of this disease have been registered, 35 of which ended fatally —a death-rate of 13 per cent.; 175 of these cases were treated in the local hospital, with a death-rate of 25, or 14 per cent.—being, on the average, the

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more serious cases. The causes of death were—Paralysis of the heart, 33 per cent.; perforation and peritonitis, 25 per cent.; hæmorrhage, 19 per cent.; congestion of lungs, 13 per cent.; congestion of kidneys, 5 per cent.; pneumonia, 5 per cent.

Since February, 1903, I treated 63 cases of typhoid fever—42 in the hospital and 21 in private practice—without one death. According to the previous death-rate this means a saving of more than eight lives. They were all typical cases of our own local typhoid, with the same symptoms as the previous cases. The diagnosis was not confirmed by Widal's test, and only in two cases by the Diazo-reaction; but roseola appeared in 48 cases; the typical fever with recrudescences, &c., in 10 cases, hæmorrhage from the bowels in 3, and relapses in 2: so that there can be no doubt about the true nature of the affection.

The triennial death-rate from typhoid in Mildura is as follows:—1891 to 1893, nine; 1894-6, eight; 1897-9, nine; 1900-02, nine; 1903, January, two; 1903, February, to 1905, January, none.

The general death rate in the local hospital was, for the year 1900-1, 14 per cent.; 1901-2, 15 per cent.; 1902-3, 12 per cent.; 1903-4, 8.3 per cent.; 1904-5, 7 per cent.; and in my last report (July A.C.) I attributed this reduction to the elimination of typhoid as a cause of death! This astonishing, and, as far as I know, quite unique result, was caused through the no-food treatment.

Every year, since I passed my medical examination in 1875, I have been treating typhoid patients, and with every patient my anxiety was renewed regarding his diet. We all know that the intestinal lesions and the tendency to diarrhœa, hæmorrhage, and perforation of the bowels in typhoid call for the most careful dietetic *regime*, in order to preserve, as far as possible, intestinal antisepsis. It is clear that as little unabsorbed food as possible should be allowed to accumulate in the intestine, there to undergo fermentation and decomposition. With fever patients the question is almost uppermost—Can they digest and assimilate any food? The great emaciation of typhoid patients who had been fed with the most nourishing diet always seems to prove to me that their food was absolutely wasted. When, in 1902, I read the admirable work of Pawlow on "The Work of the Digestive Glands," it became clear to me that feverish patients cannot digest and absorb food. Pawlow's extremely interesting experiments prove beyond a doubt the correctness of his contentions—that the appetite is the first and mightiest exciter of the secretory nerves of the salivary glands and of the gastric glands; that the work of any section of the alimentary canal is connected with and dependent upon that of the previous segment; and that the saliva is necessary, not only for mouth-digestion, but also for stomach-digestion; that without saliva there will be no production of gastric juice, without gastric juice there will be no secretion of pancreatic juice: consequently, without saliva there will be no digestion and no assimilation of the food.

Has our medical practice acted upon these principles? Decidedly not. On the contrary, we have usually acted in direct opposition to these observations by forcing our patients to eat when mouth and tongue were dry, and when the most appetising dishes could not make their mouths water. We have fed our patients in order to keep their strength up, under the mistaken impression that a fast of a few days means risking the patient's life. At the same time we did not reckon the cost on strength and energy incurred by the body's endeavor to push on the undigested food, put into an unwilling stomach, and to get rid of a fermenting and decomposing mass, producing poisonous substances through the whole length of the alimentary canal. The latest publications with regard to diet in typhoid advise even a more generous fare

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than has so far been considered correct, and try to ignore altogether those physiological truths proved by Pawlow. At any rate, Pawlow's experiments convinced me that the untoward accidents observed in typhoid patients under every diet are the absolutely natural consequences of forced feeding, *i.e.*, of the feeding in the absence of the first and mightiest factor in digestion, viz., the appetite juice. This conviction was the basis of the no-food treatment of typhoid; and when, in December, 1902, our typhoid epidemic began, I decided to put it into practice. However, it took me some time to overcome my own prejudices, and during December, 1902, and January, 1903, I treated ten typhoids with abstinence from food for from eight to ten days only. I observed a decided diminution of dangerous and distressing symptoms; but I had not the courage to refuse food until the natural appetite, with sufficient production of saliva, appeared; and I lost two of these cases—one a boy of 11, on the seventeenth day, with pneumonia; the other a man of 37, weakened through abuse of alcohol, on the twenty-eighth day, with heart failure. At this time I read about a book by Dr. E. H. Dewey, entitled "The True Science of Living," but could not get it in Australia, and had to order it from America. Before the book came to hand I had carried my theory into full practice, and had treated twenty-two cases of typhoid with total abstinence from food until they produced enough saliva to properly masticate and enjoy a hard dry cabin biscuit. Great was my satisfaction when I found that Dr. Dewey had come to the same conclusion as I, although starting from other premises; and his success in typhoid treatment encouraged me to go on and to perfect my own treatment. I found Dewey's observations correct, that after three or four days of enforced fasting—that is, after the habit-hunger had been overcome—there was usually no desire for food at all: but that an unmistakable hunger appeared with the saliva, usually at the end of the pyrexia. Sometimes this desire for food was delayed after the temperature got normal and the tongue got moist, and even after the patient had left the bed. Thus, in case 17, the patient fasted—because his hunger did not appear earlier—for fifty-one days; eleven days beyond the pyrexia. Number 25 was up and doing clerical work for three days before he started eating on the twenty-first day; and number 26 was walking about and doing little jobs about the place for five days before he could chew and enjoy the biscuit, after a fast of twenty-four days. I have added a few tables giving the highest temperature, the duration of pyrexia, of fasting, of convalescence, and of the whole time of observation and remarks, re-complications, &c., in every case; and shall here only enumerate the general results of these statistics and the most prominent complications, &c.

The temperature was usually highest on admission, or at the beginning of the treatment. In the hospital patients it never rose to this height again in twenty-four cases; in twelve cases it was highest on the sixth day; in five cases only it rose above the initial height after the sixth day. The private patients, being always the milder cases, had all their highest temperature on the first day of observation.

The length of pyrexia amongst the hospital patients averaged 21 days; amongst private patients, 11.2 days; amongst the whole number, 18 days. The usual evening temperature was from 101° to 102°.6 amongst all the patients. Amongst the hospital patients it reached 102° in nine cases, from 102° to 103° in twelve cases, from 103° to 104° in fifteen cases, 104° and over in six cases: and all these higher temperatures were only observed on a few days—usually on the first days of treatment. The temperatures of the private patients were not regularly registered, but they were considerably lower. The time of fasting was, on the average, in the hospital 22 days, amongst private patients 11.2 days; giving a total of 18.3 days. The average duration of the convalescence was 20 days in the hospital, 10.8 days in private practice;

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or, amongst the whole number, 16.5 days. The average duration of the whole case—that is, from the beginning of the treatment until the patient could resume light work—was, amongst hospital patients 41 days, amongst private patients 22 days; a total average of 34.5 days, or about five weeks. Complications were very few and mild. Perforation, usually observed in about 4 per cent. of all cases, was totally absent; hæmorrhage was seen only in three cases; delirium of short duration in four cases; recrudescence of fever in four cases; relapses in three cases; diarrhœa in four; sleeplessness and headache in three; abscesses and furuncles in three; albuminuria in two; meningitis in one; neuralgic pains in one; and double suppurating parotitis in one case. All the other complications were conspicuous by their absence; besides perforation we never saw vomiting, meteorismus, singultus, pneumonia, or bronchitis. Especially grateful was I for the complete absence of thrombosis, which, in years past, had crippled a great number of our patients.

The usual remarks in the case-book were: Patient slept well, feels comfortable, easy and cheerful, sensorium quite free, reading, &c.; and this in the second and third week, when under ordinary treatment, the typhoid patients were usually delirious, flushed, and restless. Ten of the hospital cases—those marked with a cross on the tables—were so severe and so protracted, and showed so little power of resistance, that under usual treatment they would have probably died.

The no-food treatment which proved so successful in Mildura during the last three years does not consist in the abstinence from food only. The patients were allowed pure sterilised water as much as they wanted; and it was astonishing how little some would drink, not taking more than one or two pints a day, whereas others—usually great salt eaters—would drink from five to six quarts in a night. Every day in the beginning, and every second day after the motions had lost their smell, the patients got an enema of sterilized water of about 90°: this removed some fæcal matter almost every time; the fæces soon lost every trace of diarrhoic or pea soup characteristics, and took the shape of round or oblong masses of the size of a marble or a walnut, and almost free from any smell, and of a greyish-brown color. Even after the fast of fifty-one days these excretions continued to appear at intervals of from three to five days. In four cases, after a fast of from fourteen, sixteen, twenty, or twenty-four days, respectively, and regular flushings of the colon, a teaspoonful of castor oil removed big motions—once nearly a chamberful. This brought in every case a recrudescence of the fever, of short duration. Excepting a few doses of acetozone in the first few cases, a few cold baths and spongings during high pyrexia, and an occasional dose of castor oil (as above), no other treatment was resorted to. Drugs or alcohol were not given—excepting a few doses of 15 min. of Tt. Opii in the three cases of hæmorrhage; but, in my present opinion, even that is not needed as long as the digestive canal is empty. The cases of recrudescence, or relapse, cheerfully submitted to another fast as soon as the fever re-appeared, and never gave any further trouble.

Delirium, headache, sleeplessness, usually subsided after the first three or four days. Only in two cases of a very prolonged pyrexia (numbers 20 and 32) the delirium lasted in a mild form three and four weeks, respectively. These two cases—of which one was a very frail hysterical woman, and the other was complicated with suppurating double parotitis, and both of which developed abcesses in the fifth and eighth weeks, respectively—would surely have succumbed under any other treatment. All the other complications, such as diarrhœa, albuminuria, meningitis, furunculosis, disappeared without any further interference, medicinally or dietetically.

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The convalescence in every case was uninterrupted, and there was never any trace of that protracted invalidism we so often see after the usual treatment. When once these patients started to eat, they made a marvellous recovery. It seemed that after the fast the system is in such a splendid condition for assimilation of food that every morsel given is made use of to its utmost capacity. The feeding usually started with fruit and fruit juices, oranges, bananas, grapes, followed by milk, biscuits, triscuits and shredded wheat, porridge, puddings, and eggs. Bread, meat, and vegetables were usually the last addition to the diet. All the convalescents had a good appetite; there was no picking or daintiness about the food, and the increase in weight and strength was rapid. As for the loss of weight through the prolonged fast, I am sorry I was not able to ascertain it on the scales in more than three cases: the first, a very fat man of fourteen stone, reduced his weight by four stone after twenty-eight days' fasting; the second lost two stone after twenty-four days' fasting; the third twenty pounds after twenty-one days' fasting. Generally speaking, these convalescents were not thinner, but certainly much stronger and heartier than all those treated before by me.

The most pleasing feature of these fasting patients was the absence of the usual urgent and distressing mental and bodily symptoms; so that, in a ward full with cases of typhoid in the severest stage, there was no delirium, no mutterings or carpholagia, no cough, no tympanitis, no diarrhœa or involuntary discharge from the bowels or bladder, no decubitus or restlessness; the tongue was usually clean, and, to a certain extent, moist; there were no sordes on lips or teeth. During the night, and very often during the day, the patients would sleep quietly and peacefully, like children, so that the duties of the night-nurse were very light indeed.

Before the merits of the no-food treatment were known amongst the people it took sometimes a certain amount of persuasion to induce the patients to submit to the fasting: but even then they complained about hunger only for two or three days. After that they found the water quite sufficient for their wants, and would gladly acknowledge how much better they felt now than whilst taking food, before they had my advice. If after a week or so their faith began to flag, and they expressed the opinion that a little food would not hurt them, I did not absolutely decline, but would allow them a small quantity of milk or plasmon, and wait for the result. At the afternoon visit their temperature would be high, the pulse rapid, the tongue dry, the mouth nasty and sticky, stomach and bowels distended and disagreeable. After this it did not need any further persuasion to return to the water diet. With some patients we had two or three trials like that; but the effect of even the slightest dose of food was so sure and so quick to raise the temperature and render the patient uncomfortable that we now know to a certainty "that feeding keeps up fever."

Infants are the easiest to treat in this way, as they will be quite satisfied with water as long as they are feverish. But children used to three meals or more a day will give you very little peace; and, until you have the hearty co-operation of parents or friends, it is a hard task to constantly refuse the little sufferers what they seem to want so apparently. But when after a few days the temperature is low, and a trial is made with the lightest food, the effect is so palpable in rendering the patient worse that the parents soon get convinced; and after a second trial I always had them in favor of water against food, and the further progress of the case would be undisturbed.

The no-food treatment has robbed typhoid of most of its horrors, has reduced the time of unfitness to work from the usual three months to about five or six weeks, has not only secured my patients a comparatively easy time,

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but has made patients and nurses, parents and friends, during the whole time, so cheerful and free from anxiety that it always was a great pleasure and satisfaction for me to be with them and to encourage them just for a day longer of fasting and water. I did not think it wise in the beginning to frighten the patients by telling them that they might have to fast from two to four weeks or more; and the gradual extension from day to day they did not seem to mind so much.

At the present time, since the beneficial effects of the no-food treatment have become known through the district, people in Mildura do not dread typhoid any longer, and willingly undergo the fasting; but when first I started the treatment, I had not only the resistance of the patients and their friends, but also the prejudices of the nursing staff to overcome; and many a lecture I had to deliver inside the ward and outside of it on the benefit of keeping an ulcerated intestinal canal free from any fermenting and dangerous substance.

The number of cases treated by me with abstinence from food has not been as large as I would have wished, and my observations are far from being conclusive: but the subject is of such great importance to medical men, and humanity generally, that I did not feel justified in withholding my experience until I could put a greater number of cases before the profession.

The difference between the usual style of feeding and this no-food treatment is almost incredible to anyone who has not tried it, and I therefore beg to place these short and incomplete observations before the profession of Australasia, hoping that they will soon be verified by the experience of others, and that the no-food treatment will prove in their hands the same blessing for suffering humanity as it has proved in mine.

SUMMARY OF A SERIES OF 63 CASES OF TYPHOID UNDER THE NO-FOOD TREATMENT.

	Average Duration (in Days) of—												Remarks.
	Pyrexia.			Fasting.			Convalescence.			Total Observations.			
	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female.	Total.	Male.	Female.	Total.	
Hospital patients	19·5	25	21	20	25	22	19	20	20	38	45	41	Severe cases
Private patients	10·7	12·2	11·2	10·7	12·2	11·2	11·1	11·4	10·8	21·8	22·6	22	Light cases
Total ..	16·5	21·8	18	16·8	21·8	18·3	16	15·1	16·5	32·4	37·9	34·5	

Diagnosis confirmed by roseola in 48 cases; typical fever and hæmorrhages in 2 cases; typical fever and recrudescences in 11 cases; typical fever and relapses in 2 cases.

Highest temperature (hospital cases only): On admission in 24 cases, before sixth day in 12 cases, on and after sixth day in 5 cases; up to 102° in 9 cases, 102°.1 to 103° in 12 cases, 103°.1 to 104° in 15 cases, more than 104° in 6 cases.

Complications: Delirium, diarrhœa, recrudescence in 4 cases each; relapse, hæmorrhage, headache, sleeplessness in 3 cases each; albuminuria and furunculosis in 2 cases each; abscesses, neuralgic pains, parotitis, duplex suppurat in 1 case each.

Recovery in 63 cases; death in none.

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A SERIES OF 63 CONSECUTIVE CASES OF TYPHOID UNDER THE NO-FOOD TREATMENT.

No.	Name.	Age.	Diagnostic Symptoms.	Temperature, Highest.	Duration (in Days) of—				Date of—		Remarks.—Complications.
					Pyrexia.	Fast.	Convalescence.	Stay in Hospital.	Admission.	Discharge.	
				HOSPITAL.—MALES.							
1	B. D.	37	Roseola	101·5° on admission..	13	14	18	32	31-1-03	4-3-03	Normal course
2	A. McK.	45	"	102·8° " ..	16	19	5	21	16-2-03	9-3-03	"
3	H. J.	20	Typical fever	102·0° " ..	18	18	8	26	17-3-03	4-4-03	Short relapse after 4 days normal
4	J. H.	35	Roseola	102·5° " ..	15	14	7	22	3-4-03	25-4-03	Normal
5	W. C.	29	"	102 3°	19	19	3	22	3-4-03	25-4-03	Sleeplessness and headache on admission for 5 days; bilious subject
6	A. M.	23	"	102·2°	14	14	4	18	6-5-03	24-5-03	Normal
7*	Ch. B.	18	Typical fever	103·6° on 4th day....	33	33	39	72	28-4-03	9-7-03	Hæmorrhage on 11th and 13th days; mother and sister have died of hæmorrhage in typhoid
8	G. A.	27	Roseola	104·0° on 3rd day....	17	17	16	33	4-5-03	6-6-03	Milk and water after 8 days increased fever and gave pain in stomach
9	R. R.	31	Typical fever	102·0° on admission..	32	23	29	61	9-5-03	9-7-03	Relapse 9 days after normal, with albuminuria
10	H. H.	20	Roseola; spleen	102·6° on 2nd day ..	9	10	21	31	4-12-03	4-1-04	Diarrhœa on admission, with very foul peasoup stools
11*	S. B.	25	Roseola; headache; epistaxis	103·4° on 20th day ..	27	35	33	68	21-12-03	27-2-04	Recrudescence on 17th day, after dose of castor oil
12	E. B.	36	Typical fever; pronounced smell	103·0° on 7th day....	22	20	12	34	7-1-04	10-2-04	Albuminuria disappeared with fever
13	S. W.	22	Roseola	103·4° on admission..	16	20	10	30	28-1-04	27-2-04	Normal
14	T. C.	33	"	104·8° " ..	12	15	13	28	5-2-04	5-3-04	Delirious first 3 days; after that always rational
15	A. G.	25	"	103·2° on 2nd day ..	9	12	6	18	15-2-04	5-3-04	Headache; characteristic smell
16	F. L.	9	"	103·4° on admission..	8	4	1	9	17-2-04	26-2-04	
17	D. F.	45	"	103·6° on 4th day....	40	51	17	57	17-2-04	13 4-04	Recrudescence on the 21st day; very fat stout man; walking, reading, writing 14 days before eating
18	G. G.	19	"	103·0° on admission..	8	8	25	33	21-2-04	27-3 04	Ill 14 days before admission
19	M. Y.	41	"	102·0° " ..	36	36	14	50	24-2-04	15-4-04	Recrudescence after castor oil on 22nd day
20*	H. N.	15	"	104·8° on 2nd day ..	36	36	70	106	9-3-04	23-6-04	Parotitis duplex suppurat; abscesses on back; delirious at times

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21	R. D.	24	"	104·8° on 16th day ..	36	36	31	67	12-3-04	18-5-04	Rigor on 16th day, followed by diarrhœa, which ceased after 10 days, spontaneously
22	J. McL.	24	"	102·0° on 4th day....	13	13	11	24	20-3-04	13-4-04	
23	W. H.	25	"	101·6° on admission..	14	14	9	23	21-3-04	15-4-04	
24	A. St.	14	"	102·4° " ..	14	11	19	33	15-12-04	17-1-05	
25	W. E.	30	"	103·4° on 6th day....	18	21	16	34	25-1-05	28-2-05	On admission, sleepless; headache for 4 days; doing clerical work before eating
26	Th. McD.	50	Roseola; diazo-reaction	102·6° on 3rd day....	12	24	23	47	31-5-05	18-7-05	16th day, cannot eat biscuit; teeth sore. 24th day, never enjoyed anything like it; walked about before eating; lost 4 stone
					FEMALES.						
27*	M. McPh.	23	Typical fever; hæmorrhage	103·0° on 3 occasions.	31	30	21	52	21-2-03	14-4-03	Sleeplessness; hæmorrhage 13th, 15th, 17th days
28*	M. L.	26	Typical fever	104·0° on admission..	52	32	24	76	17-3-03	1-6-03	Two recrudescences after attempts to eat; very weak woman, anæmic
29	E. O.	16	"	103·5° " ..	36	36	25	61	21-4-03	21-6-03	Two recrudescences after attempts to eat
30	L. Cl.	48	Roseola	101·5° on 3rd day....	15	22	21	43	6-6-03	19-7-03	Comfortable while fasting; uncomfortable with barley water
31	N. B.	22	Roseola; pea-soup stools	103·6° "	22	22	25	47	8-7-03	22-8-03	On admission, diarrhœa, lasted 6 days; 18th day, milk and water; increased temperature
32*	E. B.	29	Headache; epistaxis; typical fever	†104·5° on 15th day ..	35	24	31	66	2-2-04	9-4-04	Insomnia; hysterical; retention urine; abscesses on head
33*	M. H.	8	Roseola	103·6° on 6th day....	24	24	18	42	8-4-04	20-5-04	Diarrhœa for 7 days; stopped through fasting
34*	L. M.	18	"	104·0° on 4th day....	20	18	23	43	11-5-04	23-6-04	Very fat; tremulous on admission, and delirious for 3 days
35	A. C.	5	"	102·2° on admission..	5	4	4	9	12-5-04	20-5-04	Was ill some time before admission
36	M. St.	17	"	103·0° on 10th day ..	20	20	10	30	15-12-04	8-2-05	Headache; epistaxis
37	M. M.	9	"	103·0° on admission..	7	7	24	31	24-1-05	23-2-05	
38	M. G.	28	"	104·2° " ..	30	35	22	57	31-1-05	28-3-05	19th day, castor oil; very big motion; recrudescence
39	F. C.	7	"	104·0° on 3rd day....	28	29	28	57	25-2-05	23-4-05	Boils on head
40*	L. B.	17	"	104·8° on admission..	22	23	28	51	3-3-05	23-4-05	Fat; delirious first 5 days; hæmorrhage on 16th day, small
41*	Ph. A.	5	"	104·0° " ..	27	26	13	40	14-3-05	23-4-05	Meningitis
42	E. B.	25	Roseola & diazo-reaction	103·6° " ..	26	24	21	47	2-6-05	19-7-05	Three eruptions of roseola; always hungry; bad smell

* Would probably have died under ordinary treatment. † Extremely hot weather, 114°.

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Cases of Typhoid Treated under the No-food Treatment—continued.

No.	Name.	Age.	Diagnosis Based on—	Highest Temperature.	Days of—				Date of Beginning of Observation.	Date of End of Observation.	Remarks.
					Pyrexia.	Fast.	Convalescence.	Observation.			
				PRIVATE.—MALES.							
43	A. F.	45	Roseola	103·4° after eating ..	14	14	9	23	14-2-03	9-3-03	On 9th day and 14th day temperature rose after taking some food; fell again by fasting
44	M. H.	19	"	103·0° on 1st day....	10	10	10	20	23-2-03	15-3-03	
45	G. McN.	28	"	102·5° "	7	7	7	14	18-4-03	2-5-03	Had typhoid 10 years ago
46	W. E.	34	"	103·0° "	7	7	6	13	21-4-03	4-5-03	
47	R. N.	34	Typical fever	103·2° "	10	12	11	21	4-5-03	25-5-03	Had typhoid 10 years ago
48	W. M.	27	Roseola	103·0° "	8	8	4	12	27-1-03	8-2-03	" "
49	F. E.	40	Typical fever; smell	102·5° "	10	10	6	16	1-12-03	15-12-03	Lives in house of 59
50	H. W.	21	Roseola	102·6° "	14	14	17	31	5-12-03	5-1-04	
51	W. M.	44	"	103·3° "	15	15	27	42	12-2-04	26-3-04	
52	R. V.	50	Typical fever	103·2° "	12	12	16	28	16-2-04	15-3-04	
53	Ch. C.	15	Roseola	103·4° "	12	12	24	36	7-4-04	12-5-04	Brother of 55
54	J. M.	8	"	103·0° "	10	10	11	21	16-1-04	6-2-04	
55	Cl. C.	10	Typical fever	102·5° "	10	10	7	17	21-6-04	8-7-04	Brother of 53
				FEMALES.							
56	E. L.	10	Roseola	103·2° "	12	12	5	17	28-4-04	14-5-04	
57	M. C.	5	"	103·5° "	14	14	13	27	29-4-04	25-5-04	Temperature rose every time after taking food first week
58	L. D.	13	"	102·7° "	10	10	15	25	28-5-04	21-6-04	
59	S. M.	45	Typical fever; smell	103·0° "	14	14	5	19	18-1-04	5-2-04	Lives in same house as 49
60	N. M.	18	Roseola	103·4° "	15	15	6	21	31-1-04	20-2-05	Daughter of 59
61	E. B.	38	"	102·6° "	12	12	14	26	15-2-04	13-3-04	
62	S. T.	14	Typical fever	102·5° "	7	7	17	24	16-2-04	12-3-04	
63	M. A.	13	"	103·6° "	14	14	8	22	28-12-04	19-1-05	

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THE NO-FOOD TREATMENT OF TYPHOID.

DR. SPRINGTHORPE remarked that Dr. Abramowski's results, if accurately recorded, showed what the human frame could resist. Hitherto he had regarded forty days' fasting as illustrating either miracle or mal-observation. It was unfortunate that there was insufficient proof that the cases treated were really typhoidal. No doubt many cases used to receive too much food, but it was illogical to suppose that if food could be given without any mal-assimilation, as it could, the system was better without it, and it was notorious that the patient's desire was by no means a safe guide as to quantity and quality. It was unwise to use the word "impossible" outside mathematics, but it was scarcely possible to regard Dr. Abramowski's paper as conclusive.

IS THERE SUCH A DISEASE AS CROUP?

BY HARVEY E. ASTLES, M.D., F.R.C.P.E.

I have written this short paper chiefly to show the different opinions that exist on this subject, and to endeavor to arrive at a satisfactory conclusion.

Mr. Edmund Owen, Surgeon to the Great Ormond Street Children's Hospital, London, writes as follows:—Croup, diphtheria, and membranous laryngitis are forms of the same disease.

Dr. Osler, late Professor of Medicine at the Johns Hopkins University, and now Regius Professor of Medicine at Oxford, states, in his "Practice of Medicine":—That membranous croup is either genuine diphtheria or diphtheroid in character; and, in 286 cases in which the disease was confined to the larnyx or bronchi, 229 Klebs Loeffler's bacilli were found. It is to be regretted that further particulars of the fifty-seven cases in which it was not found had not been given.

Dr. Foord Caiger, in Allchin's "Practice of Medicine," speaking of the extreme difficulty of diagnosis, says:—It practically resolves itself, at the present, into whether or not the Klebs Loeffler bacillus can be detected in the discharge.

Dr. Robert Macguire affirms, in his article in "Quain's Dictionary of Medicine":—There remains still a certain proportion of cases of membranous laryngitis which are not due to the actions of diphtheritic poison.

Hilton Fogge believed in non-contagious membranous croup.

Whitlaw is of opinion that different writers have described two totally different affections under one name. Dr. Whitlaw's position as a physician to the Belfast Hospital and Lecturer on Clinical Medicine, together with his reputation as a physician and author, enables him to speak with authority when he says the affection known as true croup in the north of Ireland—and which presents the chief clinical features described by French and English physicians—is not diphtheria, but a non-contagious acute inflammation of the air passage.

The pathology of croup is a congested condition of the blood vessels, producing a swollen mucous membrane with the inflammatory exudation, which may or may not form a false membrane.

Dr. Lazarus Barlow, in his "Manual of General Pathology," touches at the very root of the matter. He says: "Now, in the neighborhood of the pharynx and larynx two classes of cases have long been known, in which inflammation is accompanied by the formation of a membrane. In the one, croup, the membrane is superficial; in the other, diphtheria, it is interstitial. Either condition produces more or less laryngeal stenosis, with hoarseness of voice

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and continued difficulty of breathing. In croup we have an inflammatory disease, non-contagious, with little or no tendency to complications, nor is it followed by paralysis.

Before the days of Loeffler bacillus, I can remember many cases when, by the aid of emetics, complete casts of the trachea were brought up. These casts were then diagnosed from diphtheria by the comparative facility with which they were detached and the thinness of the membrane itself, together with the absence of membrane from the tonsils and pharynx: no undue swelling of the glands, the freedom of the urine from albumen, no history of contagion, nor any infection arising from cases diagnosed as croup. Nor were there precautions taken to prevent it.

The following is a very fair example of a case of croup. I was called in to see a boy of 7 years of age. His mother stated that he had been ill for three days, suffering from croup: she said he was subject to it; but on former occasions, after a bath warm, a hot poultice to the throat, and ipecacuanha wine, he soon recovered. On this occasion it had not given way to the usual treatment. He was living in a low-lying locality near the Swan River, and had a temperature of 102°, voice hoarse, and continued bad breathing, with great recession of the thoracic wall. As far as could be seen the throat was clear of false membrane, but the mucous membrane looked congested, and was exuding a good deal of mucus. There was no glandular swelling, no albumen, or any history of contagion. I am unable to say whether there was a false membrane in the larynx or trachea; but the stenosis was bad enough for it to be there. I ordered him iodide of potassium, aconite, and ipecacuanha. The hot poultices to be continued, and to breathe through a sponge wrung out of hot water. This boy made a good recovery; and although there were other children in the house who had frequent access to the room, not the slightest sign of illness occurred amongst them.

A short time ago I was called in to see two little girls (sisters), who were suffering from what I diagnosed as follicular tonsilitis. They made a rapid recovery. A few days afterwards an infant, fifteen months old (one of a family of five), was taken ill in the same house. There was extreme difficulty of breathing, a hoarse voice, and rise of temperature. There was no swelling of the glands, no false membrane on tonsils or pharynx; but the stenosis was sufficiently grave for the probability of its existence lower down. I stated the case to be one of croup. In follicular tonsilitis its etiology is put down to either insanitary surroundings, a damp situation, or a wetting. This house was situated in a damp locality, but otherwise its sanitary arrangements were good. The infant made a good recovery, and no case of illness occurred with the other four children, nor was it followed by any further outbreak in the neighborhood. I am of opinion that there are undoubtedly localities where croup is seldom or ever seen, and I believe these places have given birth to the belief that true croup is either genuine diphtheria or diphtheroid in character. In localities where croup is prevalent practitioners, as a rule, become expert in their differential diagnosis.

The name Croup—which is derived from the Saxon Kroopan, "to cry aloud"—is certainly misleading, and is a better appellation for laryngismus stridulus.

Dr. Dawson Williams, in his work on "Diseases of Infancy and Childhood," speaks of croup as acute laryngitis. The symptoms, he says, are identical with those of laryngeal diphtheria, and it is often impossible to be certain that laryngitis is not diphtheria. I would say, if there is a doubt, certainly use anti-toxin as well as your croup remedies.

It is, to my mind, an important matter that this subject should be cleared up—especially for medical students, who, when qualified, should commence practice with as clear a conception of these diseases as possible.

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ON LEUKÆMIA AND ALLIED CONDITIONS IN CHILDREN.

By J. M. Gill, M.D., Lond., Sydney.

Leukæmia in children is rare, and the thorough examination of such cases apart from hospital practice very difficult: moreover, the accounts given of this disease in the various textbooks of children's diseases and of hæmatology is so imperfect that I have been led to publish these cases.

First of all, it is necessary to bear in mind the chief differences between normal blood in children and in adults. The most striking is the much greater proportion of lymphocytes in children, small lymphocytes forming 50 to 70 per cent. of all leucocytes in children, instead of 20 to 30 per cent. in adults; while large lymphocytes form 6 to 14 per cent. instead of 4 to 8 per cent. in adults. Again, in children nucleated red corpuscles readily appear in any case of severe anæmia, and quickly disappear on recovery

I propose to submit to your consideration to-day three cases of leukæmia, and one of the condition generally known as "anæmia infantum pseudo-leukæmica" ("splenic anæmia of children" of some writers): these being all the cases of the kind which have come under my care during the last two years. Of the cases of leukæmia one, of the acute myelocytic type, has been already published in the *Australasian Medical Gazette* for October, 1904; the second was of the acute lymphatic type; while the third is of the "spleno-medullary" form, the patient being still alive.

The first patient was a boy aged 3 years, who came under observation in August, 1903, and died after a fortnight in the hospital. There was no enlargement of the spleen, and the only glands enlarged were the submaxillary ones. There were many small subcutaneous hæmorrhages. The blood count was as follows:—Red cells—1,900,000 falling to 1,000,000 shortly before death; white cells—35,000 rising to 115,000 before death; hæmoglobin—not taken; polynuclears—32 per cent., the last count was 18 per cent.; lymphocytes—41 to 33 per cent.; myelocytes—25 to 45 per cent. shortly before death; eosinophiles—varied between 1 and 2 per cent. No nucleated red cells were seen. The child was not at all rachitic, and there was no suspicion of congenital syphilis. No autopsy was allowed.

The second case was in a boy aged 2½ years, who came under observation in October, 1903. This was a typical case of acute lymphatic leukæmia. There was a history of about two months' indefinite illness, and of getting rapidly worse during the last two weeks. On admission he was very anæmic, with wide-spread small subcutaneous hæmorrhages; none extensive. There was almost universal moderate enlargement of the lymph glands, generally to the size of a small marble. The spleen was slightly enlarged, and extended to about 2in. below the edge of the ribs. The liver was also enlarged. He was not rachitic. There was no membrane on the tonsils, and no ulceration of the gums. The temperature was up all the time he was in the hospital, varying between 99 and 101. He died after eight days in the hospital. The blood condition was as follows:—Red cells—1,760,000 on admission; 1,144,000 the day before death; white cells—43,000 on admission, 80,000 the day before death; hæmoglobin—35 per cent. on admission; lymphocytes—96 per cent., mostly small; polynuclear—3 per cent.; myelocytes and eosinopiles, 5 per cent. each; nucleated red cells—absent. At the *post-mortem* examination the typical appearances of acute lymphatic leukæmia were seen.

The third case is an example of the spleno-medullary type, so familiar in adults but rare in children. This child is a girl 6 years of age admitted quite recently into the Children's Hospital for hæmorrhage after tooth extraction. There is a history of being ill two and a half years with anæmia. On admission she is very anæmic. There is an enormous spleen reaching down

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to the iliac crest, but no enlargement of the lymph glands. During her stay (nine days) in the hospital the temperature was irregular, rising to 101.4 twice. She was taken out by her parents. Blood examination :—Red cells—1,520,000; white cells—208,000; hb. value—30 per cent. Differential count :—Polynuclear—50 per cent; myelocytes—30 per cent.; mast cells—9 per cent.; lymphocytes—large, 5 per cent.; small, 2 per cent.; eosinophiles—4 per cent. Two nucleated red cells were seen in counting 300 leucocytes. She is not rachitic.

The fourth case was one of "anæmia infantum pseudo-leukæmica." This child is a girl, 1½ years old on admission to the hospital in November, 1904. She has been bottle-fed since 2 months of age, and has had diarrhœa off and on ever since 12 months of age. She was first noticed to be rachitic about six months ago. On admission it is noted that the child is rather anæmic, has well-marked rickets with bow legs, and a distended abdomen. The spleen is very much enlarged, and extends down almost to the crest of the ilium. There is some diarrhœa, with three or four motions a day. The temperature rises at night to 102 or thereabouts. On December 2nd it is noted that the child is brighter, the temperature is normal, and that the diarrhœa has ceased. The spleen is a little smaller. She takes her food well and is contented. After that she continued to improve rapidly till, on discharge, she was practically well; the spleen being hardly palpable, although the abdominal distension was still present. She was given hydrarg C creta ½gr., *bis die*, till her motions became normal. There were no signs of congenital syphilis.

Date.	Red Count.	White Count.	Hb.	Polynuclear.	Lymphocytes.	
					Large.	Small.
1904.			Per cent	Per cent.	Per cent.	Per cent
November 23.....	3,700,000	31,500	60	44	6	31
	(Another differential count the same day)		—	36	40	
December 4	?	20,000	?	42.4	5.2	32
December 7	5,200,000	18,400	?	40.5	7.5	33
December 21	?	12,000	?	38	44	
January 13, 1905..	4,700,000	7,000	?	30.5	65.5	

Myelocytes.	Eosinophile.	Mast Cells.	Remarks.
Per cent.	Per cent.	Per cent.	
14	4.	1.	Three normoblasts seen while counting 250 white cells, a few megaloblasts seen.
19	—	—	—
16	3.6	.8	Six normoblasts and one megaloblast seen in counting 250 cells, red cells normal.
12.5	5.75	.75	Two normoblasts seen in counting 220 cells and two megaloblasts.
15	3.0	?	Two normoblasts seen in counting 100 cells.
2.	2.	—	No nucleated red cells seen.

Date of admission, November 21st, 1904. Date of discharge, February 7th, 1905.

The chief point of interest in this case is the high proportion of myelocytes, nearly 20 per cent. Myelocytes have often been observed before in this complaint, but 10 per cent. has been somewhat arbitrarily fixed as the

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limit. It is quite absurd to draw a line at 10 per cent., and say all cases above this are leukæmia and all below are not. The myelocytes were fairly equally divided between the large and small varieties.

Von Jaksch's anæmia must be rare in Australia, as this is only the second case I have seen in six years (I have not included the other case, as the differential count was not satisfactorily done). It seems to be common enough in the large cities of Europe; this is probably due to the infrequency of rickets with us, as this form of anæmia is almost invariably associated with rickets. This fact lends support to the view that the same causes which produce rickets also produce this disease.

A CASE OF SPLENO-MEDULLARY LEUCOCYTHÆMIA TREATED WITH X-RAYS.

By F. J. Clendinnen, M.D., L.R.C.P., &c.

Frederick P., married, aged 38 years, was admitted on 5th of November, 1904, to the Melbourne Hospital, under Dr. Maudesley.

His history previous to admission was briefly as follows:—Suffered from bronchial asthma during 1891, in Southern Queensland; disappeared when living in Thursday Island; suffered from dengue fever during epidemic of 1896 and 1897, in Thursday Island. Contracted enteric fever in 1900, in South Africa. About May, 1902, three months after arriving in Melbourne, became pale with a worn appearance, but otherwise felt in fair health; gradually became more pallid until March, 1903, when he became very weak, legs and ankles swelled at night, suffered from occasional pains at the left side about the stomach, and a feeling of fullness after meals. Sought medical advice and was prescribed arsenic and iron, which he continued for some time without much benefit. Took six weeks' rest from work; took sea trip to Brisbane, returned to Melbourne improved, less pallor; resumed work. Continued to improve until October, 1904, when he noticed his abdomen increasing in size: sought medical advice; was tapped, and large quantity of fluid removed. Had no hæmorrhage of any kind. After admission the fluid accumulated again. He was sent to me on December 1st, 1904, as a case of spleno-medullary leukæmia, to have X-ray treatment *a la* Senn.

On examination, patient looks pale, thin, and weak—distended abdomen; margin of spleen could not be made out, owing to the ascites present. After about a month's treatment the edge of the spleen could be felt about an inch to the right of the umbilicus, and lower edge about one and a half above the left iliac spine. Ten minutes' exposure over the splenic area with a medium tube, distant about 6 inches, with two and a half to three amps in the primary, were given three times a week. After about ten applications he was given a rest, owing to slight dematitis setting in amounting to mere bronzing and exfoliation of the epidermis. The ends of the long bones were also submitted to ten minutes' exposure, chiefly over both knees.

During the time he was under treatment with the X-rays, I stopped all internal medication. His temperature fluctuated between 98 and 100.6.

He was discharged from the hospital on December 9th, 1904, and attended as out-patient; for about the last three months he has only been attending now and again.

Examination of Blood.—Blood examined in Brisbane, May, 1903—actual figures not known to patient: approximately, 3,000,000 red, 200,000 whites.

Melbourne Hospital—November, 1904—Erythrocytes, 4,600,000; leucocytes, 190,000; hæmoglobin, 70 per cent.; characteristic leukæmic film.

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December, 1904—Erythrocytes, 4,300,000; leucocytes, 156,000; hæmoglobin, 75 per cent. February, 1905—Erythrocytes, 4,360,000; whites, 129,000. March, 1905—Whites, 132,000. April, 1905—Whites, 125,000. May, 1905—Not known. June, 1905—1,000,000 (?). July, 1905—Whites, 300,000. August, 1905—Red, 2,796,000; whites, 264,800; hæmoglobin, 60 per cent; large number myelocytes, also nucleated red.

Frederick P. started X-ray treatment on November 30th, 1904. December twelve sittings, consisting of ten minutes over splenic area and ten minutes over the terminal ends of the long bones. January had ten sittings likewise. February twelve ditto. March nine, April eight, May thirteen, June ten, July ten. Temperature never been up to 100 since 23rd of April, 1905.

DISCUSSION.

Dr. SPRINGTHORPE referred briefly to the light thrown upon the use of radium by the investigations upon cancer, which had been so well brought under their notice by the President of the Section on Pathology. It was generally recognised that from the material side life came in as a new kind of movement in molecules of a certain complexity. This was seen in the mitoses which accompany cell proliferation. In cancer it now seemed as if a second conception, so to speak, was started in the same cell—a sort of molecular twins—in which the second offspring grew asymmetrically and dominated the situation. This seemed to put ordinary germs out of the question, but left it still unsettled why this second birth had invaded the cell. But, in radio-active and similar emanations we had molecular movements of the same fundamental simplicity well calculated to influence these vital vibrations, and possibly presenting the key to a wise interference in abnormal conditions. The therapeutic application of X-rays and of concentrated solar and electric light might have a similar basis.

PHLEBITIS—INTERNAL JUGULAR.

By F. J. CLENDINNEN, M.D., L.R.C.P., Melbourne.

On December 18th, 1904, I was summoned to attend Mrs. B.; *æt.* 36, married, two children. I found the patient was about four months advanced in pregnancy. She was complaining of great pain in the left leg. On examination I found the leg somewhat swollen, pale, slight œdema, and in the popliteal space there was found a small lump about the size of an almond. She was kept in bed, and leg wrapped in wool, and flannel bandages applied. Apex of heart in normal position; systolic mitral bruit, second aortic sound slightly accentuated.

Previous History.—When a child, had measles. At the age of 19 she suffered from amenorrhœa and anemia, due, as she states, to shock after her sister's death. At the age of 21 was laid up for four months with dropsy of legs. No history of fluid in chest or pericarditis.

Family History.—Father and mother dead. One brother died of diphtheria, and another of stoppage of the bowels; one sister died of consumption, and the other of diphtheria.

Eight years ago I attended her with her first child. She then had about 30 per cent. albumen in urine, and I expected all manner of complications; but she had quite a normal confinement, and made a good recovery without slightest hitch. The second confinement, two and a half years ago, was also normal; still, the urine contained a large quantity of albumen.

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On Friday, March 31st, I was summoned about 6 p.m., and found patient suffering from hæmorrhage, which started at 4 p.m. Os was small; plugged vagina and waited awhile. Returned in the evening, removed plugs, douched, and replaced more plugs. About midnight found things not improving: decided to dilate and deliver—fully made up my mind that it was a case of *placenta prævia*. Chloroform was administered, and I proceeded to dilate by digital method. Inserted my hand after dilating up to four fingers, ruptured the membranes and turned: delivered female fœtus, stillborn. Expressed the placenta. No further hæmorrhage: pulse quick.

April 1st and 2nd.—Temperature normal; pulse 88.

Monday, 3rd.—Patient complained of pain in the head, very like neuralgia. Pulse, 76; temperature, 99°; urine, 40 ounces.

Tuesday, 4th.—Pain still in head, with slight throbbing; everything else normal. Ordered phenacetin and caffein cit.

Wednesday, 5th.—Patient complaining of pain in right side of the neck, with a stiff feeling; intense pain on moving the head; the throbbing in head worse.

Thursday, 6th.—Examined neck, and found a small lump, tender, above sternal end of clavicle, over the internal jugular vein; pulsation above this and up side of neck.

Thursday, 6th.—Throbbing in head very much worse, with paroxysms of pain, which started at the lump on the right side of the neck, gradually going through the head with great throbbing and a bursting sensation; gradually dying away, leaving a burning sensation down the spine. She was unable to sleep, owing to the intense pain and throbbing in the head.

Friday, 7th.—All day the pain was very bad; the attacks much more frequent. Had consultation with Dr. Stawell, and we agreed to give her one-sixth of a grain of morphia and one minim trinitrin, two-hourly for a few doses, afterwards four-hourly.

Saturday, 8th.—Slept about three hours after morphia; pain and throbbing slightly better. Had another bad attack of pain in evening.

Sunday, 9th.—Pain and throbbing again troublesome; not able to get any sleep. From midnight very restless.

Monday, 10th, at 8.30 a.m.—Patient collapsed, got very cold and clammy, almost pulseless. After treatment revived, and went on until 9 p.m.: collapsed again; pulse, 140. Revived again under stimulating treatment. Ordered her strychnine, digitalis, and sp. am. aromatic and bromide, to be injected per rectum.

Tuesday, 11th.—Had three or four hours' sleep after the bromide. Pulse varied, 90-100; temperature, 99°.6. Took plenty of milk food; bowels freely moved; urine, 30 to 40 ounces in twenty-four hours. Slowly but steadily improved from that on. Urine specific gravity, 1020; albumen, 30 per cent. Few hyaline and granular cells; no blood casts, epithelial, or fatty casts; bladder epithelial—characteristic of large pale kidney. Blood pressure, 166.

I thought this case sufficiently interesting to bring before the members, as I have been unable to find a similar case yet published. This patient had no eclampsic attacks during any of her confinements. I may state that this is the third case I have had of patients suffering from albuminuria who have suffered during the middle months of pregnancy from phlebitis, and all in the left leg. It can be readily understood why they should occur in the popliteal, probably due to traumatic origin, injury to the intima of the vessel—similar to popliteal aneurisms. But does the toxæmia, not puerpural but urœmic, play an important part in coagulation of the blood in these cases of albuminuria?

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THE PRINCIPLES OF CHEMISTRY AND THE STUDY OF MEDICINE.

By C. Reissmann, M.A., M.D., Camb., B.Sc., M.R.C.P., Lond.

Motto—"Ein arzt, der nur die Medizin kennt,
Kennt nicht einmal die Medizin."
—Aphorism of a Spanish Teacher.

Theme.—In a very great measure the advance in our knowledge of the science of medicine is the direct result of chemical research. There are many problems in medicine—problems often of the first importance—which cannot be studied nor understood without a general acquaintance with the principles of chemistry in its several branches.

Recent research in chemistry so frequently, and often so unexpectedly, has an important influence on the progress of medicine that many of us medical men feel that we must be able to keep in touch with its present developments; to watch this sister science, and ever to be ready to avail ourselves of such of her discoveries as may be useful to us in the diagnosis and treatment of disease.

To be able to do this we must possess some foundation of chemical education. This does not mean that we must make an exhaustive study of chemistry, nor that we must study chemistry to the exclusion of other branches of science: we need not even burden our minds with many of the facts now acquired by medical students. For instance, a knowledge of the physical properties of chemical substances, though valuable, is of relatively minor importance. At most we can only hope to know but a few of them—for of the compounds of carbon alone more than one hundred thousand have been described. It is, however, of the very first importance to be familiar and thoroughly conversant with the principles of chemistry—descriptive, analytical, physical, and physiological, which is the alphabet and grammar of a science in which we live.

Think only of the new synthetic preparations which are brought to our notice by the enterprising commercial traveller, or by conspicuous advertisements. We are baffled by their number and the complexity of their formulæ. To understand the chemistry of them requires an amount of technical knowledge which few of us possess. We are then tempted, either to condemn these preparations without consideration, or else to accept wholly all the vendor may paint in his attractive advertisement. In most cases it requires no specialised technical knowledge, but only a good general acquaintance with the principles of chemistry, in order to recognise whether or not the claims made in favor of a drug rest upon the safe foundation of accurate reasoning from observed facts. Thus, if someone should assert that an emulsion of petroleum is a wholesome food for consumptives we should be ready with the reply that petroleum is a hydrocarbon, and hydrocarbons are not food stuffs. If we are told that petroleum emulsion is an efficient germicide, we should ask for the evidence of a competent bacteriologist.

I suppose most of us have to make a chemical examination of urine every day of our lives, but do we always succeed in detecting all the most important pathological ingredients? Why does myelopathic albumosuria count among the rarest of diseases? I suspect that it would not be so extremely rare if it were not that sometimes when examining urine we have not observed what we have seen, or else we have not known what we have observed.

For the proper treatment of a disease such as diabetes, where there is a want of assimilation of an important food, a knowledge of chemistry is all-important. In a patient suffering from diabetes the power to assimilate

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dextrose is impaired, or lost, but most patients can nevertheless assimilate a certain amount of lævulose. The lævulose is stored in the liver as glycogen, and it seems probable that it can completely replace dextrose in the diet: therefore it is sometimes prescribed. But, as a rule, diabetic patients do not complain because they are forbidden to take sugar, for they can still sweeten their foods with saccharine or saxin: they do complain, and sometimes very bitterly, because they may take neither bread nor any form of starchy food. Starches are not allowed because they are converted into dextrose, which to them is useless as food, and may even, as some think, act as a poison.

Now if there should be a starch which, on digestion, yields lævulose and nothing else, is there any reason why this starch should not be given? Such a starch, indeed, exists; it is found abundantly in the pure state in dahlia bulbs, and it is known as inulin. The simplest way to prepare inulin is to mince dahlia bulbs, digest them with a small quantity of water, and strain. The water contains nearly the whole of the inulin, a large quantity of which will settle to the bottom of the vessel in twenty-four hours. It may be dried to a white powder. A much larger yield is obtained if the inulin be precipitated with alcohol and then collected, for it is somewhat freely soluble in water.

I have had no difficulty in preparing a large quantity of this starch. It has a somewhat sweet taste, but is otherwise tasteless. If biscuits could be made containing inulin I believe they would be a valuable food for some diabetic patients. The attempts I have myself made in this direction have not been very successful.

I have selected diabetes as an example, but in every disease in which there is faulty digestion or mal-assimilation of food, a knowledge of chemistry is useful, if not indispensable.

Histology.—Consider now Histology and the use of staining reagents. At one time these were used in a haphazard fashion. Does not this remind one of the epoch of empirical methods in chemistry? This period of physiological alchemy is only now passing away. To-day we regard stains as chemical reagents. When we stain an animal tissue with a dye we perform a chemical reaction very much in the same way as a chemist tests for an inorganic base in a test tube. As soon as this principle was properly grasped a great advance was made in physiological and pathological histology. By such chemical means is the intricacy of the central nervous system being unravelled at the present time.

The method of Golgi—a method which Sir Victor Horsley says is revolutionising all our definite knowledge of nerve fibres—what is this method but a chemical reaction?

The staining methods of Marchi or Ramon-y-Cajal, of Nissl, and of Weigert are definite chemical reactions, and until we recognise this all search for further improvements is mere groping in the dark.

Weigert.—The first to demonstrate bacteria by means of chemical dyes was Weigert. It was he who introduced the use of aniline colors in microscopical technique. Weigert's first attempts were empirical, but later they followed a logical sequence.

Most of the methods now used to demonstrate nervous structures are founded on Weigert's researches. These researches led Weigert to the discovery, which I will shortly relate, that the successful staining of nervous tissue depends on the recognition of a definite chemical principle.

It had long been known that nervous tissue has a special affinity for many highly oxidised metallic compounds—as osmic acid and chromic acid. The search for a special method of staining medullated nerves led to important results, not the least of which was the discovery of the tubercle

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bacillus: for, in his first communication, Koch demonstrated the tubercle bacillus in sections stained with alkaline methylene blue and counter-stained with Bismarck brown. But this was the method which Weigert was using to penetrate the structure of the central nervous system. Here it failed. He therefore tried methyl violet, and again counter-stained with Bismarck brown, and was now rewarded by seeing the network of Gerlach in the grey substance of the cord, a structure hitherto only demonstrated by gold preparations. For several months he continued his experiments without success, until at length he tried an aqueous solution of acid fuchsine—a dye not hitherto used in histology. Sections of the cord were stained dark red. To remove the excess of stain he used an alkaline fluid, and he chose alkaline alcohol as the sections must subsequently be hardened in alcohol. To his astonishment they were completely decolorised. Thinking them destroyed, he threw them into a dish of water, when, to his joy, they again became red, and now showed the same differentiation of structure as sections stained with methyl violet and Bismarck brown. Gerlach's network was demonstrated, and the white substance appeared dark, the grey substance light.

But the method, successful as it was for sections of medulla and cord, failed to show the nerve fibres in the brain.

Meanwhile, Paul Ehrlich had improved the method of staining tubercle bacilli by using a strong mineral acid in place of Bismarck brown. Weigert adopted the same principle to stain brain sections: he used acid fuchsine and washed with dilute hydrochloric acid, and succeeded in demonstrating the nerve cells and their nuclei. But he still failed to demonstrate the fine nerve fibres of the cerebrum. Ultimately he saw that success was only achieved in sections hardened in chromic acid. This acid acted as a mordant, and subsequent staining depended upon a chemical reaction between the dye and a compound formed by the mordant with some constituent of the nerve structure. Various metallic salts served as mordants—salts of lead, tin, iron, and vanadium; but copper salts gave the best practical results, and hæmatoxylin proved the most successful dye. Whereas, in the method of Golgi, there is an element of chance whether or not a given specimen will be successfully stained, Weigert's method has this to commend it: that every properly prepared specimen will show both the coarse as well as the fine nerve fibres. The bichromate of potash, in acting upon the medullary substance, is partially reduced to an oxide of chromium; it is in this way that the brown coloration is produced. The reduction is stronger in the medullary substance than in the other constituents of nervous tissue: thus the ganglion cells and neuroglia reduces chromium salts in a lesser degree. This produces the difference in color between white and grey matter, and in normal and degenerated parts of the nervous system.

Thus three years' labor resulted in the discovery that a chemical principle must be recognised for the successful demonstration of the fine nerve fibres in the central nervous system, and subsequent investigations in this branch of anatomy have all followed this principle which was discovered by the labors of Weigert.

A careful investigation of Marchi's reaction for the demonstration of tracts of nerve degeneration has resulted in the discovery of a chemical test by which a severe and active organic nervous disorder can be distinguished from a functional nervous disorder: for in the former cholin, a substance which can be readily detected, appears in the blood, while it is absent in the latter. My friend Otto Grünbaum has applied this test. He found that in normal blood cholin is absent, or present only in negligible quantities. In four cases of hysteria, and one case of tobacco-poisoning, the result was negative. He obtained positive results in three cases of disseminated sclerosis,

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in two of paralysis agitans, in three of tabes dorsalis, in one of progressive muscular atrophy, and in two of transverse myelitis. This method was introduced and has been successfully employed by Professor Halliburton and Dr. Mott. A full account of it is given in Professor Halliburton's book on " Bio-chemistry of Muscle and Nerve."

The detection of iron in the liver by a simple mirco-chemical re-action is the chief argument upon which Dr. Hunter based a theory of the nature and cause of pernicious anæmia.

Ammonium molybdate, followed by pyrogallol, is a chemical test by which a phosphorus-containing substance can be detected. It has proved to us that phosphorus compounds are confined almost exclusively to the nucleus of a cell.

The chemistry of micro-chemical reactions is a branch of chemical science which promises us a wealth of new facts. This is not the only department of chemical science from which our own science derives advantages.

Low Temperature.—Experiments at low temperatures have been carried out by Cailletet, Olzewski, and Dewar, and a temperature of 257° has been registered. Availing himself of these researches Macfayden has prepared a vaccine against typhoid fever, by crushing typhoid bacilli frozen with liquid air.

Velocity of Chemical Action.—The rapidity with which a chemical re-action is completed depends upon the temperature. A reaction which at ordinary temperatures is accompanied by the evolution of heat and light may, at a very low temperature, be completely arrested. As an illustration I may remind you that, at the temperature of liquid hydrogen, fluorine gas is without action on scilicon, boron, carbon, or mercury, though at ordinary temperatures it combines with each of these with the liberation of much energy. This is an extreme example of the general principle that the reaction velocity of chemical agents is increased by a rise of temperature. The connection between such a purely chemical principle and the practice of medicine may appear to be remote, but it finds an application in the dosage of medicine; for by it we know that chemical reactions will occur more rapidly in a feverish patient than in a normal individual, and, therefore, *cæteris paribus*, the dose of a drug prescribed for the former should be smaller than for the latter.

Do we always remember that the chemical action which takes place when we use antiseptic solutions for the destruction of septic germs requires a certain time for its completion? A septic instrument is not rendered sterile by dropping it for a moment in a one in twenty solution of carbolic acid; much less if that solution is warmed by the addition of a further quantity of hot water.

Mass Action.—The department of chemistry which has thrown most light in some of the darker alleys and by-lanes of medicine is that which itself has been most illumined in recent years, namely, chemical dynamics, or the theory of the progress of chemical reactions and the theory of chemical equilibrium. These two phenomena—the velocity of chemical reaction and chemical equilibrium—are explained by the law of " mass action." Every substance, says Berthollet, which enters into chemical combination reacts by reason of its affinity and of its mass. It will be acknowledged, I think, that this is one of the most important natural laws which has been enunciated within recent times.

If equivalent masses of a weak organic acid like acetic acid, and of a strong mineral acid like hydrochloric acid, are allowed to react on an equivalent quantity of a base, such as soda in aqueous solution, it is found that the acetic acid takes one thirty-fourth of the soda and the hydrochloric acid takes the remaining thirty-three thirty-fourths; but if we use more than one

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equivalent of acetic acid, this acid, though relatively weak, fixes more of the soda, and less soda is taken by the hydrochloric acid; and the greater the amount of acetic acid present the greater is the amount of soda it will fix and the amount of hydrochloric acid which will remain uncombined. The weak acid displaces the stronger because it is more abundantly present.

Put into chemical language, one says, chemical action is proportional to the active mass of the reacting substances. The active mass of a substance is the amount of the same in unit volume.

The law of mass action explains the secretion of hydrochloric acid from alkaline blood. By reason of its mass the carbonic acid in the blood sets free a small quantity of hydrochloric acid from the sodium chloride, which is also present in the blood. This minute quantity of uncombined hydrochloric acid is selected by the oxyntic cells in the cardiac portion of the stomach, and the carbonic acid by its mass influence sets free another small quantity of hydrochloric acid. This is again seized by the oxyntic cells, which in time are able to abstract a considerable amount of acid and deliver it into the stomach.

In the study of medicine I suppose there is no subject of greater complexity than the theory of immunity. If one considers Ehrlich's theory of immunity with his conception of receptors and amboceptors of immune body and complement of toxins and toxones, one is struck with admiration at his extreme ingenuity; but one dearly longs for simpler exposition. Now Masden and Arrhenius have advanced a perfectly simple explanation of immunity, by applying to the ascertained phenomena the law of mass action. They assert that toxins and antitoxins react by reason of their affinities and of their mass, and the reaction toxins plus antitoxin equals a neutral body [toxin-antitoxin] is analagous to the inter-action of a weak base, as ammonia with an acid of weak avidity, as boric acid.

Electrolytic Dissociation.—The theory of electrolytic dissociation—that in dilute solution inorganic compounds dissociate into their respective ions, which carry electrical charges of opposite sign—frequently has a practical application in the practice of medicine.

The disinfectant property of mercury salts depends on the mercury ion, and not on the inorganic ion with which the mercury is combined. In a given solution of mercuric chloride the disinfectant action is proportional to its degree of dissociation, that is, to the number of mercury ions present.

But the degree of dissociation of a dissolved electrolyte is diminished when an electrolyte containing a common ion is added to its solution.

Therefore, the germicidal action of mercuric chloride is lessened by the addition of sodium chloride or hydrochloric acid to its solution.

Pasteur.—About this time five years ago there passed away one of the greatest chemists the world has known. The grandest illustration of the influence of the principles of chemistry on the study of medicine is to be found in the life of the chemist Pasteur; and we now remember with sorrow that at every incursion he made on the domain of medicine he was looked upon as one who was poaching on the preserves of others. But he was a chemist to whom the greatest of living surgeons, Lord Lister, wrote as follows:—"Allow me to take this opportunity to tender you my most cordial thanks for having, by your brilliant researches, demonstrated to me the truth of the germ theory of putrefaction, and thus furnish me with the principle upon which the antiseptic system can be carried out."

Studies in crystallography taught Pasteur the use of the microscope. The microscope furnished him with the means of penetrating the mystery of fermentation. An investigation of fermentation led him to see the error of the theory of spontaneous generation, and to the discovery of micro-organisms

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as the cause of disease. From this it was but a step for him to preventive inoculation, the cure of anthrax and hydrophobia, and of many other diseases.

I have given you a few instances in support of my assertion that the principles of chemistry—by which I mean the fundamental laws and theories of chemistry—should be mastered by every student of medicine, because he will have frequent opportunities to apply them in his practice. They are almost as useful to him as are pure mathematics to the student of engineering.

Gentlemen, this theme, if time and a fuller knowledge of the subject on my part would permit it, might be elaborated in an extensive treatise. The great influence of the principles of chemistry in the study of medicine is one of the chief practical results which we owe to this age, which has been called the age of natural science.

A physician may no longer be a specialist in one branch of natural knowledge called medicine. I believe that, with fuller knowledge and wider experience, we shall discover that medicine, like chemistry, is but a branch of the great tree of natural science whose stem is physics.

It is often said that an educated man is one who knows something of everything and everything of something. An educated physician should likewise know everything of medicine and something of every other science.

It is an historical fact that great and rapid progress has been made since we began to study the phenomena of physiology and of pathology by the methods of physics and chemistry, and abandoned those void and abstract speculations that were used and misused by the philosophers of the early part of the last century.

Chemistry, like physics, is one of the preliminary subjects of study prescribed in the curriculum of medical education. Some think little of it, and find no more of interest or of value in it than the man who, lacking a sense of harmony, finds in the rehearsal of a great opera.

I have deemed this subject of sufficient importance to bring before this Congress, because most of you are closely associated with medical students, who look to you for guidance and advice; and I ask, if you agree with what I have said, that you will convince them that if in the study of medicine they would be masters they would do well first to master the principles of chemistry.

ERYTHEMA INDURATUM, OR BAZIN'S MALADY

(*Erytheme Indure Scrofuleux*).

By F. J. Clendinnen, M.D., L.R.C.P.,

Mrs. Q., *æt.* 32, married; three children, the last two years ago—all healthy. *Family History.*—All healthy.

Previous to marriage was engaged at business which necessitated standing greater part of the day. Since marriage, always ailing; never has good appetite. Bowels regular. Suffers from cold hands and feet; no chilblains. Lived in Adelaide, but for ten years in Queensland. Whilst in Queensland was treated with specific treatment. In Adelaide was treated with rest and local treatment.

When I saw the patient, who looked anæmic, she was unable to get about, as her legs were in a painful condition with numbers of sloughing ulcers. This condition started two years last June, as red nodules with a broad base. This gradually darkened in color, till it attained a purplish hue. The outer margin of the nodule softened until it eventually broke down and caused a circular

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slough, leaving a patch of necrosed tissue in the centre which eventually sloughed off and left ugly indolent ulcers—rather suggestive of gummata. It started in winter (June): no febrile symptoms. The nodules were at first very painful till they discharged, when they became more bearable. The early stage resembles erythema nodusum were it not for the circular hard lump in the centre.

All the authorities agree that complete rest is necessary, and that local treatment should be employed, and the patient's strength supported. I decided to try the ultra-violet rays. I told her to come to me, and she replied "that she could not walk." However, she attended, conveyed to my house in a cab for the first week; afterwards walked to station, and came by train. They all healed up in course of about five weeks, with the exception of a fresh nodule appearing in another part of the leg. She was unable to attend for further treatment owing to approach of her confinement.

The treatment with ultra-violet rays was applied daily for from five to ten minutes during first week; afterwards three times a week. No internal treatment was administered; the only local treatment consisted of boric foments—together with the ultra-violet rays. All the ulcers treated were healed within six weeks. Several indolent ulcers of long standing have been healed up by this method, which consists of alternating current transformed up to about 6,000 volts to an iron electrode lamp, with a condenser in parallel.

I carry out a method entirely my own, in which I use no compression with rock crystal, but, as I term it, an open method. I have adopted this plan simply from experiments I have made in electrical discharges through the air. I have also adopted another mode by painting a celluloid film with a fluorescent substance; placing this over the part and applying the rays, the fluorescence remains for one hour or more, according to the charge.

BACTERIOLOGICAL EXAMINATION OF CASE.

By Dr. Huckell.

A cover slip preparation of the pus stained with eosin and Loeffler's blue showed the usual number of broken down white corpuscles, and cells, and crowds of cocci. There were also present bodies resembling the stellate arrangement of clubs which is found to obtain in actinomycosis. These took up the basic stain, but not very strongly. These appeared to be of the nature either of one of the mould or else one of the pleomorphous bacteria of the streptothrix order.

Agar and blood serum cultures were then made of the pus: a great number of colonies appeared. A pretty exhaustive examination of these, however, revealed only the well-known staphylococci, both the albus and aureus. The presence of the streptothrix could not be confirmed by culture, but it may be that the proper culture medium was unfortunately not hit upon. We know that the actinomycosis presents only a mycelial arrangement and gonidia-like bodies in artificial culture, and not the more familiar stellate growth, and we might therefore expect to get a different appearance in this hypothetical organism, but on culture results were apparently negative—that is,so far as an hitherto undescribed bacterium is concerned.

Differential Diagnosis.—On first sight one would say that the ulcers were of specific origin, but this may be eliminated, seeing there is no specific history; moreover, by its not responding to specific treatment.

From erythema nodusum, its duration, etiology, they suppurate; no history of rheumatism, no joint affections; it is worse in winter; no endocarditis.

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THE MEDICAL TREATMENT OF APPENDICITIS.

By F. Lucas Benham, M.D., M.R.C.P., Lond.

It is with much reluctance and hesitation that I venture to speak on this subject. It has been so much discussed of late years that very little that is fresh remains to be said. The question seems, however, not to be exhausted yet, judging from the contributions which still help to fill the medical press. Nearly all such recent contributions are, however, written from a surgical point of view, and assume that the surgical treatment of the disease has largely superseded the medical: at any rate, I think that this tendency is getting a stronger hold. For my own part, I think that this surgical aspect has been carried to excess; and I propose to offer a few remarks which may help in judging what purely medical treatment can accomplish; and whether it is best to rely on such treatment, or to hand over cases to the surgeon at an early stage; and whether surgical operation is required at all in any considerable proportion of instances. Just like those variable stars which suddenly start from insignificance and invisibility to a position of the first magnitude, so has the disease in question come into prominence of late years. It has been asserted that it is a new disease: that it is a disease that has increased in frequency and in severity: and that surgical operation gives better and more sure results than medical treatment. That it is a new disease is quite an untenable proposition. Its identity as a disease arising from lesion of the vermiform appendix has been recognised at least as long ago as 1836, and no doubt its existence could be traced much farther back. In 1836 it was distinctly described by Addison (Bright and Addison's "Practice of Medicine"). Burne (1) also described it in 1837. The doctrine was maintained principally by the Guy's school and was upheld by Wilks and Moxon ("Pathological Anatomy"). Dr. Crisp (2) published a series of thirty-two fatal cases in 1856, and eight more were reported to the Pathological Society between that time and 1870. Lewis, of New York, also published a collection of forty-seven fatal cases in 1852. In 1871, Bristowe (3) wrote a very lucid account of the nature of the disease. He "defined cases of typhlitis and perityphlitis as those in which there is perforative ulceration of the cæcum or vermiform appendix, and in which, therefore, there is either limited suppuration in the neighborhood of those parts, or sudden peritonitis. Perforation, in the great majority of cases, no doubt occurs in the vermiform appendix. It is not improbable that in some cases the perityphlitic abscesses are peritoneal abscesses." Still, for certain reasons—the small fatality and few *post-mortem* examinations—the correct pathology was not generally known or accepted. The cæcum and cellular tissue were generally supposed to be the seat of perityphlitis (*vide* Niemeyer), and an intraperitoneal abscess was hardly thought of. Many cases, too, were imperfectly diagnosticated, and were looked on as "gastric fever," "inflammation of the bowels," "peritonitis," and "intestinal obstruction." Probably many fatal cases, in which the characteristic symptoms of typhlitis were wanting, were so classed. Dr. K. Fowler said, at the Clinical Society in 1901, that eighty-five cases of typhlitis or perityphlitis were admitted to the Middlesex Hospital in ten years; all recovered, and in only two cases were incisions made. But during the same time there were fourteen necropsies with perforated appendix, but the diagnosis had been peritonitis, intestinal obstruction, enteric fever, &c., and in none of these were there the ordinary symptoms of typhlitis.

Nor is it probably any more true that the disease, although a familiar one already, has appeared in greater frequency. At first sight, there seems

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a good deal to be said for the hypothesis; it seems almost self-evident. From the large number of cases reported, as well as the much larger number admitted to hospitals, compared with those of former times, and especially the fatal cases, of which we so seldom heard before, and even from the cases within our own experience, which assumed a novel and more serious character when our attention was drawn thereto, it is very difficult to avoid the belief that the disease is present in a new and more serious phase.

Nevertheless, I cannot help thinking that the supposed increase, both in frequency and severity, is a myth—a pure illusion. It is true that opinions are divided. In support of a supposed increase we have the weighty authorities of Sir W. Gairdner and Dr. G. S. Keith. The former (4) says that he saw his first fatal case in 1884; only four others were found in the records of the Edinburgh Infirmary for ten years: he affirms that the share of the appendix had not been overlooked, but the mortality was probably not more than 5 per cent. The latter (5) said that there was nothing more remarkable in the medical world than the extraordinary increase in cases of appendicitis. It was almost unknown, he said, in the middle of the last century. During forty years' practice in Edinburgh, among all classes, he did not remember a single case, and had heard of none for the last twenty-five years among his old patients. He ascribes the supposed increase to full feeding, especially of butchers' meat. Many others too, have written in support of the same view, and have gone the length of assigning a cause—the use of enamelled cooking-pots, frozen meat, vulcanised indiarubber of bottle-stoppers, &c., &c.

On the other hand, Messrs. Treves, Mayo Robson, Dr. West, and very many others doubt or deny the increase. The *Lancet* itself says (6), "There is, in fact, but little if any reason for thinking that a real increase has occurred in the number of cases of inflammation of the vermiform appendix. The apparent increase is probably due to transference of many cases to the surgeon and exploratory laparotomy for obscure diseases." And, again (7), "Improved diagnosis will probably account for some of the apparent increase of cases." Thus it seems to me that at any rate much stronger proof of the alleged increase is required to be convincing The greater number of cases sent in nowadays to hospitals (where most statistics are compiled), with a view to operation, accounts in great measure for the apparent increase. For instance, at St. Bartholomew's Hospital in 1882-4, 111 cases were admitted, with seventeen deaths; in 1899-1901, 301 cases were admitted, with thirty-eight deaths; and of these cases 217 were operated on. In the Birmingham Hospital the figures correspond. In the Zürich Hospital also, during the twenty years 1880-1900, forty-five cases were admitted during the first fifteen years, 158 cases in the last five years. Moreover, this supposed increased prevalence extends all over the civilised world, for all countries tell the same story.

The personal factor has a good deal to do, I believe, in forming conclusions on the point. As an illustration of this, I think I may instance a statement of Sir F. Treves (8), that the disease is especially prevalent in tropical and sub-tropical climates; because a large proportion of cases in which he had removed the vermiform appendix for recurrent attacks came therefrom. Is it not far more likely that persons suffering from the complaint and living in a distant British provinces resorted to him in particular because of his wide reputation?

Though I cannot admit any proof of the increased frequency or severity of the disease, I can see no *a priori* reasons why it should not be so. We may compare cancer and insanity, whose absolute increase is still discussed and unsettled. Pancreatitis is a new disease in the sense that it has only lately been identified; yet no one would deny that it has always existed,

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and as frequently in former times as now. But diphtheria has almost certainly increased; and many other acute specific diseases, dependent on the activity of microbes, vary in prevalence; they have their periods of waxing and waning—a phenomenon noted by Sydenham, and ascribed by him to the epidemic constitution of the year. What is influenza, in all probability, but an extra virulence of the organism which causes common colds? But most of these diseases are contagious, which appendicitis probably is not; nor has the latter been proved to be essentially a microbic disease. It is held by some that influenza has been a powerful predisposing cause of appendicitis; and there might be a good deal to be said for this view, only the supposed alarming increase preceded the outbreak of influenza. The only points about the disease which are really new are—1. The name "Appendicitis," which, by the way, is a barbarism; it was objected to as long ago as 1859 by Mr. Jas. Long, of Liverpool, who rightly preferred "Typhlitis." 2. A far more exact pathology, due to the more frequent explorations in abdominal surgery and the more numerous necropsies in hospitals; and especially to the knowledge of the correct pathology having become diffused and general, and having led to an improved diagnosis. The statistics of the German army show this well. Cases of appendicitis have rapidily increased in number, but those attributed to disease of the stomach, liver, &c., have *pari passu* declined. 3. The advanced surgical treatment of the disease.

The modern serious attention to the matter began in the United States of America; and was, I believe, initiated by a paper by Fitz in 1886, who had collected reports of a number of fatal cases.

With the rise and spread of abdominal surgery, the peritoneum having yielded its secrets to adepts, the surgeons, always anxious to enlarge the sphere of their operations, speedily took the matter up, and went rapidly ahead in America, where they made a practical application of the new pathology in their own way. The wave reached England. Cases of appendicitis had been operated on (*i.e.*, more than merely opening superficial abscesses) by Messrs. Symonds and Godlee (a case apiece) in 1885 (9), and the appendix had been removed many a time in the course of abdominal operations by Spencer Wells and Knowsley Thornton. Public attention was, however, first drawn to the question in London by a paper by Mr. (now Sir F.) Treves on "Relapsing Typhlitis treated by Operation," read before the Medico-Chirurgical Society (10), in which he recommended removal of the appendix in a quiescent interval after repeated attacks.

The application of the appendix theory was soon extended by surgeons to the active state of the disease. A discussion was opened at the Medical Society of London by Dr. Bull, of New York (11), who advocated early operation. Further discussions took place at the annual meeting of the British Medical Association at Leeds in 1889, and at the Clinical Society of London in 1891; and the medical journals have been full of the subject—both facts and opinions—ever since; and meanwhile attempts have been made, with considerable success, to withdraw the disease almost entirely from the domain of medicine to that of surgery. I was, I think, present myself at the discussions at the Medical and Clinical Societies in London, and have since followed the subject in English periodical literature, with curiosity and interest.

In former times surgeons used to operate only when the abscess reached the surface, or was extending dangerously; next, they advised operation as soon as suppuration had taken place, or was suspected to have done so; but, finding this not very successful, the mortality being quite 20 per cent., they are now getting to recommend the American plan of operating, not

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merely if there is no improvement within thirty-six hours, but immediately the diagnosis is made; besides immediate laparotomy in cases of general peritonitis, and removal of the appendix after convalescence, if that has not been done already.

Now, my contention is that most of this advanced surgical treatment is uncalled for and even harmful. It is supposed to be based on the improved pathology, but I doubt the justice of the application. The acquired knowledge should be used, not abused. I am reminded of a certain old lithotomist, mentioned by Sir H. Thompson in his lectures, who was accustomed to be wonderfully successful in his operations until he made a special study of the anatomy of the perinæum. After this, in trying to do still better, steering by his anatomical chart, he only did worse than before. Thus, better knowledge does not necessarily involve better treatment; but the fault is in ourselves, and the lesson is how to use our new knowledge aright. It is admitted by most who have devoted attention to the subject that a very large proportion of cases will recover without surgical intervention. It is impossible to be exact, as few statistics of former years are to be had. Dr. W. Russell, in an interesting paper (12), gives a number of statistics from Germany and elsewhere, and calculates that 90 to 95 per cent. of the cases recover without operation; and this accords with the opinion of Treves and others. Of private cases, probably less than 5 per cent. are fatal: of hospital cases, not more than 10 or 15 per cent. If there has been any increased fatality in recent years, it is probably due to increased surgical activity. Numerous opinions have been expressed that medical treatment is sufficient and the best, for most cases at any rate.

Dr. Fagge says (13), "Even when typhlitis presents itself under the guise of intestinal obstruction or of diffused acute peritonitis, it seldom destroys life if judiciously treated."

Dr. Hare (14) said that he had never found any need for surgical interference in his own cases of typhlitis or perityphlitis. He advised the free use of leeches, which were too much neglected now.

Dr. Fowler (15) protested against the idea that most cases could not get well without surgery. He believed that all cases arose from the appendix. Medical treatment was best for early cases, except where general peritonitis was present.

The debate whether the disease should be treated principally medically or principally surgically is not conducted by the whole body of physicians ranged against the surgeons; for many physicians prefer to hand over their most serious cases for operation; and many surgeons are content that a large proportion of cases should be treated medically. Among the latter class of surgeons, Treves is one of the most moderate and consistent. He defines (16) the disease as a "localised peritonitis in the cæcal region." Though he advises surgical interference when suppuration is known or believed to exist, he seldom has recourse to the knife before the fifth day, *i.e.*, unless there are very urgent and emphatic symptoms. He says, "medical treatment is, on the whole, remarkably successful. Any proposal to treat all cases of typhlitis by operation would lead to disaster. In the London Hospital nearly all cases recovered under ordinary medical treatment (there were twenty-five fatal cases during the last few years). Possibly it was better to do too little than too much and to return to the period before removal of the appendix had set in." And again (17), "The very great majority of cases get well spontaneously. Operation during an acute attack is attended with great risk to life—20 per cent. or more. A fair-sized perforation or a fair amount of gangrene could exist without serious symptoms; and he had

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found a concretion lying outside the appendix one month after the attack without a single drop of pus, when all symptoms had subsided, though there must have been rupture, perforation, or gangrene."

Mr. Langton also (18) believes that physicians could cure most cases.

Mr. Ballance (19), however, takes the opposite view. He strongly advises operation in all stages—especially within the first forty-eight hours, before rupture or ulceration has occurred; but he admits that many cases run quite a mild course and get well without any surgical intervention at all. His argument is that medical treatment in any particular case is uncertain as to its result, while surgical procedure is certain; and he quotes Legueu—"The prospect of spontaneous recovery and of medical treatment is a decoy, to rely on which is to be led into a snare."

Dr. Bull, of New York, adopts as an axiom that the risk of operation and exploration is less than the risk of the continuance of the disease. He also remarked (20) that he doubted whether patients would ever consent to have the appendix removed in the quiescent period. His doubts have proved quite unjustified; for patients who have suffered at all from appendicitis are only too eager to undergo this internal circumcision, and to submit to an operation of any sort, especially since an illustrious personage suffered from this disease and was operated on, they think it must be the proper thing to do in every case.

The modern surgical treatment is based on the maxim that wherever there is pus it should be evacuated by incision; or else it should be prevented by removal of the focus of disease beforehand. Though this rule is generally true, there are exceptions of course, and this is one of them. We know now that nearly every case of appendicitis or perityphlitis arises from the vermiform appendix: that inflammation of this organ leads to ulceration and perhaps perforation, with inflammation of the adjacent peritoneum: the contents of the vermiform appendix may exude, or a portion may slough away. This process of ulceration and sloughing of course takes some little time; and, meanwhile, adhesions form between the appendix and neighboring parts—abdominal wall, omentum, intestines, &c.—so that when perforation occurs and the contents are discharged that part of the peritoneum is already pretty well shut off, and thus an intraperitoneal abscess cavity forms. Probably an abscess forms in very many cases; but, before it attains to a large size, it bursts into the cæcum, which acts as a natural and safe drain—quite different from the case of an empyema bursting into the lung—and then the abscess gradually closes. Treves says (21), "When a typhlitic abscess is evacuated, it is not uncommon to find that the abscess communicates by a ragged aperture with the cavity of the cæcum. In nearly every instance there is evidence to show that the abscess had commenced outside (in the appendix) and then burst into the caput coli." The exceptions where the abscess discharges through the skin or bursts into the peritoneum are very rare. Sometimes the pus will dry up and become encapsuled or absorbed. Probably this is the course of the 90 or 95 per cent. of cases of perityphlitis which are known to recover; and little attention was paid to the local symptoms when the inflammatory swelling was believed to be extra-peritoneal and non-suppurative. But, when it became known—just as M. Jourdain, in Moliere's play, came to know that he had been talking prose all his life without being aware of it—that the perityphlitic swelling was an intraperitoneal abscess, medical men became alarmed at even simple normal cases; and instead of leaving them alone, to follow their natural course, some of them seem to have lost their heads, distrusted the medical treatment that they had formerly employed with success, and began to deem surgical interference necessary; and the results have been perhaps less fortunate than hitherto.

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The vermiform appendix is certainly specially exposed to certain accidents, but it is also specially protected from the results of these mishaps. Its fixed position in a sheltered corner of the abdomen, its small calibre, and the absence of a free current of foreign contents, make its relations quite different from those of the stomach or intestines in the event of perforation. The chances are as much in favor of a localised inflammation and abscess resulting from a perforation of the appendix as they are against it in the case of perforation of the stomach or bowel. Further proof of the abscess discharging into the bowel may be sometimes had by observing pus in the stools: I have noticed it several times, and it should always be looked out for. There is often a remission of the symptoms about the same time. Sometimes, as the abscess enlarges, it may give way a little here and there in other directions than into the cæcum, and such occurrences may be indicated by more or less serious symptoms of sudden collapse. They are not necessarily grave, as fresh adhesions may form and again enclose the pus, as they usually do. Sometimes the pus finds its way between coils of bowels or into the pelvis, causing symptoms of more or less severe peritonitis. Even this is far from necessarily fatal, even if the spread is of considerable extent. Occasionally, when the abscess has attained a considerable size before bursting into the cæcum, its cavity may afterwards become distended with intestinal gas. I remember a case which happened many years ago, where this seemed to be the explanation of the sudden development of a good-sized tympanitic tumor in the abdomen, in the course of an attack of perityphlitis: this case recovered perfectly, and that attack proved to be the last of the disease.

A few cases happen where the abscess spreads to a dangerous extent; and then the help of the surgeon may be required.

Early rupture into the peritoneum, if it should cause diffuse suppurative peritonitis, is so generally fatal—even 70 to 80 per cent. when operated on—that surgical assitance is of little avail. But, as Treves remarks, "A case of death from perforation is very rare and does not warrant the routine early opening, even in the majority of cases."

Treatment.—I have read a quotation from Dr. Osler that there is no medicinal treatment of appendicitis. With the greatest respect for this author, I venture to say, as Sterne does of his text, in one of his sermons, "That, I utterly deny." A very great deal can be done by medical treatment, including the use of drugs, even in grave cases. It must be borne in mind that, though some cases are so light that they recover speedily and almost spontaneously, with rest alone, there is almost always some peritonitis, and often an abscess is formed. But neither the peritonitis nor the abscess is necessarily fatal; even extensive peritonitis may be recovered from. I recollect a case in 1889 which interested me a good deal, as the discussion on surgical treatment of the disease was then fresh and active. It was that of a lad who had a severe attack of appendicitis as the result of a fall on the kerb, while running in the city of London. When I saw him, about two days after the onset, there were not only well-marked local symptoms—swelling, etc., in the iliac region, but also more general peritonitis. He soon developed extreme tympanites, obstruction of the bowels and regurgitant vomiting, yet he made a perfect recovery.

I do not remember having seen a fatal case either during life or after death until the present year, when I met with one at last. It occurred in a lad, aged 16, who suffered from long-standing tubercular caries of the spine, and a more recent similar affection of the right hip, so that the lad was a cripple. There were no sinuses, though I suspected (erroneously as it turned out) a psoas abscess. He developed appendicitis, and though the symptoms were comparatively mild at first—he was so used to pain that he did not

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make much complaint—I recognised general peritonitis; but I was in doubt whether the cause was the appendix, tubercular peritonitis, psoas abscess, or the hip joint. He died from rapid collapse on the fourth day; and a *post mortem* revealed a sloughy appendix containing a concretion and universal suppurative peritonitis without any attempt at localisation by adhesions. I do not suppose that any mode of treatment could have saved his life.

The most important thing of all in every case is complete rest in bed from the outset. There is nothing more liable to bring on an exacerbation of the disease than allowing the patient to rise and walk about after a few days, when an abscess is beginning to form, although there may be a lull in the symptoms, as often happens.

Next in importance is almost complete starvation for a day or two, and very careful and scanty dieting for some time afterwards—especially the avoidance of solid food.

As far as more active treatment is concerned, the application of several leeches gives great relief at the outset: it may cut short a recurrent attack.

The administration of two drugs in particular is essential from the beginning:—1. Opium, in moderate but sufficient doses; the object being not only to mitigate pain, but to keep the bowels quiet, so that adhesions may form and the area of disease be limited. It is commonly advised by surgeons nowadays to abstain from opium, in order not to "mask the symptoms"—a most dangerous piece of advice, which, if followed, is only too likely to lead to the case falling into, and perhaps through, their hands. For the same reasons, drastic purgatives must be avoided, although it is well to have the bowels cleared out at starting. 2. Mercury. A dose of calomel iij-v gr. or more should be given immediately, partly to evacuate, partly to cleanse and disinfect the bowels. It should be repeated in smaller quantities at intervals during the next few days. During the whole course of the disease, a grain or two of calomel will at any time check any slight tendency to aggravation, as shown by pain, pulse, temperature, &c. But, in addition to the exhibition of calomel internally (with opium), mercury should be freely applied externally, in the form of ung. hydrarg., all over the seat of inflammation. It most distinctly relieves pain (as it does also in cases of female pelvic peritonitis), and it is more efficacious when combined with ext. belladonnæ—℥j., ℥j. to ℥j.

Fomentations can also be applied over this ointment.

Some physicians have found very great service from saline laxatives from the very first. They empty the bowels and relieve the congestion of the coats of the intestines. I always administer them in a mild form, and I do not think that they oppose the opium. Towards the end of the illness, aperients are still more required—salts or sulphur. I have seen the urine rapidly become pale and clear at this period after giving sulphur: whether *post* or *propter* I am not sure.

After the first day or two, it is nearly always my custom to order pil. plumbi. e opio, taking care that the pills are fresh and soft, so as to be absorbed. The acetate of lead is very beneficial in controlling inflammation; and it is a useful remedy, as Graves taught, for relieving the distension of tympanites.

Turpentine, a recognised remedy for peritonitis, is often of great use also, especially in the tympanites which may come on at a later period from peritonitis.

Throughout the complaint, the pulse, temperature, tongue, and urine, as well as local symptoms—pain, tenderness, and distension of the abdomen—are all important signs, and should be carefully watched; but probably the pulse is the most valuable index of all.

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After an abscess has formed, care should be taken to look out for pus in the stools; it will not infrequently be present. If the bowels require relief, a simple or glycerine enema is the best means to employ.

It is important to insist on complete rest in bed and strict light diet until all active symptoms have subsided—the pulse remaining normal, the temperature subnormal and steady, the tongue clean, the urine clear and pale, and the local pain, tenderness, and swelling having disappeared. If all these are natural, there need be little fear of a relapse or recurrence.

If any swelling or hardness persists over the cæcum, with tenderness, not due to fæcal accumulation, a blister helps to clear it away.

If care and attention be devoted to every case, it will very seldom be necessary to invoke the aid of the surgeon; and recovery will almost surely follow.

Patience may be required on the patient's part. A difficulty that we often have to encounter nowadays, especially since the King's illness, is the belief or superstition on the part of the public that every case of appendicitis requires an operation, and that to avoid it shows ignorance or want of skill on our part. If by any mischance the case should end badly, we might get blamed for not having had recourse to surgical interference. This may sometimes cause a little extra anxiety, but the termination usually proves it groundless. I have never had to advise an operation yet, and have not regretted not having done so.

Surgery may be required in some cases, of course. It may be justifiably resorted to in those cases of septic peritonitis which arise at once, without time for demarcation of an abscess; but the mortality of an operation in such cases is very great—70 or 80 per cent., hardly less than if left alone. Also if the general condition is serious from retained and spreading abscesses. The most dangerous and most obscure of these are those which spread upwards along the ascending colon behind the cæcum. As Treves says, the longer an operation is delayed the easier it is to reach pus.

I have tried to show in the above review that the resources of medicine are by no means despicable, even entirely the reverse; and that medicine can produce as good and even better results, even in severe cases, than surgery, even at the hands of the most skilful operators.

Were it true, which is very doubtful, that an operation at the very outset would not only remove the disease immediately, but be attended by infinitesimal mortality, it would still be preposterous to subject every patient thereto, considering that so many recover easily without; for an operation is in itself an evil, *i.e.*, so far as the patient is concerned—a fact which our friends the surgeons sometimes seem to overlook.

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THE CHEMICAL EFFECTS OF THE X-RAYS.

By F. J. Clendinnen, M.D., L.R.C.P., Lond.

At the last Congress, I mentioned my views concerning the stimulating effect of the X-rays. It was stated by a member that he had healed an ulcer of the breast by means of the X-rays, and I put forth my theory that the beneficial result was probably produced by an oxidising agent, *e.g.*, ozone or peroxide of nitrogen. We are well acquainted with the effects of peroxide of hydrogen if syringed into a sinus—how it stimulates the healing process.

I wish to bring forward some experiments that I have been carrying out, which show that probably the healing and stimulating effect of the X-rays is partly due to nitrogen compounds, nitrous and nitric acids being formed by the passage of the rays through the air. I may state that what drew my attention to the probability of this was the bronzing or pigmentation of the skin that occurs frequently in cases treated by the X-rays. We all know that if nitric acid comes in contact with any proteid matter that it will turn it yellow; this is due to the formation of xanthroproteic acid. We also are aware that this xanthroproteic acid, when it comes in contact with ammonia, deepens in color.

This is what occurs probably, these nitric and nitrous ions coming in contact with the skin give ries to the discoloration, and the ammonia in the skin darkens the color, and probably a further oxidisation, forming a brown. This theory, I think, also helps to explain the necrotic changes that take place in the tissues under prolonged application of the X-rays. These nitric ions are carried into the tissues, and set up that destructive condition which is so difficult to heal—a condition simulating a deep burn from nitric acid.

I will now explain how I set about my experiments. Firstly, I started an X-ray tube working, the anticathode pointing downwards into a glass vessel containing a very weak solution of phenol-phthalein already acted on by an alkali, *i.e.*, turning it a pale pink—this pink solution makes a very delicate test for acids.

The tube was allowed to work for ten minutes. During this time, the color of the liquid did not change, but, on shaking it up, the color gradually disappeared, showing, evidently, that an acid was present. In order to verify the test, I played the rays into an empty vessel rinsed out with distilled water for the same time as before, then shook up the test solution, which became, as before, colorless. To this colorless solution I added an alkali (soda), and the pink was readily restored, which evidently proves that the discoloration was due to an acid, and not to an oxidising agent, otherwise it would not be restored, except by a deoxidising agent.

Being armed with the assurance that an acid was present, the next thing to ascertain was what acid this was. Most probably it was nitric, the air being composed, roughly speaking, of nitrogen (78 per cent.), and oxygen (21 per cent.). If any compound of nitrogen and oxygen is formed, it is most likely to be NO_2 (nitrogen peroxide), which is the most stable, and this, chemically acted upon by water vapor in the air, with the result that nitric and nitrous acid are formed. *E.g.*, NO_2 = nitrogen peroxide. $2\,NO_2 + H_2O = HNO_2 + HNO_3$ nitrous and nitric acid.

In order to test the presence of this acid, I obtained some diphenylamine and made a 1 per cent solution with chemically pure concentrated sulphuric acid, which forms diphenyl-sulphonic acid; this being a very delicate test for nitric acid. It is said that one part in 3,000,000 can be detected by the above. I placed this standard solution in a burette.

I place beneath the X-ray tube an empty glass jar, previously washed thoroughly with distilled water, and start to energise the tube. At the end of ten minutes, I put in 1 ounce of distilled water in the glass jar, and place

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on it a ground glass lid in order to shake it up and let contents stand for a while. I now transfer the distilled water from the jar into a glass measure, and run in from the burette 1 c.c. of the standard solution of diphenylsulphonic acid, and at the junction of the two liquids, if nitric acid is present, there will be a blue band.

I. Made a blank test with distilled water and solution of indicator. No reaction.

II. Ran 1½ c.c. of indicator in 1½ ounce of distilled water, through which the rays had passed for ten minutes. A white flocculent substance appeared in the liquid, which soon began to show evidence of crystals forming, which separated in the light, and on standing the whole precipitate crystalised, remaining suspended midway in the solution.

III. Repeated the above experiment, taking care that the sulphuric ran to the bottom of the liquid to be examined, and a decided ring of a faint blue color appeared, with traces of precipitate as in II.

IV. Repeated blank test, and obtained slight opalescence without blue ring.

V. Passed rays through aluminium sheet, covering glass vessel containing 1½ ounce of distilled water for ten minutes. Repeated test with 1½ c.c. of indicator solution. Obtained same result as in III.

I wish to gratefully acknowledge the valuable assistance rendered to me by Professor Masson and Mr. M. P. Hansen.

NOTES ON CASE OF COMPLETE TRANSPOSITION OF ORGANS.

By A. Jarvie Hood, M.B., Glasgow.

Owing to the rarity of this condition, I consider it justifiable to bring the notes of this case before the Congress.

The patient, Toshugo Kubo (a Japanese cook), age 36, was admitted in the J ward of the Sydney Hospital on the 4th of June, 1904, complaining of cough for three weeks. His knowledge of English was so slight that it was impossible to elicit any definite history of his illness from him, but a Japanese interpreter gave the information that for some years he had been subjected to severe exposure, and had gone through the China-Japanese war.

According to the interpreter he had been fairly well up to three weeks ago, when his cough began. This symptom had come on gradually, and, so far as could be ascertained, there had been no definite rigors; but the expectoration had been very profuse during the whole of the three weeks. When admitted his principal symptoms were a constant cough and dyspnœa, with profuse and frothy expectoration. The urine contained a small quantity of albumen, but no blood or tube casts.

On Examination: Chest.—Defective resonance was marked on the left side, at the vertebral border of scapula, breathing was harsh, with rales all over both bases behind and in front. The heart sounds were particularly well heard over the posterior chest wall.

Heart.—The apex beat was in the sixth inter-space, 2 inches below and to the right of the right nipple. Heart dullness appears all to the right of the left sternal border, and is continuous with the liver dullness. The heart impulse is very diffuse. The sounds are irregular, and the first sound replaced by a systolic murmer most distinct over the fifth costal cartilage on the right side, but also heard along the lower half of the sternum. The second sound sometimes appears fairly distinct, but at other times is replaced by a systolic murmer: all these murmers are heard distinctly all over the chest behind.

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COMPLETE TRANSPOSITION OF ORGANS.

Diagnosis during life verified by post mortem, August, 1904.

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Liver.—The liver and heart dullness are conjoined, and form an area extending from 2 inches above the right nipple downwards and to the left, crossing the costal margin in the left nipple line and extending 2 inches below it; then across, keeping about 2 inches below costal margin.

There is well-marked œdema of both legs; pulse thin, and at times hardly perceptible. Osseous and nervous system apparently normal.

The diagnosis was evidently one of transposition of the organs, with, at least, mitral incompetency and the usual signs of cardiac failure.

Notwithstanding the measures adopted, he steadily got worse; the urine became very scanty, and the signs of dropsy increased. He died on the 9th of August, and the *post mortem* was performed the same afternoon by Dr. J. S. Thomson.

Post Mortem: Nature of Case.—Complete transposition of the organs; cardiac dilatation; mitral and tricuspid regurgitation; chronic venous congestion of organs; fatty and cirrhotic liver.

External Appearance.—Japanese. Surface of the body very œdematous; arms, legs, scrotum, greatly swollen; *rigor mortis* not marked.

Thorax.—(a) Cavities.—Each pleural sac contained a few ounces of clear yellowish fluid. Pericardium healthy. (b) Respiratory System.—Right lung weight, 1 pound 10 ounces; this lung had only two lobes; it was very congested and œdematous. Left lung weight, 1 pound 13 ounces: this lung had three lobes (well marked), congested and œdematous. (c) Circulatory System.—Heart weight, 1 pound 4 ounces. The heart was situated chiefly on the right side of the body with the apex pointing towards the right. The aorta arose from the right ventricle, and the pulmonary artery from the left. The venæ cavæ emptied into the left auricle, the right innominate vein crossing over to form the superior vena cava. The pulmonary veins emptied into the right auricle. The right subclavian and common cartoid arteries arose separately from one another from the arch of the aorta, while the left one came off as the left innominate artery. The aortic arch passed almost directly backwards, and then turned down on the right side of the spine. The right auriculo-ventricular valve (mitral) had two flaps, and the left three: both were widely dilated, also the ventricles and auricles. The heart muscle was fatty.

Abdomen.—Peritoneum healthy.

Alimentary System.—(a) Liver weight, 3 pounds 13½ ounces. The larger portion was situated on the left side, with gall bladder: in fact, there was complete transposition of all the abdominal viscera, including the relation of vena cava to aorta, the former being on the left side of the latter. The cardiac end of the stomach was on the right and pylorus on the left, spleen in the right hypochondrium, appendix and cæcum on the left, ascending colon on the left, descending colon on the right. Rectum entered the pelvis from the right. Liver fatty, cirrhotic, nutmeg. Gall bladder and ducts healthy. (b) Stomach and Intestines.—Catarrhal condition of mucosa. Appendix healthy.

Genito-Urinary System.—Right kidney weight, 6¾ ounces. Capsule stripped readily; organ swollen and congested; no diminution of cortex. Left kidney weight, 6 ounces, being similar to the right.

Bladder and Ureters.—Healthy.

Genitalia.—Œdema of scrotum: otherwise normal.

Spleen.—Weight, 2¾ ounces; firm and dark; glands healthy.

The brain and spinal cord were not examined.

Diamosis.—Endocarditis and dextro-cardia.

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A CASE OF ACUTE (SUPERVENING ON CHRONIC) PANCREATITIS.

By F. Lucas Benham, M.D., M.R.C.P., Lond.

The patient was a young woman, aged 24, engaged in domestic service. She had never had any serious illness; and, in particular, she had never suffered from jaundice or biliary colic. She always had a large appetite, but was sometimes dyspeptic, and was at the time anæmic.

On January 17th she had been eating some unripe grapes. Next day (18th) she had a pain, in the region of the stomach, on rising in the morning, which she ascribed to the above-mentioned indiscretion in diet. She got about and worked during the day, but could not easily hold herself upright, but bent forward to relieve the pain. She went to bed early in the evening. I was sent for to see her, and visited her about 8.15 p.m. I found her lying in bed on her back, with the legs partially drawn up. She was of dark complexion, but thin and anæmic, and was not jaundiced. She did not look dangerously ill. Temperature, 99.6; pulse, 90; tongue moist, slightly furred; thorax, heart, and lungs, normal; abdomen somewhat distended, walls very hard and rigid, especially at the upper part; the lower portion, including the right iliac region, being much less affected. There was great tenderness and she could not bear much pressure, especially over the epigastrium and below the ribs on the left side. No tumor could be felt. Liver dulness was present. Bowels confined. She had already taken 3 grains of calomel, which had caused vomiting. Morphin, ¼ grain, with atropine, was injected subcutaneously, and calomel, iij grain, was repeated. The pain was soon much relieved, though not altogether removed. Ordered to take nothing but a little milk with barley-water and arrowroot.

January 19th.—She had had an easy night, with some sleep. A Seidlitz powder given early in the morning had caused the bowels to be moved. I saw her again at 10 a.m. Temperature, normal; pulse rather below 90; Pain was much less, but the tenderness had not quite gone. The abdomen was softer and could bear handling, but not firm and deep pressure. Nothing abnormal was to be felt. The patient said she had never suffered from severe pain after food, from vomiting, or from hæmatemesis. Her mother had arrived and wished to take her home—about three miles distant. As she seemed fit to be moved, I gave my consent to her being driven there. It seemed to me then that the case might be one of intestinal derangement only, though overnight I had suspected peritonitis, and perhaps perforation of a gastric ulcer—most likely into the lesser cavity of the peritoneum. The mother promised to send for me if anything went wrong. I did not see her again during life, and the next that I heard of her was from her father, who called on me two days later (21st) to say that the patient had died rather suddenly early that morning. The account that I received of the further progress of the case was that the girl remained about the same for the rest of the day on which she returned home. Next day (20th) she was sick, and a sister who visited her in the afternoon thought she looked very ill, although the patient herself and her mother did not think it was serious. At 3 a.m. next day the patient became very ill indeed. Her father tried to get a medical man who lived nearer than myself, but he could not come. Death followed, from collapse, at 7 a.m.

An autopsy was made eight hours after death. The body was fairly nourished, but thin and anæmic. Cadaveric lividity was present and well-marked *rigor mortis* in all the limbs. Froth issued from between the lips. The rib cartilages were not calcified. There was very little subcutaneous fat.

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On opening the peritoneal cavity, no lymph was seen, and there was no redness or injection of the intestines: there was some clear yellow serum in the pelvis. The omentum contained very little fat: it was mostly a transparent membrane. In several of the small patches of fat were bright yellow specks, or knobs (fat necrosis)—mostly not more than $\frac{1}{8}$ inch in diameter. These were scattered about over the omentum, and to a less degree, over the meso-colon.

The stomach appeared rather small. It was not inflamed: there was no evidence of perforation or of the escape of any of the contents. On cutting through the transverse meso-colon, the posterior surface of the stomach was brought into view and found quite healthy also. The organ was removed and opened along the lesser curvature, and was found to contain only slimy, green, bilious, mucous fluid. Mucous membrane healthy, except for an area of extravasation—chiefly along the greater curvature and on one or two folds of the mucous membrane. Behind the stomach, in the region of the pancreas, there was seen a large nodular swelling, about as large as half an orange, very red in patches, with smaller yellow patches: it was hard and immovable.

Liver.—Large, pale, and fatty. Gall-bladder full of calculi, which were white and facetted. Three of them were as large as marbles, the rest were much smaller.

Spleen.—Rather small, normal in structure.

Kidneys.—Of full size. Capsule stripped readily. Cortex smooth and of good thickness. There was a superficial cicatrix in the upper part of the right kidney.

Pancreas.—The tumor above referred to was evidently the pancreas. It was removed with great difficulty, as it was bound down firmly to the spine and mesentery by dense fibrous tissue, and was tightly adherent to the duodenum and adjacent bowels, so that it was not separated without lacerating them. Below it was an enlarged and partially calcified lymphatic gland. On removing the organ it appeared to have a fibrous capsule—very red and injected with ecchymoses. Patches of the surface were green, others yellow. The organ was firm, but not indurated. On section, the lobules were distinct and for the most part normal to the naked eye. The fibrous septa were very red. Here and there, especially on the surface, groups of lobules were green, amorphous, and gelatinous—almost diffluent. Others had a yellow color. The same was seen, to a less extent, in the interior of the gland. The whole pancreas was considerably larger than normal.

Heart.—Pale and flaccid. No epicardial fat. Valves all quite sound. Right ventricle contained some loose clot as well as some firm and tenacious coagula—some black, some pale and gelatinous—entangled in the *columnæ carneæ*. The walls were pale and soft. The left ventricle nearly empty.

Lungs.—Normal: a few slight thin bands of adhesion on the left side.

Intestines.—Normal (externally). No injection or lymph.

Uterus and bladder.—Normal.

Remarks.—Though a considerable number of cases of acute pancreatitis have now been published, all that occur are of sufficient interest to be recorded. The points of interest about the above case are:—

1. Taking all the symptoms during the course of the illness from beginning to end, they presented a fairly typical example of acute pancreatitis. At the beginning, and when the case passed from my notice, I was quite unable to recognise the disease. I suspected that there was local peritonitis—possibly from perforation of the posterior wall of the stomach; but as the symptoms had improved so much during the first night, I could not be sure. I had only met with one fatal case of the disease before: it was in London, several years ago. I saw it in consultation, and thought it was one of local

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peritonitis; my colleague took it to be one of intestinal obstruction, or *vice versa*. The patient was sent into the hospital; laparotomy was performed, and death ensued. The cause of death was discovered; and that was the first I ever heard of the disease, which had then only recently been described.

2. The age of the patient is unusual. It is very uncommon, I believe, to find the disease in a young woman of 24. It is also uncommon to meet with gallstones at that age. The co-existence of two such rarities suggests a causal connection between them.

3. The association of a quantity of gallstones with pancreatic mischief. This is almost constant in pancreatitis, both acute and chronic, though the exact nature of the relation is doubtful. It has been supposed that a gallstone blocks the outlet of the two ducts and thus causes regurgitation of bile into the substance of the pancreas. In some cases this explanation is quite possible; but in others, as in the present, where there has never been any jaundice, it is scarcely tenable. Also, jaundice is very often present without causing obvious damage to the pancreas. I am sorry that the dense fibrous tissue surrounding the parts prevented me from making a dissection of the ducts. It is very probable, however, that the presence of gallstones in the gall-bladder in some way causes irritation of the pancreatic ducts and glandular substance, which may set up inflammation and destruction of the gland.

4. The dense fibrous issue which in this case enveloped the pancreas, especially at the head, and caused firm adhesion to the duodenum, &c., afforded indubitable proof of previous attacks, or of chronic disease; yet, from the history of the case that was obtained, there was no evidence of any serious illness of the sort; which shows how mild, or latent, it may be.

The matter is of considerable importance; and it is much to be desired that there should be means of readily identifying attacks of pancreatitis, even though slight. If this can be done, much can often be effected by appropriate treatment. In some cases, where chronic pancreatitis has been recognised, a cure has been effected by surgical operation directed towards the gall-bladder, draining it and removing any calculi present. In most of these cases there has been jaundice, which has served to direct attention to the primary seat of the disorder. In a few cases, I believe, a simple laparotomy has led to recovery. Still, there is no need to resort to surgical interference unless the attacks are frequent and severe, or of long standing. Milder ones can probably be overcome by simple medical treatment. It would be more satisfactory, however, to be able to distinguish them with certainty, and to know whether they are invariably dependent on the presence of gallstones, &c.

TUBERCULIN IN DIAGNOSIS AND TREATMENT.

By W. Camac Wilkinson, B.A., M.D., Lond., F.R.C.P.

Infectious diseases of specific origin are the result of an affinity existing between the specific cause or its product, or both, and the tissue elements of the man or animal. The result of the disease depends on the action and reaction of these two essential factors. If the specific cause is very virulent or very abundant, it overpowers the vitality of the invaded tissues, and destroys the organism. On the other hand, the resisting energy of the tissues may overpower the agents of the disease, and either destroy them or neutralise their dangerous products. Tubercle bacilli, the specific cause of all forms of tuberculosis, manifest a remarkable affinity for certain parts of the human

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body, notably the lungs, and the result of tuberculous disease of the lungs depends on the virulence and number of tubercle bacilli and the resisting energy of the invaded tissues.

In the treatment of such an infectious disease there are two rational methods, one directed against the specific cause, and therefore called specific, the other aiming rather at strengthening the resisting energy of the invaded tissues. These two distinct methods of specific and general treatment should be combined if we wish to get the best results.

The specific method of treating pulmonary tuberculosis consists essentially in the use of tuberculin in its various forms, which helps to destroy the bacillus and to neutralise its toxic effects. This method is in fact a process of active immunisation, entirely different to the antitoxic treatment of diphtheria, in which the antitoxin is supplied ready-made. In principle there is no difference between the tuberculin treatment for the purpose of curing tuberculosis and the use of Haffkin's fluid, or similar agents, for the purpose of preventing for a short time such diseases as plague, cholera, or typhoid fever. If, then, the method is advocated in order to protect a healthy person against a disease—which, in a mathematical sense, he has a very small chance of contracting—it is not unreasonable to urge the use of the method if it offers even a small chance of curing one who has already fallen a victim to the disease. Those who have used and advocated the use of specific prophylactic fluids containing germ products for the prevention of plague cannot, with any show of consistency, oppose the use of tuberculin, which is, after all, merely a vegetable product of the living tubercle bacillus. Many are, in their ignorance, guilty of this strange inconsistency. On the other hand, I may disarm some opposition by saying at the outset that the extraneous use of tuberculin is not the one only indispensable means of curing tuberculosis—even in an anatomical sense. We know of no disease in which death is inevitable. Always some individuals are ordained by nature, or are strong enough, to survive an attack of the most virulent disease. Plague does not kill all its victims; nor yet tetanus or anthrax. This power to resist lies in the tissues. Let us go a little further. This power to resist depends upon the ability of the tissues to produce a sufficiency of antibodies which either kill the specific agency or render their products harmless. It is too often forgotten that the very and only cause of the production of these antibodies in the tissues is the specific cause of the disease. *The disease works out its own cure by means of its own cause.* Not only the disease, but even the mechanism of immunity that cures it are both the results of the living germ through its specific products. Except in this way, no infectious disease cures itself or protects against itself. Even when we seek to strengthen the energy of tissue, so that the disease may be resisted, we are blindly assisting nature to work a cure in nature's own way. In pulmonary tuberculosis sanatorium treatment, open-air treatment, the rest cure, and other methods act by stimulating the system to evolve the mechanism of immunity in response to the action of the products of the tubercle bacilli in the tissues, the most important of which is the tuberculin. It may be a shock to some of the advocates of sanatorium treatment to hear that they themselves, without knowing it, have been innocently vaunting the efficacy of tuberculin in the treatment of tuberculosis. On the other hand, those who advocate the artificial supplementing of nature's own direct weapon of defence are quite consistent in upholding the value of other methods, especially sanatorium treatment. Daily experience tells us that pulmonary tuberculosis is a disease that too often fails to work out its own cure. Some fault in the mechanism of immunity allows the germ to continue its work of destruction, or, maybe, to lie in ambush for months and years. The whole purpose of treatment is to stimulate the tissues, to build up and

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finish the mechanism of immunity, so that the tissues shall no longer be at the mercy of the tubercle bacillus. *A priori*, it seems logical to stimulate the mechanism by means of the specific products called tuberculin: *a posteriori*, other methods evolved through empiricism are vaunted as the *ne plus ultra*, and specific methods are held to be superfluous.

Unfortunately there is no royal road to an accurate estimate of the value of treatment in a disease so irregular in its onset, so complicated in its nature, and so variable in its course as pulmonary tuberculosis, and the best of us may hardly avoid the pitfalls of inconsequent and circuitous reasoning. Least of all can we judge of the effects of treatment if we report and exhibit our immediate successes. Those who have had a large field for observation are at times staggered at the vagaries of the disease. The science of recent years, especially through the labors of Vagedes and Kossel, working under the inspiration of Koch, tell us in unmistakable language that one of the chief causes of variation has been largely ignored—the variable virulence of the essential factor, the tubercle bacillus. In the old days the natural explanation of the variable course of pulmonary tuberculosis was found in the over-rated element of predisposition. The tendency of the old school is still to labor the question of predisposition. The tendency of the new school of heretics is rather to give due weight to the definite, tangible, and measurable actor—the infective agent in all its variations and vicissitudes. Naegeli's statistics would seem to show that we are all predisposed: *Tedermann ist ein bisschen tuberculos*. The strongest athlete, in the heyday of health and youth, may be just as little immune as the slender weakly factory girl, with the flat chest and so-called phthisical habit. The former may succumb to rapid and acute phthisis; the latter may show only an inert localised lesion. Predisposition at least is a quality that cannot be estimated, nor yet eliminated.

It is difficult beyond ordinary comprehension to give a right value to the effect of any method of treatment in a disease that runs such a variable course as pulmonary tuberculosis. The disease may exist for months and years without arresting the attention of patient or physician. It may become quiescent at any time; it may relapse after months or years; it may reach a serious stage involving the greater part of a lobe, and then end in perfect restoration of health. In spite of treatment of any and every kind, the disease may never relent; the victim knows no respite till death; without any treatment, the disease may come to a standstill, and never again disturb the health. The most affectionate regard for logic may not restrain us from attributing success to our own small efforts and failures to the vagaries of the disease. Our memories seize fast hold of successes; we leave others to record our failures, or leave them unrecorded. Let us adopt what measures we may, more or less failure is the rule, and yet we place a high value on many of our methods. In no other disease is it so difficult to estimate fairly the effect of treatment upon the course of the disease as it is in pulmonary tuberculosis. One sees cases of pulmonary tuberculosis, even complicated with tuberculous ulceration of the larynx or epiglottis, get well of themselves without any special treatment, without sanatorium treatment, without injections of any sort, without local applications. The best results of sanatorium treatment do not present such a satisfactory picture of success that we should rest satisfied. Analyse the statistics of Weicker's Sanatorium, or Englemann's more extensive analysis of the results in German sanatoria. We shall have to travel far before we can be satisfied. To paraphrase Lord Rosebery's epigram, the physician who is satisfied with sanatorium treatment is lost. The rational method of Brehmer was sound as far as it went, though often laborious and tedious in practice, but no other method was possible in his day. Sanatorium treatment holds out much hope of success, especially

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in the early stages of the disease; but surely any method that fails in at least 50 per cent. of the cases does not satisfy the conditions of an ideal method. The plague, dreadful as it is, is more merciful—it kills quickly, but it hardly kills 50 per cent. of its victims. The results of sanatorium treatment disclosed in Weicker's careful tables shows that there is plenty of room for improvement.

In the first place, early diagnosis is the key to successful treatment. In the early stages the lesion of active or latent tuberculosis may produce physical signs like those of obsolete foci, and tubercle bacilli may not yet have escaped from the tissues; the lesions are not tuberculous, but part tuberculous. How can the physician attempt to unravel the mystery of these various lesions? If the tubercle bacilli have disappeared, there can be no tuberculosis; and if there is no active tuberculosis, there is no reaction to tuberculin. That is a vital point. Tuberculin alone gives the clue, whether the lesion is active or inert. If the focus is inert, it may harbor tubercle bacilli that will become active at some future time. Yet, if the tuberculin reaction fails, it is likely that the tubercle bacilli are so surrounded that the tissues can themselves destroy the bacilli. On the other hand, a reaction to tuberculin proves that the lesion is not inert. This reaction is a danger signal of the first importance. It warns the physician of a danger hidden from sight, like the fog signal warns the sailor of a danger in a mist which the eye cannot penetrate. By the systematic use of tuberculin we may learn the true nature of many of those lesions which Naegeli too readily assumed to be tuberculous. Many of these lesions may be merely passive, quiescent, but still ready to be kindled into activity by inflammations or infections. Sad to say, these closed lesions thus neglected may become the source of the acute and rapidly progressing forms that develop in early adolescence. Now, if these terrible disasters may happen, there has been no cure, even though the lesion is for a time closed and inert. By no other way than by the systematic use of tuberculin can we distinguish these latent cases from absolute cures; and if tuberculin is not used, no one has a right to use the term *cure*. If the tuberculin test is negative, and after an interval of three months or six months again negative, there is some certainty that a cure has been accomplished. When the prejudice against tuberculin has died its natural and inevitable death, one of the most valuable uses of tuberculin will be found in determining the effect of treatment of any and every sort. Meanwhile, except by those who use the test, the term *cure* must be avoided. It is satisfactory to note that the Tuberculosis Congress in London, 1901, deliberately adopted the opinion that tuberculin was an invaluable agent in diagnosis. The education of English opinion needed ten years before the truth of Koch's original statement was recognised.

Sanatoria cannot dispense with tuberculin as a diagnostic agent, and gradually tuberculin is taking its right place in all the sanatoria of Germany. If tuberculin is not used, mistakes must occur. True cases of tuberculosis are overlooked; and, on the other hand, there is the risk of condemning a man to an expensive and irksome course of treatment for a disease which he has not got. In one year Weicker rejected forty-eight spurious cases already diagnosed by good examiners as early cases. In the eager chase after early cases, otherwise trustworthy examiners may condemn to sanatorium treatment many cases in which the diagnosis rests not upon trustworthy evidence—such as tubercle bacilli in the sputum, or a definite reaction to tuberculin—but rather upon the subjective impressions of the observer as to the character of respiratory movement or auscultatory signs. The tuberculin test alone can save the profession from the yes-no diagnosis. But for the tuberculin test, Weicker might have greatly improved his statistics by including the

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rejected cases, and ultimately *recorded them as cases of cured tuberculosis!* Christian science can do as much for these cases as the best sanatoria. Let us take care we give no such chances to the Christian scientists to show their skill. It is an easy off-hand criticism to offer that tuberculin in diagnosis may lead to the inclusion of cases of latent tuberculosis that may never further develop. The same may be said of many of the cases upon which the statistics of sanatoria are based. Who will dare to say that it will not develop, if a reaction to tuberculin occurs? On the other hand, I have saved many persons the expense and inconvenience of exile to the country, to which they had been sentenced by various medical men. Further, when one uses tuberculin for treatment also, if perchance tuberculin has given a doubtful or false indication, the mistake is soon remedied. Thus, if the reaction be no greater than 99°.5-100° F., and tuberculin is given for treatment, a constant absence of reactions with larger doses rapidly increased arouses suspicion. In my experience with proper doses a temperature of 100° is positive. A rise of temperature to 99°.5-100° is doubtful. *Tuberculin is quite harmless if there is no tuberculosis.* Why, then, should we fear to use it in the cause of sanatorium or other methods of treatment, when it surely reveals the presence of tuberculosis lesions? I have no hesitation in saying that tuberculin is an invaluable agent in the selection of suitable cases for sanatoria, and also a ready means of gauging the success or failure of treatment. A more open question is the value of tuberculin as a curative agent. It is extremely difficult to measure the value of different methods of treatment in a disease that runs such a variable course. For any and every method of treatment successes will be claimed. The only test is the *relation of successes to failures.* If we investigate the statistics of sanatoria, we find that the successes of to-day are the failures of to-morrow. Every medical man has had successes, but far more failures. Records are practically worthless *unless they show that after an interval of time—say two years—the patient is the better for treatment.* It is the tradition of sanatoria to publish results as soon as patients leave the institution. This pernicious practice must give rise to misconceptions and disappointments. Dr. Sinclair is an expert in sanatorium methods, and I fully recognise the humane and unostentatious labor of the last two years of his busy life. He knows too well the vagaries of the disease he has to deal with to raise hopes that may not be realised. He knows as well as I do that the immediate results of sanatorium methods, as of other methods, are satisfactory in every point but the most important—*they do not last.* The sequel to the chapter of improvements that stand recorded when the patient leaves the sanatorium must be stated with equal truth, and, sad to say, is a long wearisome chapter of relapses terminating in death after one or several years in the majority of cases—even though at the outset special care is exercised in selecting cases favorable for treatment.

Anyone who has studied the analysis of 30,000 cases, or thereabouts, treated in German sanatoria, will hardly find occasion to boast of brilliant success. The fruit of the English sanatoria and of our own sanatorium in New South Wales is not yet ripe for analysis—I said so in 1903, Ransom says the same in 1905. What is true in 1905 was doubly true in 1903, though I was laughed to scorn when I made the statement. Engelmann's statistics show that of the cases in Stage I. 7.6 per cent. were disabled or dead in one to one and a half years; 19.7 per cent. in two to two and a half years; 33.3 per cent. in three to three and a half years; 55.6 per cent. were disabled or dead in four years. In all stages the records were infinitely worse: in one to one and a half years 25 per cent. were dead; in two to two and a half years 38.4 per cent.; in three to three and a half years, 55 per cent.; in four years the deaths amounted to 66.7 per cent.

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Moreover, the failure of present methods is written large in the black letters of the statistics of every country. The after history of 20,878 cases treated in 1897 showed the following permanent results (Engelmann). In 1898, 66 per cent. permanent results; in 1899, 48 per cent. permanent results; in 1900, 39 per cent. permanent results; in 1901, 30 per cent. permanent results. In Weicker's statistics—2,469 cases. In following year, 55 per cent. permanent results; in 2nd year, 50.7 per cent. permanent results; in 3rd year, 41.4 per cent. permanent results; in 4th year, 35 per cent. permanent results.

Two years after treatment 25 *per cent. are dead*; *four years after treatment* 50 *per cent.*; *and six years after treatment* 63 *per cent. are dead.*

Even Dr. Sinclair has been premature with his records. When I published my results in 1902, my work extended back five years and more. Dr. Sinclair's extend back hardly more than two years, and most of his successes have been reported within a year of treatment. The condition of time has not been fulfilled, and his records can at most form a starting-point for further observations. Many of the apparent successes of to-day may prove the certain failures of to-morrow. Still, the after history of these recorded cases year by year will show the truth about the results of sanatorium treatment.

We learn already from Dr. Sinclair, as I insisted years ago, that a three months' residence in a sanatorium may be well enough from an insurance point of view, since it may restore the industrial competency of a workman for one or more years; but in three months one cannot expect to arrest the disease, and it is arrest of the disease which the workman is led to expect and desires. The workman wishes not only to be able to work, but to recover his health. In most cases the work of sanatoria is patchy and unfinished. Patients are discharged—improved, no doubt—but with the disease still potential, if not active, and merely waiting a favorable opportunity to reassert itself. My purpose at this Congress is to bring forward reasons for advocating the use of tuberculin as a remedy, so as to bring the work of the sanatorium to a higher level of efficiency and completeness, for the benefit of the unfortunate victim of pulmonary tuberculosis—and even of the whole community. The better the work of the sanatorium, the better for the community; and at the present time the inmates of our sanatorium are denied the advantages of tuberculin treatment that are enjoyed by patients in some of the best German sanatoria. The ideal treatment is specific treatment combined with those general methods adopted in the sanatorium that are of undoubted value in increasing that tissue energy upon which the success of tuberculin treatment depends. Weicker lost hope after a careful experience of the ordinary and best sanatorium methods of Brehmer; he weighed sanatorium treatment in the scales, and found it wanting. He tried tuberculin, and, combining both methods, he has taken heart again. Another very great authority, Mœller, has become a convert to our side, but he states that he uses tuberculin especially when sanatorium methods absolutely fail (see Zeitschrift, für Tuberulose); and in such cases he has been astounded at the splendid effects of tuberculin. My simple criticism of this somewhat half-hearted advocacy is that surely, if tuberculin has been proved by him to be of enormous value as a curative agent, when *sanatorium methods have failed*, there is no *a priori* reason why it should be far more successful in those cases in which sanatorium methods do good.

Years ago, when I was making my early observations with tuberculin, I studiously avoided the introduction of factors that might vitiate my conclusions. Accordingly I instructed patients to live under the same conditions to which they had been accustomed. Having thus proved the uniform im-

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provement could be only due to the new factor—the tuberculin—I lost no time in adopting all measures that might reasonably be supposed to help in the arrest of the disease and the cure of the patient.

Strange as it may seem, I am the only specialist in diseases of the lungs in Sydney who has gone to the trouble of providing sanatorium treatment for private patients—even though I consider the sanatorium methods of subordinate importance. Other medical men, who openly proclaim that sanatorium treatment is of primary importance, have taken no steps to secure for their private patients that method of treatment which they tell the world is the best, and, indeed, the only treatment. It is idle to pretend that the haphazard exile of patients to the country constitutes sanatorium treatment. I leave it to the over-zealous advocates of sanatorium treatment for the poor to explain why it should not be equally successful in their well-to-do and rich patients. Let those who wish to be enlightened upon the great virtues of sanatorium methods read Mr. Ransom's recent article in the *British Medical Journal*. He quotes Walther, of Nordrach, to this effect, that if by cure we mean that all subjective and objective signs of disease have disappeared, no more than 11.20 per cent. can be so classed, although improvement occurs in 70.80 per cent. of the cases. Ransom writes himself, "I think it may fairly be said, as the result of a large mass of continental statistics, that only about 30 per cent. of patients discharged from sanatoria for the poor maintain the capacity for work more than four years." He also writes, "In England sanatoria for the poor have existed too short a time (writing himself in 1905) to allow of statistics of much value being collected from them." I said the very same thing quite two years back—in 1903. Later Ransom says, "Exactly half the patients discharged have maintained the improvement gained for over a year, while half have got worse or are dead."

Professor Mœller, of the Belzig Sanatorium, near Berlin, and a recognised authority on tuberculosis, six years ago an utter sceptic upon tuberculin treatment, is now one of its most earnest advocates. He tells us that, in cases in which sanatorium methods have entirely failed, tuberculin has proved to possess undoubted curative properties, and that while *sanatorium treatment never cures in three months*, permanent cures are often obtained with tuberculin. In his first record Mœller showed gross results:—Sanatorium methods alone, 10.9 per cent. cured; sanatorium methods and tuberculin, 36.3 per cent. cured. In the different stages:—Stage I. Sanatorium methods alone, 31.8 per cent. cured; sanatorium methods and tuberculin, 75 per cent. cured. Stage II. Sanatorium methods alone, 1.9 per cent. cured; sanatorium methods and tuberculin, 20 per cent. cured. Stage III. No cures possible, except as curiosities.

According to Mœller's later experiences sanatorium treatment may cure pulmonary tuberculosis in the first stage (Berlin Board of Health system) in 25.30 per cent. of the cases, while tuberculin in his hands has secured the same result in 84.6 of the cases. I venture to say that when Mœller uses larger doses, or repeats the course, he will find that tuberculin will cure almost 100 per cent. of the cases. Thus, I hold with our great teacher, Professor Koch, that *tuberculosis of the lungs in the first stage can be cured with certainty by tuberculin*. In the second stage, which Mœller maintains is never cured by sanatorium treatment alone, tuberculin gives *permanent results in from* 40 *per cent. to* 60 *per cent. of the cases*. Even in the third stage tuberculin sometimes yields remarkable results that cannot be approached by sanatorium methods. When these splendid results are claimed for tuberculin by those who have had an extensive and prolonged experience with it, are we

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going to be so foolish as to pay any heed to the carping surreptitious criticism of those sterile authorities who have the hardihood to express adverse opinions on a matter concerning which they have no personal knowledge ?

In medicine, as in surgery, pioneers have often had to bear much unfair criticism. Experience is our great teacher. I do not know any great authority who has used tuberculin, with the conditions and limitations laid down by Koch, and found tuberculin wanting. On the other hand, I have seen no carefully compiled records that prove tuberculin to be either harmful or useless. We do not condemn operations because novices and those who have not learned their art make mistakes and fail. There have been terrible tragedies arising from various operations in their early history, but the operations have survived. The failures and tragedies associated with tuberculin are not the fault of tuberculin, but the fault of those " fools who rush in where angels fear to tread." Failures may be the stepping-stones to high success, and the failures of to-day are converted by increasing knowledge and experience into the successes of to-morrow.

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SECTION OF SURGERY.

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ABDOMINAL SYPHILIS.

By Frederick Dougan Bird, M.B., Ch.M., M.R.C.S., Eng., Melbourne.,

President of Section.

While appreciating to the limits of my capacity the honor which allows of my presenting myself to you to-day as President of the Section of Surgery, I am deeply diffident of my ability to interest you in a presidential address, and I can only beg you "gently to hear and kindly to judge" my attempt. On account of the size of the subject, and the fact that the various professional annuals do the thing so much better than I can, I have avoided attempting a review of the present surgical position, and have chosen a special subject which has attracted my attention for a good many years. In the abdomen, that portion of the body which shows the surgeon the greatest help in his work, and also possibly his greatest difficulties, and which has a perennial interest of the highest order, I have found the conditions about which I wish to speak to-day.

While syphilis has been made responsible by various authors for most things, from the production of cleft palate and club-foot to the filling of our lunatic asylums, but little has been written in comparison about the manifestations of this disease in the abdomen. Mistakes are so easily made in connection with syphilitic lesions in the abdomen, and these mistakes so generally mean greatly increased risk to the patient from operation, that I need not ask your pardon for endeavoring to elucidate to some extent these conditions from the thin thread of my personal experience. A somewhat systematic method of considering these lesions is the best way of detailing to you what I know from the study of a number of cases.

In returning to the ancient saintly habit of contemplating the navel, I must admit that while I have seen epitheliomatous and tubercular diseases, fæcal and urinary fistulæ, and dermoid developments here, I have not encountered anything that I took to be syphilitic at the umbilicus itself. However, round about the navel I have met with true subcutaneous gummata, the size of small marbles, fairly defined movable growths, slightly tender, which simulated multiple sarcomata closely. They appear just about the level of the waist, and may be due to the dragging of the clothes in the female, which, acting so continuously, amounts to traumatism. Similar, but smaller, subcutaneous gummatous nodules are well known about the female knee, and their position is similarly determined, I believe, by the irritation of the garter. When the breast is voluminous, and weighs much upon the upper limit of the corset, subcutaneous gummata may form in like manner along the line of pressure.

Increased experience makes me allow increasing importance in my mind to the influence of traumatism as a localising factor in the production of syphilitic manifestations. These strictly subcutaneous gummata are mainly important from their showing that their owner has the specific virus still in

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his or her person. A single specimen may be removed to make quite sure by microscopical examination that it is neither one nodule of a sarcomatosis nor an outlying metastasis of an unobstrusive gastric or mammary cancer.

Considering next the parietal wall itself, I have had the good fortune to see a number of tumors (it is hard to call them otherwise) in the parietes due to syphilomatous growth. These could have been divided into cases where the parietes alone was implicated, and cases where underlying structures were infiltrated and by being fixed to the wall where the syphiloma existed formed part of the swelling. The first-named are so like sarcomata of the abdominal wall as to surely cause mistake on the part of the surgeon if he is not cognisant of the syphilitic possibilities of the case. And a mistake of the first order it will be if he operates for the removal of the supposed sarcoma, for if the swelling is of any but small size, and it generally has considerable extension, the patient's abdominal wall is grossly injured past repair by the cutting necessary for its removal.

It is very hard to believe that this discoid well-defined lump can possibly be anything but a circumscribed tumor; and unless the knowledge of the patient's past tells us, or the finding of undoubted traces of syphilis elsewhere puts us on the right diagnostic track, no certain view can be taken, and an incision into the growth is necessary for the removal of a portion of the tissue for the microscope. This insuperable difficulty in clinical diagnosis certainly exists in the hard example of parietal gumma, which takes such a time as a sarcoma might take to grow, though of a shorter existence than a fibroma; but all the cases I have seen were not so dense and fibroid. These softer ones may grow more rapidly than any sarcoma, and may disappear as rapidly. In one case in particular was I much astonished at the marvellous disappearance of these masses. It was that of a married woman, *æt.* 46, who had several apparently healthy children, had had one miscarriage, and presented no signs of syphilis elsewhere. She came to me slightly jaundiced, with a tender swelling in the gall-bladder region. This swelling was slowly travelling in the direction of the umbilicus in the same manner as an enlarging gall-bladder would. However, the swelling implicated the parietes, and my diagnosis was cholecystitis, with cholangitis and the probable presence of gallstones. Operation showed me infiltration of the parietes, to which were welded on the gall-bladder and a portion of colon. The mistake was easily made by me, as I have seen cases very similar in which gallstones were trying to extrude themselves at the umbilicus.

Though I was now used to recognise these syphilomatous growths, I might have easily failed in diagnosis at operation if it had not been for the peculiar character of the infiltrated rectus muscle, which gave a gritty feel on section. This is a marked characteristic of muscle tissue embarrassed by syphilomatous change. The sensation given to the hand through the knife on section of such a muscle is highly reminiscent of the cutting of beef which has been badly corned, and the appearance, which shows much increase in the stroma of the muscle, suggests, through another sense, the likeness to corned beef. This similarity revealed to me the right diagnosis, which was confirmed by D. Mollison's microscopic examination of a portion removed.

Though I did not open into the gall-bladder in this case, bile came through the wound in a few days' time, and continued to flow for several weeks. Now this lady during her convalescence developed rounded masses in various parts of her abdomen, which masses undoubtedly infiltrated the parietes and deserved the name of phantom tumors, because of their fitful appearance and equally capricious disappearance; they were much softer than the original tumor, and caused me much exercise of mind at first. I would mark one down on Monday, and on Wednesday it would have vanished, a similar tumor

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developing elsewhere in the abdominal wall. My concern at the first was that I should incise these lumps, but, fortunately, they were too evanescent. All this time the patient was being energetically treated with iodide of potassium and mercurial inunction. This lady regained her health completely in about four months, and, as seems to be the rule in these unusual manifestations of syphilis, no other undoubted evidence of the disease could be obtained.

Another illustrative case was that of a young married woman, who developed a considerable tumor in the gall-bladder region. This was operated on in a country hospital, where the surgeon, a very excellent practitioner, found such a jungle of adhesions that, after some exploration, which did not discover gallstones, he closed the abdomen. The patient recovered wonderfully, the tumor disappearing, all but some thickening beneath the scar. In a few months' time, however, her health failed again, and the tumor returning her doctor sent her down to me. The history of the case and the parietal infiltration constrained me to the right diagnosis, which was confirmed by the removal of a small portion for the microscope. Complete permanent restoration followed drug treatment. The power of exploratory operation by itself to almost effect dispersion of the syphilomatous condition was of great interest to me in this case.

The existence of the syphilomatous process in the tissues does not necessarily interfere with the production of a good scar made for diagnostic or exploratory purposes, as in only one case that I have seen has there been a tendency to weakness of the cicatrix.

It is equally embarrassing when such an agglomeration of syphilomatous material is discovered in the appendical region as occurred in a case under my care some years ago. The patient, a previously healthy young fellow, had a deranged alimentary canal, and was evidently ill. He drew my attention to pain and tenderness in the right iliac fossa, where I found a swelling such as is often seen in appendicitis, with a good barrier formation. The diagnosis was made, and as the swelling gave no signs of disappearing, at the end of a few days an incision was made into it, but I did not open the peritoneal cavity, as the muscle was in a condition which immediately turned my thoughts to syphilis. The microscope proved that their direction was right, and the patient regained his health, with the evanescence of the tumor under liberal anti-syphilitic medication. In this case the central portion of the tumor was hard and immovable, but the peripheral parts changed in a most perplexing manner. Here, again, no history or vestige of syphilis was to be found. Both these cases suffered much in health with the formation of these strange tumors, while previously to the appearance of these swellings their health had been excellent, although they must have been syphilitic for some considerable time before the evolution of syphilomatous masses. A deep etiological interest attaches itself to a further consideration of these cases. In a person admittedly syphilitic, though in apparent good health, does some accidental lowering of health cause the formation of these growths? Or is there some localising factor, probably an unregarded traumatism, which determines the agglomeration of small round cells, that in their turn, by their morbid metabolism, poison the patient, and thus reduce his health? The latter, I believe, is the true record of the loss of health and strength which is so noticeable in these cases. It is only likely that the vast collection of small infiltrating cells will give rise to strange metabolic results, which are of the nature of a poison, especially when the tumors formed by them may be so wanting in stability in all or part of their diffusion.

These parietal tumors occur not only in adults, but also in infants and children as developments of inherited syphilis. In one case during the time

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I was treating the father for tertiary syphilis (stenosing the right bronchus), the mother gave birth to a child apparently healthy. In about a fortnight the infant became sickly, and a lump came into existence to the right of its umbilicus. This lump grew rapidly to a large size when compared with the abdomen, and incision with removal of a portion demonstrated the syphilitic nature of the swelling.

Under mercurial treatment the child flourished; but the father died of his disease. Fortunately here, with the undoubted syphilis of the father before me, I avoided the danger of performing an extensive mutilating and unnecessary operation on the one hand, and the hopelessness of allowing the child to die of what I might have considered rapid sarcoma on the other. The mistake of resorting to a large operation which is not needed is very likely to occur in cases where the tumor is hard and confined to the parietes. Moderate incision into them with the giving of mercury and iodide is the best treatment for these hard parietal tumors, and the cutting for the removal of a portion for the microscope gives a start for the absorption of the tumor. Some of the less stable forms are dispersed by mere incision, and it is my opinion that all are more readily disposed of by the performance of an initial incision, which has the added advantage of settling the diagnosis.

The worst treatment is total removal of these tumors, leaving an irremediable gap in the abdominal parietes. Among the many wonderful things we witness in our work, not one, I think, is more startling than the disappearance of these syphilomatous masses of large extent and dense consistence under proper treatment. The likeness to tumor is still more marked when we realise, as I have been able to do, that the central softening so characteristic of syphilis formations is by no means a necessity in these masses. That a single surgeon should meet with the number of cases which have come my way goes to show that these strange syphilomatous swellings are not so uncommon that we can neglect the chance of their being present. From what information I have been able to gather of their frequency, they are more common than sarcoma or fibroma of the abdominal parietes.

There is yet another tumor with which they may be confounded. Myxœdematous deposit, when it occurs in the abdominal wall, is usually spread out impartially in the subcutaneous tissue, but sometimes it may be consolidated into a discrete lump which forms an undoubted tumor, and then the likeness to syphilomatous swelling is great.

Very small indeed is the amount written about gummata of the abdominal wall, but great is the literature concerning syphilis of the liver—that municipal destructor of the body. That syphilis finds a permanent home in the liver is a very old tale, and although we do not credit it with being the primary seat of syphilis, whence ulcers radiate even to the genitals, as Fallopius did, we have large, if vague, ideas of the importance of syphilitic hepatic disease.

These ideas mainly cluster round the congenital forms, and rightly so, as these largely predominate numerically. It is of the acquired form mainly that I wish to speak. Its fissures and scarrings, its lobulations and deformities are well known, and when seen at operation are of obvious origin, as are the cicatrices left by gummata; but gummata, which have not been absorbed when small, as usually obtains, and have attained large size, will give signs before and during operation which are very confusing. The following is a good instance of how mistaken a surgeon may be. A gentleman of the age of 41 (a likely age for gumma of the liver), leading a healthy country life in a district where hydatids are common, consulted me about marked loss of health, strength, and weight, and a want of symmetry in his ribs. He had noticed for some months that his right side was decidedly and increasingly more prominent than his left, and that exertion was apt to cause pain over this enlarged region. The

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rounded smooth swelling to be easily felt below the ribs, the increase of the liver dulness upwards, and its continuity with the evident tumor, decided me to tell the patient that he was suffering from hydatid of the liver. The wasting and the pain on exercise were factors that did not fall well into line in the diagnosis; but I certainly have seen cases of hydatid disease with as much and more pain, and some with wasting, though these last are more likely to be cases of multiple omental cysts. Operation was decided on—much more, I admit, to remove the supposed hydatid than the lingering doubts which existed at the back of my mind. Incision showed a large yellowish-white shiny mass, which even then might be taken for hydatid. However, closer inspection showed that small radiating fibres passed to the liver in many directions from the main mass. The tumor felt solidity itself, and was not quite as well defined as a hydatid adventitia is. A trocar inserted in several places discovered no fluid, and gave no impression of a cavity. The hole made by its insertion did not alter on its withdrawal, and the conclusion was soon come to that it was a very large gumma.

Gummata, like hydatids, tend to seek the surface of the liver; and very large ones must, perforce, abut on the periphery. Both prefer the anterior surface rather than the region of the porta. As the gumma reaches the surface a perihepatitis occurs, and this, of course, means pain, which may radiate, as in other hepatic conditions, to the shoulder tip, by way of the phrenic, or through to the back by the splanchnic route, but which is most likely to be present in the diseased area and its immediate surroundings. There is no doubt that pain is one of the main symptoms in late syphilitic disease of the liver.

Gummata of small and moderate size seem generally to be absorbed even without the influence of drugs, leaving only a scar at their former site; but one of the size obtaining in the present case acts as a malignant tumor: its morbid metabolism poisons by its products, and it is my opinion that this patient would have died in a very few months from absorption of the juices of this vast collection of loosely organised cells. As it was, iodide and mercury were given with the happiest results, local and general, and during his convalescence the patient remembered what he had previously denied—the existence of chancre, acquired some five years before. It may be noted that a longer period than this, as a rule, elapses before the occurrence of gumma in the liver.

Really large gummata—such as the one present in this case—must be rare conditions; but small gummata which cause no symptoms, or only slight perihepatitic pains may be, and probably are, common, and we know of their existence only by cicatrices found *post-mortem*, or, peradventure, at an operation. Part of the surgeon's *metier* in an exploratory operation in the vicinity of the liver is undoubtedly to feel the space between the liver and diaphragm for adhesions or anything that may break the smooth hepatic contour; but he should use more than his sense of touch: he should inspect with his eyes the surface of the liver, searching for any scarring or markings which may not be raised above or depressed below the surface, but which, nevertheless, are highly suggestive of syphilis. These cicatrices, clefts, fissures, and furrows, markings and lines, are well known as the results of liver syphilis; but I think that attention has not been sufficiently directed to striæ much more delicate than these. They are of a pale, transparent, bluish tint, looking very much as if the liver surface had been traced over with a small paintbrush dipped in weak nitrate of silver solution. They are not dense enough to cause any inequality of the surface, either as a ridge or a contrasting depression, and they meander towards the liver edge as rivers in a map to the land's edge. For some years now I always look for these markings in operations, and I have

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learnt to associate them with syphilis. In cases where syphilis was suspected I have even added an inch or so to my incision (chary as I am of unnecessary length in it) in order to be able to inspect the liver for these markings. They have to be looked for; but I believe, when found, they are good criteria that the patient is a syphilitic. Again, in syphilitic cases I have several times seen small calcareous masses in the centre of starred cicatrices. The proportion of importance allowable to syphilis as regards the formation of adhesions between the liver and the diaphragm is difficult to allocate, and involves a consideration of the causal agencies of perihepatitis; but my mind runs syphiliswards when I find adhesions without obvious cause in the dome of the diaphragm connecting it with the liver.

In the case just related there was no question of the tumor being a dilated gall-bladder; but a gumma has been mistaken for it, and of course the likeness to secondary malignant disease of the liver is considerable. Primary tumors of the liver are rare, rarer than gummata, so that the possibility of a tumor being a gumma, which is easily removed by medicine, must be taken into earnest consideration before a large and risky operation is attempted in order to remove the growth.

The presence of irritation as localising the deposition of gummatous material is interestingly shown in cases where a growing hydatid adventitia is surrounded by an atmosphere of syphilomatous tissue, and is itself similarly infiltrated. The rubbing of the anterior surface of the liver against the ribs may, when the surface is once roughened, constitute by its continuity a traumatism, and account for the preference gummata have for the anterior aspect. Syphilis is apt to exhibit itself in the terminal portions of an artery, a habit well seen in the branching seaweed-like markings on the inner aspect of the cranium, and this terminal endarteritis may furnish in the hepatic artery the initial roughness which is rubbed into actual gumma by the ribs.

Of those strange cases where the liver is covered by a thick and dense envelope of glistening white fibrous nature, I cannot speak with any definite knowledge. One would think these cases, whether only part of a general chronic proliferative peritonitis, or poliorromenitis, or separate hepatic conditions, were likely to be of a syphilitic nature; but I cannot say whether syphilis is the cause, or even one of the causes, of them, or of the somewhat similar condition in the thorax, viz., chronic indurative mediastinitis, or of a combination of these conditions, which is dignified by the name of chronic multiple serositis. There are, however, two other strange conditions—one involving both thorax and abdomen, undoubtedly syphilitic of the acquired form; the other purely abdominal and, I believe, syphilitic of the congenital form.

Taking the former, we find present a very large area which is blended together into an apparently solid mass, dull on percussion, callous in nature, largely destitute of function, and especially wanting in the movement naturally associated with the parts. A heavy dense cloud of syphilitic tissue has settled down over the parts, entirely and impartially obliterating the salience of the anatomy. Thus the lower half of the lung, the diaphragm, the intercostal muscles, the spleen, the parietes of the abdomen, and any organs near may be obscured and enveloped in this mass. Similar cases of less extent have been operated on as sarcoma, Paquelin's cautery taking the place of the knife, or a hopeless prognosis has been given, and the paralysing fog of syphilis allowed to spread. Here is a case, the recital of which is typical. A. B., a previously strong man, *æt.* 39, complained of severe pains over most of his left side, with much shortness of breath on even slight exertion; but his cough was what bothered him most, for it racked him continuously. There was no accompaniment of expectoration. He was much wasted, very anæmic, and looked

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ill. The signs of disease were vested in the painful region, which was all very solid on percussion. Air did not enter the lower half of the left lung. There were no adventitious sounds. The fluorescent screen gave a most interesting, as well as a most unusual, picture. The transradiant area below the diaphragm on the left side was replaced by dense shadow, which extended upwards through the diaphragm far into the lung. While the centre of this was very dark, the edge of the shadow was not sharply defiend, for dark streamers radiated in all directions. The diagnosis lay between huge syphilitic gumma and malignant disease. The exploring needle helped to decide, for its passage in several directions through the part was accomplished without the loss of even a drop of blood, which was very much against the growth being sarcoma, and a beautiful exemplification of the bloodless nature of the central parts of a gumma. The administration of full doses of iodide, in addition to mercurial inunction, rapidly restored the man to health, while the diminishing darkness viewed on the florescent screen afforded a most instructive spectacle, and as this syphilitic fog cleared off, the movements of the left diaphragm could again be seen.

In the second of the two conditions referred to, the syphilitic result had been too long present for drug medication to have much effect, and death ensued in the two cases I have seen. They were wonderfully alike, both young women under 25; in both ascites was present in large amount, and in both the superficial abdominal veins and the veins in the flank were much enlarged. One had jaundice. Both were tapped for ascites several times, and on the abdomen filling up again I was asked to explore. This exploration resulted the strangest palpation I have experienced. There was a large amount of fluid, and in this the exploring fingers felt the liver and its pedicle and the spleen as if they were plaster models, so firm and unyielding were they. The edge of the liver was rounded, the gall-bladder partook of the general hardness, and the liver was of a dark purple color, with lighter mottlings and streaks. There was evidently nothing to be done in an operative way; and a *post-mortem* examination some two months after showed in both cases much narrowing of the inferior vena cava, the walls of the portal vein were densely thickened and its lumen partially occluded by old blood-clot. It was decided by the pathologist that syphilis was responsible for the condition in either case. The disease had been so long in existence, and the damage done was so irreparable, that iodide and mercury administration could do no good. Wonderful as are the effects of these drugs in promoting the dispersal of syphilomata and gummatous infiltrations, a time in the history of these deposits must come when, if they have not softened, a degree of fibrosis is attained upon which iodide and mercury have but little power.

It is pleasing now to turn to a class of cases in which a correct diagnosis, leading to a correct treatment, will ensure a speedy remission of symptoms. While syphilis of the liver has been exhaustively treated of by many writers, the manifestations of the disease in the gall-bladder and gall-ducts have had little said of them. It is many years since I became cognisant of what syphilis might do to a gall-bladder. The first revelation was in the case of a previously healthy young woman, now wasted, jaundiced, with pain over the gall-bladder region, and increased resistance there; the history extending over many months. The gall-bladder at operation was seen to be studded all over with what were apparently carcinomatous nodules, the cystic and common ducts were enlarged and hardened, and there were many adhesions in the portal fissure. Drainage of the gall-bladder was effected. In doing this I took the occasion to remove one of these raised hardened excrescences with a gristly feeling, and Dr. Mollison informed me that it was a gummatous mass. The gall-bladder shows beautifully how tissues along a definitely marked arterial

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tract may be picked out by syphilis, which leaves the neighborhood free at all events of macroscopical disease. Thus evidences of syphilis may be obviously scattered along the cystic artery and its branches, while adjoining tissues owning another arterial supply show no manifestations at all. Here is quite another form of syphilis of the gall-bladder, in which other parts were implicated. A lady, *æt.* 39, began to feel epigastric pain and discomfort about a year before I was asked to operate on her. Her illness gradually evolved into one simulating very closely gall-stone in the common duct. The attacks of pain were typical; her jaundice, which was after a time always present, became much deeper during the later stages of the attack, which lasted several days. She would then make a partial recovery, which was again terminated by an attack. The pain was very great, and was relieved to some extent by vomiting. During the period of comparative freedom from pain she had tenderness in the epigastrium and over the gall-bladder, and once her medical attendant believed that he could feel a distended gall-bladder. She had lost much weight, and was evidently losing ground after each attack. On opening the abdomen I found a yellowish-white, much thickened gall-bladder fundus uncovered by the liver edge. It was unusually firmly connected with the liver. The thickening extended to the cystic duct and the common duct, in which, however, no stone could be felt. There were a good many adhesions about the portal fissure. The ducts were hard and dense to the feel, like a lead pencil. Cholecystotomy was performed, and a small portion of gall-bladder wall was removed for the mircoscope, by means of which Dr. Mollison found marked evidences of endarteritis. Bile percolated slowly through the wound for a fortnight. I could not help remarking that the changes in the gall-bladder were mainly in the outer and middle coats and not in the mucous membrane. The gall-bladder was not dirty in its inside, as at first inspection would have made one expect. There were no stones or débris at all. During the later stages of the operation I came to the expressed conclusion that the case was syphilitic. She improved much with mercurial inunction and iodide, getting fat and well, although her jaundice took a long time to pass off. Now comes the interesting part of her history. On leaving the care of her own medical man she went to a distant part of the State, and gave up her treatment after about six weeks. Gradually the jaundice came back, the attacks of pain reinstated themselves with much regularity every three weeks, and in three months' time she was nearly as bad as ever. She then came under my care in Melbourne, and a vigorous rubbing in of ung. hydrargyri twice a day with large doses of pot. iod. by the rectum have restored her to apparent health. I have spoken about the duct feeling like a portion of lead pencil; it is in my mind that this is a distinctive syphilitic phenomenon. I have met with it in several cases which I believe to be syphilitic, and which were improved, and seemingly cured, by antisyphilitic treatment; but as I had no microscopical confirmation of their nature, I do not wish to press their acceptance. It is difficult to be sure how jaundice is brought about in such a case as I describe; it may be due to one or more of the following conditions—gumma pressing on and involving the common duct, adhesions kinking the common duct, syphilitic infiltration and hardening of the head of the pancreas, syphilitic infiltration or ulceration and contraction inside the common ducts, similar conditions in the intra-hepatic ducts; cicatricial deformity of the liver may produce such dragging on the duct that its lumen may be insufficient for the passage of bile; and, lastly, syphilitic adenitis in the portal fissure may furnish the requisite pressure on the duct. The jaundice of the secondary stage of syphilis, which, I take it, is commoner than this tertiary jaundice, and which is seen concomitantly with the roseolous skin, is also difficult of explanation, though, we can here add, a specific cholangitis and a condyloma at the papilla to the

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other possibilities of production. I have also seen jaundice in the cases of enveloping gumma, where many structures are involved, and here pressure of the gummatous material outside the ducts, intra and extra hepatic, probably accounts for the condition. These latter cases seem commoner from my own experience, and what very little of literature there is, than syphilitic manifestations confined to the gall-bladder, ducts, and portal fissure. Jaundice and syphilis are not linked ideas in one's mind as jaundice and cholangitis, or jaundice and stone, jaundice and cancer of the head of the pancreas; but certainly a due association should exist, not because jaundice in tertiary syphilis is common, but because if the cause is not recognised and treated the patient will die; whereas the exhibition of mercury and iodide will certainly relieve and apparently cure, if used before the gummata have formed too dense a fibrous capsule and the infiltrations have resulted in too permanent a fibrosis; and I believe both these pathological terminations are reached only after a long period. Even the jaundice of secondary syphilis has dire danger if undiagnosed, as its tendency is to persist for a long time, and a very long continuance in some cases is associated with acute atrophy of the liver.

I have left in Melbourne a patient exhibiting yet another variety of gall-bladder syphilis. She is a middle-aged lady, who has been wasting much and departing from health for many months. Four weeks ago she noticed a swelling in the right lower abdomen. It was not very painful or tender, but steadily enlarged. When I saw it, carcinoma of the cæcum seemed the most likely diagnosis. The lump was hard, nodular, and very movable, laterally and upwards. She passed a good deal of blood and mucus at times, and had much constipation. Incision showed a deformed liver of the saddle-flap variety, with, in addition, a large flattened Reidel's lobe, at the extremity of which was a much distended yellowish-white gall-bladder. This contained more than 2 ounces of clear mucus; its walls were fleshy and very thick, even to half an inch or more, the mucous membrane participating but very slightly in the thickening. The wall was of the same yellowish whiteness as the surface all through. Encysted in the neck was a large gall-stone. The liver being well dislocated, I could satisfy myself that the ducts were macroscopically healthy and contained no other gall-stones. The deformity of the liver was largely accounted for by the presence of fibrous trabeculæ meandering through it in many directions, giving the appearance of an early stage of botryoid liver. The gall-bladder was removed, and made an interesting specimen, which left doubts in one's mind as to whether the great mural thickening was due to ordinary chronic inflammation, carcinoma, or syphilis. It may be remarked that the greatest depth of wall was not where the gall-stone reposed. Dr. Mollison is certain of the syphilitic nature of the specimen, and the patient is doing well on the usual medication. There can be no doubt, I think, of the propriety of classing syphilis as one of the causes of gall-bladder enlargement.

It may be permitted me to interpolate here the mention of an excellent example of the well-known "botryoid" liver, which is the untreated outcome of gumma and gummatous infiltration of the liver, which, when affecting the left lobe, may give rise to very remarkable epigastric tumor or tumors, and if the right lobe is mainly implicated, masses simulating a gall-bladder stuffed with stones or cancerous nodes. The patient, a sea captain, *æt.* 39, with a syphilitic history, complained of severe pain in the epigastrium and vomiting. He was not jaundiced, but of a remarkable earthy color. A rough irregular swelling could be felt below the ensiform cartilage, and the palpable edge of the right lobe was of a similar character. Any indiscretion in diet, especially alcoholic excess, brought him nearly to death's door with hæmatemesis and prostration. In his fatal attack the question of opening his stomach and

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getting at the ulcer, which Dr. Maudsley and I predicated as the most likely source of the hæmorrhage, obtruded itself. However, he sank so rapidly that operation was not permissible, and an autopsy failed to show any ulcer in the stomach, but the gastric and œsophageal veins from which the fatal bleeding had come were very large indeed. The liver was much deformed all over, and its advanced pathology seemed hardly compatible with the periods of fairly good health which the patient enjoyed when he took iodide and care of his diet.

In trying to pursue the somewhat systematic scheme which I suggested at starting, we may now consider stomach syphilis; and some of its manifestations are truly marvellous. If a man who knew the appearances of ordinary syphilis in other parts of the body were to imagine for himself the most remarkable forms he could picture for gastric syphilis, "the brightest heaven of his invention" would probably contain no more extraordinary tumor than that which occurs in the gastric wall in some rare cases of syphilis. I do not refer to those cases where parietes, gall-bladder, pylorus, and other structures are enveloped in a cloud of gummatous material; such a case I have seen, and they fall into line with cases I have already described, differing only in taking in the stomach as well as other parts. In the instances I wish to dilate upon a large definite solid new growth occurs in the stomach wall; it does not lose in density from its centre to its periphery; it does not gradually fade away into normal tissue, but it has its solid bosses and firm partially detached masses, with as clear-cut edges as the carcinoma which it so strongly resembles. It is a separate, distinct tumor, which can be picked up between the fingers—a performance impossible in connection with the widely diffused gummatous cloud wrapping together several separate tissues. It is very like cancer or sarcoma to look at, and, with the exploring finger only, I think a differentiation cannot be made. It is this strong physical likeness to malignant disease, quite apart from similitude in symptoms, which renders the surgeon cautious to an advanced degree. The following case is a handsome example:—

Mrs. W., *æt.* 36, had enjoyed good health until two years before she consulted me. At this time, gradually, began dyspeptic symptoms and, concomitantly, she lost weight, strength, and her robust appearance. There was no obvious cause for all this, for she was and had been leading a healthy country life. This downward course was slow, until about three months before I saw her, when she began to have pain and much tenderness in the region just below the eighth costal cartilage, on the left side, and here a lump was soon to be felt. Her health now deteriorated rapidly; she became very anæmic and distressed indeed; she could hardly walk. Her pain was continuous, though worse at night when she lay down. She could scarcely bear her clothes over the tumor, so tender was it. A hurried examination showed that the abdominal wall moved fairly well, except in the region where the pain was. As she was very wasted the rib margins stood out in strong relief, and below the left one a distinct tumor was visible and palpable. It was irregular in shape and gave somewhat the sensation of an inflammatory swelling, but no fluctuation could be felt in it. Respiration did not affect its position; it was not movable within the abdomen, and attempts at moving it caused intense pain. No glandular enlargement was noticeable anywhere. The patient's temperature was raised to 101° Fah., but she had had no rigors, and there was no redness of the skin over the tumor. She had no vomiting, and it seemed quite inadmissible to obtain stomach contents by tube. It was evident that operation was necessary at once, the working hypothesis being that the symptoms were due to a perigastric abscess with very thick walls. Operation was begun with an incision cautiously made over the

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swelling; as no pus was met with, the cut was enlarged, and the peritoneum opened to a small extent with some difficulty, as it was adherent to an underlying mass, which was explored with a hypodermic syringe without striking pus or a cavity. The sensation derived from the insertion of the needle was not suggestive of carcinoma, but rather of inflammatory tissue. It felt very much as if the needle had been pushed into a hard apple, and, to continue the simile, the hole left was such as would remain in an apple. The peritoneal cavity was now demonstrated above and below the adhesion, and it was noticed that the parietal membrane was altered, being thickened and injected. The investigating finger now met with a large mass evidently situated in the wall of the stomach. The tumor was as big as the palm of the hand, with processes issuing in several directions, but there was no attempt on the part of the growth to travel along the lesser curvature. The circumferential lymphatic glands were enlarged, reddish, and fleshy. With the eyes shut, the growth, which could now be moved about, felt very much like a carcinoma indeed, but it did not look altogether like one, especially about the part which had been adherent. It seemed too fleshy, not cicatricial enough, for chronic cancer; there was not the dull grey opaque white of carcinoma, but a semi-translucent, almost opalescent, look about it. For all this, the idea of carcinoma would come first to one's mind on seeing the tumor as well as feeling it. The condition of the posterior wall of the stomach was now investigated, and while opening the lesser cavity a clue to the nature of the case was found in the presence of white shining glistening bands of fibrous adhesions scattered in the great omentum quite irregularly and without obvious causation, there being no adherence of the omentum to the parietes or the stomach. One of these bands was circular and surrounded a hiatus in the omentum, having streamers issuing from it in several directions. There were also many linear strands of dense fibrous tissue mingled in the omentum which evidently had no use in limiting inflammation or extravasation from the stomach, and which appeared of greater age than the tumor itself.

Nothing unusual was found on the posterior surface of the stomach. These omental appearances decided me against any operation, so the abdomen was closed after the removal of a small piece of the tumor for Dr. Mollison, who assured me he had never seen endarteritic changes more easily guaranteed as syphilitic than those in the specimen. In five weeks the patient, under appropriate treatment, had gained health, strength, and good looks, and lost her tumor as far as external palpation could recognise. This gives us examples of two forms of syphilis in the stomach; a third is well exemplified in the following cases:—1. G.C., male, *æt.* 37, had been ill more or less for two years with some gastric trouble, diagnosed as gastric or duodenal ulcer. The prominent symptom when the case first came under my notice was persistent vomiting, which allowed of no food being retained. Food caused immediate pain in the epigastrium, which was speedily relieved by vomiting. Hæmatemesis had occurred twice in the last few days, once amounting, according to the patient, to the vomiting of a pint of blood. An examination of the patient showed him to be of a peculiar blanched complexion. His abdomen showed fulness in its upper half, and peristaltic waves could be seen passing from left to right across his dilated stomach. No tumor could be felt anywhere, and no superficial enlarged glands were present. There was a tender spot in the epigastrium to the right of the middle line, and firm pressure here caused great pain. Operation was decided on, and revealed a lump formed by a much thickened and stenosed pylorus and marked gastric dilatation. A gastrectomy was almost decided on, but an examination of the lymphatic glands showed them to be large, fleshy, reddened, and succulent, and I contented myself with removing some of these for examination, having suspicions

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of syphilis. The glands showed no signs of malignant structure, only inflammatory changes. The arteries in many places had very thick walls, and in some instances had become thrombosed. Here, again, the tumor had a semi-translucency almost like that of the pudding known as blanc-mange. A course of mercury and iodide improved the patient very much; he gained weight, lost his pain and no more blood, but his gastric dilatation did not go down, and three months after I again opened his abdomen, finding the tumor very decidedly smaller and the glands more normal. Instead of performing gastro-jejunostomy, I chose pyloroplasty. The former would, I think now, have been the better surgery. The pylorus proved to be very thick, and there was the scar of an ulcer inside. The operation was difficult, but a good-sized lumen eventuated. The patient at present, three years after the operation, is in very fair health, but has to take great care as to what he eats, and is always benefited by an antisyphilitic course of medicine. I never see him but I am thankful I did not perform an unnecessary gastrectomy, and wish I had done the more appropriate gastro-jejunostomy.

Here, again, no trial of the acidity of the stomach contents seemed justified. The amount of blood lost was remarkable from an ulcer undoubtedly syphilitic.

The second and very similar case is not so pleasant for me to relate. It was that of a man *æt.* 56, whom I saw in February of this year. He complained principally of wasting and vomiting. Symptoms began about six months ago, in which period he has lost three stones in weight. No severe pain at any time has attacked him, but he has a hot sensation after food, and there is some tenderness in the epigastrium, where a sense of resistance is present but no palpable tumor. His stomach is much dilated, as proved by CO_2 distension, and by the fact that he vomits more than he takes, and observes in the vomit food taken several days before. Chemical analysis of the vomit fails to show the presence of free HCl. Operation disclosed a pyloric growth apparently carcinomatous and suitable for removal. The anæsthetist was anxious that no time should be wasted in deciding as to procedure, and I performed a partial gastrectomy. The specimen proved to be a syphiloma with much fibrosis and cicatricial contraction. Five days after the patient died of pneumonia, much to my regret, my solace being that there was no leakage either at the closed end of the stomach or at the junction on its posterior surface.

It seems that a large syphilitic infiltration mass may appear at the pylorus; this may correspond closely with the ulcer inside; or the infiltration may overlap the ulcer much in all directions; or there may be a syphilomatous tumor and no ulceration; or an ulcer presumably syphilitic may occur with a minimum of infiltration. The difficulties surrounding the diagnosis of syphilis show up here very strongly. A neighboring gland or a piece of the actual tumor may show microscopical changes in the shape of thickened arteries, endarteritic alterations, endothelial irritation, and thrombosis, that may safely be ascribed to syphilis, but with the ulcer these signs cannot be laid at the door of syphilis alone. A non-specific ulcer situated in a place much irritated by stomach contents and muscular spasm will show some of these same signs in parts at all events of surface, so that no certain judgment can be given. However, arteries in other parts of the body which evidence no obvious macroscopical disease may be thickened and exhibit the phenomena of endarteritis, and this would go a long way to prove the pyloric ulcer syphilitic; but we cannot very well in the living subject cut out a piece of liver in order that the microscope should decide whether the ulcer is syphilitic or not. Then, again, can a person who is syphilitic have an ordinary chronic ulcer which is not syphilitic?

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It is these things which make the discrepancies so great in the opinions of authorities on the subject. The analogy of ulceration of the leg helps us a little; a syphilitic is exposed to the same causes producing ulceration of the leg as a non-syphilitic, but in the former the syphilitic taint is an additional factor tending to lessen the resistance and perpetuate the ulceration. We all know how syphilis shows itself in waves of activity in the organism generally; how its natural history is illustrated by periods of obvious disease sandwiched between periods of apparently good health; how in tertiary syphilis, even, a man may have a good year and then a bad year, a good six months and then a bad six months; now these waves of disease manifestations have their counterpart in gastric and duodenal ulcerations due to syphilis. A strong opinion of the syphilitic nature of an ulcer at or near the pylorus can be given if evident healings have taken place in several areas, if patches of active cell growth can be seen in old fibroses; but to judge of this in the living subject during the course of an operation is very difficult, and however valuable these evidences of repair intermixed with active disease may be in the museum, they do not aid the surgeon much in the few minutes he has at his disposal.

High authority here helps us, as Professor Allen tells me that he is fairly certain that non-cancerous ulcers actually at the pylorus or immediately under cover of its valve on the duodenal side are, in his matured opinion, very generally syphilitic.

The therapeutic test is the best means of diagnosis, and this, too, is open to misconstruction on several counts. Syphilomata are more easily recognised as not being cancer the younger they are, *i.e.*, when they are fleshy, succulent, and inclined to show the opalescence I referred to. This opalescence is very different, I may remark, from the angry purplish appearance seen in a quick growing malignant adenoma. The more fibrotic they become, the more they are liable to be mistaken for carcinoma, and the similarity is very close in cases. The lymphatic glands differ in the two diseases, being softer, redder, fleshier in syphilis than in carcinoma, but it is only in well-defined varieties that a diagnosis could safely be made from glands without the microscope. However, what is almost as important is that a suspicion of syphilis may arise in the surgeon's mind from an inspection of the glands.

These pyloric syphilomata cause contraction without the presence of an ulcer, so that the dilatation of the stomach is no help to us. The surgeon has to make up his mind quickly. In cases where the signs make him suspicious even slightly of syphilis I feel sure he ought to restrain "the hands which have made him wise," and content himself with removing a gland close to the disease and perhaps a small piece of the tumor, closing the abdomen after a somewhat ignominious retreat.

If the pyloric lumen is evidently very small from cicatrisation, when felt by the invaginating little finger, a gastro-jejunostomy will be the proper procedure if the case is syphilitic, but the risk of gastrectomy when medicine or the much less severe operation of anastomosis will restore the patient to comfort is an awkward one to face.

Now is the time for the surgeon to remember how associated are the organs in this region; how the stomach duct, which the duodenum may be called, the liver duct and the pancreatic duct all meet at one place; how these organs and their appendages are supplied by a common trunk, the cœliac axis; and how inflammatory processes in one organ can easily spread to the others, the subsequent inflammation giving rise to much greater effects than in the original organ. Reminiscent of these things, a rapid investigation by the surgeon of the surrounding parts may afford him much help in coming to a decision.

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Markings of various sorts on the liver, patches of purposeless adhesions and stray fibrifications in the omentum, and possibly general fibrosis of the pancreas may put him on the right diagnostic path. However, in spite of every help—the history of the case, the look of the tumor, a general survey at operation, and microscopical assistance afterwards—it may be difficult to determine unmistakably. Such a case is at present under my care. I could not make up my mind at operation that the case was cancerous: Dr. Mollison found some endarteritis in the glands I removed, and no carcinomatous elements, but could not give a definite opinion. Fortunately, because of the great contraction of the pylorus, I had performed a gastro-jejunostomy, and a few days after started the patient on an antisyphilitic course of mercurial inunction and iodide by the rectum, with the happiest results. Even now, however, I cannot be quite sure of the syphilitic nature of the case, but I can be, and am, very thankful that I did not perform gastrectomy, which, judging from the shock present after the less severe operation, would have probably proved fatal. It is likely that a good percentage of supposed cancer cases, which live a long time after gastro-jejunostomy, are really syphilitic, the diversion of the gastric current and the mere operation itself having a very beneficial effect. In this last relation, what a number of cases have been reported where the abdomen has been opened to find a chaotic mass of adhesions too intricate to unravel, the surgeon has reluctantly closed the abdomen, and the patient has promptly lost his symptoms. All these, of course, cannot be claimed as due to syphilis, but a percentage, I feel pretty sure, will fall under the first class of intra-abdominal syphilis of which I spoke.

There is yet another connection of syphilis to be spoken of in gastric pathology. Carcinoma may be engrafted on a syphilitic base. This occurs at the entry and exit of the alimentary canal, and may not be uncommon in the stomach. The University Museum in Melbourne has more than one specimen, which, on the weighty dictum of Professor Allen, is of this nature. At the present time I have under my care a lady whose gastric tumor, on the assertion of Dr. Mollison's microscopy, participates in both diseases.

Sarcoma may enter the lists, and is to be recognised generally because of its discoid rather than spheroidal tumor, because of its tendency to avoid the lesser gastric curvature and to disregard the integrity of the duodenum; but while these characteristics serve to differentiate it from carcinoma, it may be hard to exclude syphilis when sarcoma is present. It is probably rarer than either carcinoma or syphilis, and here again we see how malignant disease may choose a syphilitic site upon which to develop, for we have in the Melbourne Museum a specimen of gastric sarcoma, with the major portion of the stomach, which I removed some two and a half years ago (the man being still alive and in good health), and which is declared by Professor Allen and Dr. Mollison to be sarcoma growing from a syphilitic foundation.

Of chronic hypertrophic gastritis due to syphilis it is difficult to speak clinically, though pathologically it is a definite entity.

These considerations enhance the already great responsibility laid upon the surgeon in gastric surgery. The indurations he has to differentiate are those of simple chronic ulcer, and simple fibrosis, of carcinoma, of sarcoma, of syphilis, or of combinations of these last. Is it any wonder that he sometimes makes a mistake in his choice of operation, when we consider the complexity of the evidence, and the short time in which he must arrive at his decision?

Much as I dislike reopening the abdomen after a short interval, I believe the best surgery will more often than in the past lead to such a course, fortified as it will be the second time by microscopical evidence.

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In spite of gastric ulcerations, fibroses, contractions, infiltrations, and actual tumor masses of gastric growth, we have not exhausted, I believe, the roll of macroscopic syphilis in this region.

Concrete lumps of syphilitic origin may occasionally be found in the cellular tissue behind and at the sides of the first and second parts of the duodenum, and by their presence and almost cartilaginous hardness produce jaundice. These are distinct from syphilis of the pancreas, which is said to be rare, Petersen having found it in only one case out of 88 p.m.'s on the subjects of tertiary syphilis. It seems likely that some of the cases of chronic pancreatitis forming tumor are due to syphilis and some of the fibrotic cases also, but I think we cannot speak with much certainty yet.

Syphilis of the small intestine I have seen in two forms, both of which tend towards narrowing of the lumen, but neither of which, I believe, has the extreme constriction which I have seen in sigmoid ulceration due to syphilis. Even as gummatous deposit at the pylorus may produce obstruction without ulceration, so the small intestine may be strictured by similar deposit without a corresponding ulcer inside.

Syphilitic ulceration of the small intestine is rare, but has often been described. We have a good example in the Melbourne University Museum, in which the ulcers are oblong, about three-quarters of an inch in length, situated at the fundus of the intestine, and presumably starting in Peyer's patches, Their bases are indolent and indurated, thickened and opaque, with much congestive engorgement. On the peritoneal surface the ulcers are represented by little filamentous tags and adhesions. These were diagnosed as syphilitic before microscopical examination, mainly because they would fit no other classification, though, of course, the induration of their bases was suggestive.

Of the second variety, which is very different, I have seen no description. The ulcers are small, run transversely to the intestinal lumen, exist in considerable numbers, and reveal their position on the peritoneal surface by silvery scars, very like linæ albicantes indeed. Each scar of itself would do but little to narrow the gut, but existing in large numbers, as in this case, they diminish the calibre very considerably, giving rise to symptoms of obstruction. On my return to Melbourne I expect to have to remove some 18 inches of intestine for this cause from a lady upon whom I have operated before, and who I know has this peculiar condition which much evidence permits me to certainly class as syphilitic.

These numerous ulcers from time to time give rise to blood in the stools with much mucus, and attacks of an obstructive character. It is strange how the terminal result in skin ulceration differs from what is seen as the final scar in internal ulcerations due to syphilis. The papery scar left eventually by extensive and long-continued cutaneous ulceration has little tendency to contract, in obvious contradistinction to the strongly marked disposition to cause stricture, even to total closure, which we see with such disastrous endings in syphilitic ulcerations of the trachea, of the bronchus, of œsophagus and pylorus, sigmoid flexure and rectum. These ulcerations of the small intestine which I have just described seem to occupy a position between the external and internal ulcerations of syphilis, leaving small thin silvery scars on the peritoneum, which, however, in their multiplicity undoubtedly lessen the calibre of the intestine.

As far as I can make out, the region of the cæcum, the ileo-cæcal valve, and the appendix is but little susceptible to syphilis—an exception in strong contrast with the frequent manifestations of tubercular disease.

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In spite of having had a number of appendices examined microscopically, I know of only three which can safely be classed as syphilitic. They were very chronic cases, and the specimens were densely fibroid and hard, with very little appendical lumen left.

It is an interesting speculation, upon which, however, I can throw no light, as to what part syphilis plays in obliterative appendicitis, and in those cases where the appendicitis has been of a chronic nature from the first. It is probably only one cause, and not a common one, of chronic inflammation of the appendix. Of course the appendix and cæcum and any part of the colon may be engaged in a large enveloping gumma, such as I have described earlier in this address, but more concrete syphilis affects but rarely the large bowel until we reach the sigmoid, when we find both ulceration and tumor-like formation not uncommon. Both may be associated with similar lesions in the rectum. I have seen stricture of the sigmoid due to girdle ulceration of the gut so tight as to be almost impermeable, and this with very little tendency to induration around.

Another case in which there was undoubted syphilitic ulceration healed by drug treatment developed marked obstructive symptoms, which were entirely relieved by the freeing and division of strong adhesions round the sigmoid, though the operation was started with the idea of resection.

Syphilis is undoubtedly a cause of peritoneal adhesions, even as traumatism produces them; but it is very difficult to assign the proper importance to either as a causal factor. Wherever there is, or has been, an ulcer or gumma, the nearest peritoneal surface may be the seat of adhesions; but also, I believe, the syphilitic poison can produce adhesions without ulceration or obvious gummatous deposit.

Remarkable are the developments of the disease we are discussing in the retro-peritoneum, and some of these are massive in their formation. There is no doubt that mesenteric and mesocolic glands enlarge in the secondary stage, in that mucous membrane disturbance which corresponds with the cutaneous efflorescence, but the size they attain is inconsiderable, and their diagnostic value and clinical importance are slight. In the tertiary stage, however, the retro-peritoneal glands generally may become transformed into very large tumor masses easily mistaken for sarcoma. Their stability is but slight, and in some instances mere inspection by operation is sufficient to induce their absorption, frequently to the astonishment of the surgeon who has given an unfavorable opinion. It is well worth while, in every case of supposed sarcoma of the retro-peritoneal glands, to remember the possibility of syphilis being the cause—a cause the effects of which are easily removed by simple operation and drugs. Not so tumefied, and with their cells not so loosely aggregated, however, are all these glands altered by syphilis; some are hard, even shotty, and but moderately enlarged, similar in physical aspect to the glandulæ concatenatæ when enlarged in the neck; and gradations occur, I believe, between these extremes. Both these enlargements may be associated with syphilitic disease of the rectum, which is itself often associated with marked pyogeny, and this may be largely responsible by toxicity, for the great size at times attained by the syphilitic glands, as well as for their succulent instability,

To my mind the surgical moral to be drawn from a consideration of retro-peritoneum tumors is that in no case should the diagnosis of sarcoma be considered final until operation has proved the absence of syphilis and hydatid, the former being a simulator of the softer sarcomata and the latter of the harder varieties.

Again, syphilis works marvellous mutations in the cellular tissues itself. It appears to be responsible for a strange wasting fibrication of the usually fat-

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aden sup-peritoneal tissue, and at the opposite extreme there may arise from the syphilitic poison a widespread homogeneous gelatinoid mass, in which the kidneys, great vessels and glands may be embedded as in glue. The same material filling the cavity of the sacrum and surrounding the nerves and vessels of the pelvis may easily convince the surgeon of the presence of inoperable cancer, as it may also in the mesometrium. In such cases, I think, the extraordinary results are largely due to the spread of toxic matter from a pyogenic infection of syphilitic disease of the rectum and sigmoid. Surgeons have long since realised the far-reaching effects of a mixed infection in tubercular disease, but I think have hardly yet appreciated its importance in modifying syphilitic manifestations.

Mine will not have been a failure in this presidential address if I have directed your attention to the fact that syphilis is not very rarely the cause of pathological conditions within the abdomen, and that these pathological conditions are easily confused with other commoner diseases, especially carcinoma.

When one has once grasped the idea of the role played by syphilis in abdominal pathology, so deceptive are the facts, so elusive are the differences between it and other chronic inflammatory conditions, that one is apt to see syphilis everywhere, to discover a bandit behind every bush; but what I have set down in this paper has been of my own knowledge, or of others whose judgment I trust, and I have discarded many cases which, syphilitic in my belief, have not been fortified in diagnosis by the microscope; thus I hope you will think that I have "taught myself that honorable stop, not to outsport discretion." Yet there float into one's mind the words of the dying Ricord: "They reproach me with having seen syphilis everywhere; I, on the contrary, reproach myself with not having recognised it often enough."

Though the study of syphilis—"the specific disease which stands almost alone in mystery and malignancy"—is one of the most fascinating mental exercises that we have in our profession, we cannot but fervently hope for the good of the race that the *Spirochæte pallida* of Schaudinn will prove to be the germ of the disease, and Metchnikoff and Roux are very hopeful of the parentage.

Until we have eliminated syphilis—and it has been in possession of the human race from times immemorial—so long will man be unable to come into his kingdom, his complete mastery over Nature, the *Regnum Hominis* which appeared to the mental eye of Bacon as a prophecy for the fulfilment of which modern science bids us to cheerfully hope.

THE OPERATIVE TREATMENT OF MOVABLE KIDNEY.

By WILLIAM S. COLLINS, M.B., LOND., M.R.C.S., Wellington, New Zealand.

In introducing the subject of operation on movable kidneys to you, I wish it to be understood that I do not consider an operation is called for in a large number of cases. It is surprising in how many people the kidneys are found movable, and yet in whom this moblity seems to cause very few symptoms. Movable kidneys are most common in women, and are coexistent very often with some disease of the genital organs. In determining whether in a given case an operation is likely to be attended by benefit, one

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has first of all to find out if the kidney is the real cause of the patient's sufferings. You have to find out whether the movable kidney is healthy, or the seat of carcinoma, tubercle, stone, or abscess. You have to eliminate the symptoms which may be caused by dyspepsia and constipation, appendical disease, gallstones; in women, by ovarian and uterine disease. I have frequently found movable kidneys in women who suffer from dysmenorrhœa, also in women who have had severe labors, and in those who have become thin and emaciated from having had children too frequently. Those cases where the kidney has been dislocated by injury are most likely to be benefited by operation. In highly neurotic women who have movable kidneys I should hesitate in advising operation, nor would I operate on women who are approaching the climacteric period. In most poorly nourished women I believe that an operation can be avoided by rest and improvement of the general health.

Seven years and a half ago I was consulted by a lady for pain mostly in the right side, nausea, vomiting, and general abdominal discomfort. She was thin and emaciated. She had had three children in seven years. Her symptoms were always relieved when she was lying down. Both kidneys were found movable, the right more than the left; the right kidney was, moreover, tender on manipulation. I explained to her that I believed the mobility of the kidneys had something to do with her symptoms, and stated also that if she would rest and be fed up she might possibly avoid the necessity for an operation. On making her lie flat in bed without a pillow, and by raising the end of the bed 14in., I found that it was much more difficult to feel the kidneys. I suggested that she should lie in bed for six weeks or two months with the end of the bed raised 14in., and be in the meantime fed up. This she consented to do. By appropriate feeding she improved in general health, and at the end of two months she was allowed to move about. Since then I have seen her occasionally, and there has been no return of her symptoms.

For many years I had been dissatisfied with the results of the operations for movable kidneys, and it was this case which first suggested to me the operation I now employ for fixing the kidney. It always appeared to me that by the operations usually performed the kidney was fixed in an abnormal position; that it was fixed opposite the incision in the abdominal wall (in the majority of the cases the kidney could be easily felt after the operation in the position in which it was fixed); that in this new position it was more liable to be affected by the movement of the muscles to which it was attached or adherent; that it was more liable to injury and exposure to cold; and that even though fixed, and though the symptoms which had been caused by its mobility had ceased to exist, yet in time it became enlarged, tender, and painful, and caused as great discomfort as it had previously done when it was movable. I have on three occasions operated on kidneys which had been fixed by the ordinary operation, and have separated the adhesions and refixed the kidney again in its natural position, and up to the present with good results. According to the statistics given in "The International Textbook of Surgery," in operations on movable kidney there are 22 per cent. of relapses, and in about 10 per cent. there is only some improvement. In all operations for movable kidneys I believe that it is necessary for success to replace the kidney as far as possible in its normal position, and it is with the view of accomplishing that object that I use the operation I am about to describe to you.

In the operation which I advocate the kidney is replaced as far as possible in its natural position, and when the operation is completed the lower pole of the kidney is above the level of the umbilicus. The patient, having been

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anæsthetised, is placed in the lateral position, the surgeon standing behind the patient. The length and position of the twelfth rib is carefully defined, and a point ½in. below the rib and 2½in. from spine is marked out.

A kidney-cushion or pillow is placed under the opposite flank of the patient. From the point marked out, an oblique incision is made downwards and forwards for about 4in. The skin and fascia are divided, the anterior fibres of the latissimus dorsi and the external and internal oblique muscles. If possible, division of the last dorsal nerve is to be avoided, owing to the pain which is caused after the operation, but usually it is impossible to prevent division of it. The lumbar fascia is cut through and the perirenal fat exposed, the fatty capsule is opened, and the kidney felt for and examined *in situ*. Should there be any suspicion of disease, the kidney should be delivered out of the wound (in order to do this more easily the wound may have to be enlarged), and examined carefully. The fatty capsule is now removed, the kidney capsule is nicked at the centre of its convex border, and divided on a director from end to end, and reflected to the extent of about ½in. on each side. The upper pole of the kidney is bared more freely from its capsule than the lower pole. A temporary ligature of silkworm-gut is passed through the substance of the kidney, and the kidney replaced in the abdomen. The object of this temporary ligature is to steady the kidney during the subsequent steps of the operation.

The fingers of the operator's right hand are passed up under the ribs into the position in which the kidney is to be fixed, in order to feel what space there is. The patient is now placed in an extreme Trendelenburg position, and immediately the kidney is seen to move upwards under the ribs into its natural position, unless prevented by traction on the temporary fixation ligature. It is now gently and without force pushed up as far as it will go by the fingers of the right hand of the operator being placed under the lower end of the kidney. A silkworm-gut ligature is passed through the lower end of the kidney, including reflected and attached capsule, and parenchyma of kidney, and both ends of suture are drawn out of the wound and held by forceps. A second silkworm-gut suture is passed through the extreme end of the kidney, and also drawn out of the wound and held in a similar manner. The temporary steadying ligature is now removed. Each end of the first suspending ligature is threaded on to a curved needle, the fingers of the operator's left hand are passed into the wound and underneath the tissues forming the upper part of the wound, the twelfth rib is felt, by the fingers the needle is guided and passed through the abdominal wall, emerging on the surface just below the last rib. Both ends of the ligature are in this way passed through and the ligatures are about 1½in. apart on the skin surface. The ends of the second suspending ligature are passed in the same way, emerging on the skin just below the last ones and in the same line. The second ligature is tied first over a piece of gauze, and then the first one. Both these ligatures must be tied without exerting any forcible traction on the kidney; the amount of traction necessary is only that which is necessary to approximate the lower end of the kidney to the abdominal wall. If the dorsal nerve has been divided and the cut ends can be found, they may be sutured together. The wound is closed with deep silkworm-gut sutures in the ordinary manner. The patient is removed to a bed, *the foot of which is raised* 14*in.*, and I consider this to be a most essential part of the treatment.

After the operation she is made to lie on her back, but with rather an inclination over towards the side upon which the operation has been performed. *The patient kept in this position with the end of the bed raised* 14*in. for three weeks.*

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The wound sutures are removed on the seventh day, and the suspending ligatures at the end of three weeks. If there has been much manipulation during the operation, it may be advisable to introduce a drainage-tube at the back part of the wound and leave it in for twenty-four hours. As a rule, however, this is not necessary.

The main features in the operation are the stripping of the capsule, the fixation of the kidney in its normal position while the patient is in the Trendelenburg position, the passing of the ligatures for the anchorage through the lower end of the kidney, and the keeping of the patient in such a position after the operation that there will be the least possible disturbance to the organ caused by coughing, vomiting, &c. The kidney by this method of operation is fixed in its normal position with very little injury to the kidney itself or disturbance of the surrounding parts, and it appears to me to be more satisfactory than fixing the organ exactly opposite the external wound or planting it on to a muscle.

On May 5th, 1905, at a meeting of the Wellington Division of the New Zealand Branch of the British Medical Association, I again read a description of the operation, and asked if any of the members had come across any of the patients on whom I had operated where the kidney had again become movable, and I was gratified to find that none of the members had seen any cases of failure.

Dr. Faulke stated that he had done some cases latterly, and the operation was a great success. He found that there was too much tension from two sutures, and so used three. In his later experience he found that there was no occasion to raise the end of the bed after the operation, and since discarding this position his cases had done equally well.

Since the above discussion I have heard of one case on which I operated over two years ago, in which the kidney had again quite recently become displaced, and on inquiry I found that it was in one of these cases which had been operated upon before by one of the usual methods, and where the kidney had become much enlarged and painful after the first operation.

STEREOGRAPHY.

By C. H. Souter, M.B.

There is nothing new in the subject of stereography. So many of us are amateur photographers nowadays that it is somewhat surprising that this well-known method is so seldom made use of. There is, however, an easily recognisable advantage in the binocular over the monocular photograph. I hope it may not be impertinent to give a very brief description of the manner of taking a stereographic picture, and the special points that are most important. It is necessary to take two photographs of the same object in such a manner that one photograph shall represent the object as seen by the right eye, and the other photograph shall represent it as seen by the left eye. For this purpose two cameras are used—fitted each with a lens of equal focal length to that of its fellow. For convenience the two cameras are made in one, and divided by a collapsable partition between the lenses. The plate-holder or dark slide may carry a half-plate or film of corresponding size. For correct work, and more especially in medical and surgical subjects, it is better, in fact essential, to have an adjustable front by which the lenses can be brought

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nearer or further away from one another at will, in order that, as far as possible, the same angle may be subtended for near as for distant objects. If your lenses are about 3½ inches apart, distant views will appear in bold relief; but if more than 2½ inches from centre to centre separates the lenses, exaggerated perspective will be seen in photographs of very near objects (say about one to two yards away). Thus the nearer the object the closer should be the lens centres to one another.

All the good makers supply stereographic outfits, and any half-plate camera can be converted into a stereographic camera by fitting on a Thornton-Pickard adjustable stereo time and instantaneous shutter, and procuring a pair of well-matched but not necessarily expensive lenses for it. All that remains to do is divide the camera inside with a piece of accordeon pleated black stuff sprung in between the front and back. The original half-plate holder or dark slide will do. I have brought this subject up to night, and draw your attention to certain characteristics of these pictures, in the hope that some day producers of medical and surgical works will use this means of illustrating. Authors and publishers are coming to depend more and more on photography as a means of illustration in surgery and medicine. It is right that this should be so, for although the old jibe that a man may "lie like a tombstone," might well be read nowadays "lie like a photograph," it remains true that, untampered with and properly interpreted, a photograph is an actually scientific record of what was there to be seen. Unfortunately it is easy to tamper with photographs. The usual portrait photograph is so much faked or "touched up" that the original often only knows it for his counterfeit presentment by taking the artist's word for it. I do not think I can say a greater thing in favor of the systematic use of the stereograph than this—you cannot retouch or otherwise tamper with a stereographic picture without the fact being plainly evident to the most casual observer. As I consider this a really most important fact regarding stereoscopy not only from an illustrator's point of view, but from that of the medical jurist, I will detain you a moment to see how this may be clearly shown. It may be foreseen from the nature of things. If you imagine two pictures or views—one seen by the right and one by the left eye—the two visual axes converging at the centre of a certain object say a hundred yards away, it will be plain that no two points in the intervening space that are passed through by the visual axial line of one eye will also be passed through by that of the other. Thus in any plane at right angles to that of the mean line of sight the right and left lines of sight will never pass through corresponding points in the pictures. They will do so at the plane of the object on which they are focussed. On examining a pair of stereoscopic prints carefully, it will be seen that the central focal points of the pictures correspond, but that the relations of all other points of the pictures differ in the two pictures.

Another way of putting this is to say that if one look at a clear glass ball held a yard away from the face, the centre of the ball will be the centre of the picture for either eye, but the right eye will see further round the right side and the left eye further round the left side. This would also be true of a stereograph of such a ball. Now if one take a single photograph and make some alteration in it by retouching, it may be so skilfully done that close scrutiny will fail to detect the alteration. If this be tried with a stereographic pair of pictures it will fail most completely from two causes. Firstly, it is impossible to retouch one picture so absolutely similarly to the other as to make even a mechanical correspondence between the two. Secondly, even could this be done, it is obvious that as the point of view of each object in either picture differs some degrees (especially for close objects), the retouching would have to correspond with this difference in the respective

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pictures, a physical impossibility. The practical result of all attempts to "retouch," or "touch out," or "spot," or make any adventitious marks on a stereoscopic pair of photographs is that these marks, seen through the stereoscope, appear to stand forward in a plane of their own, not subtending any particular angle of the eye, and having no part in the composition of the picture as focussed by both eyes. This can easily be demonstrated. Take one of the slides before you and endeavor with a pen or pencil to make a mark on both of the pictures, so that it shall produce a concrete effect with binocular vision through the stereograph: you will find it impossible.

This fact I consider of much medicolegal importance.

The other and much more obvious virtue of the stereoscopic process for illustrating surgical and medical work is two-fold. The fact that there is perspective and relative distance, and even *actual* distance given to the component parts of the picture causes the stereograph to furnish a vastly more accurate impression to the eye than the most brilliant and detailed single photograph can, and it further follows that inferior, over or under exposed, and badly printed photographs, which would be useless for illustrative purposes used singly, are, if stereographic, still capable of showing a great deal of useful detail.

There is but one thing that stands against the use of stereographic photography for printed works. It is the misfortune that as all photographs are reproduced by copying through a mesh—finer or coarser according to the class of work—all "process work," as it is called, shows the copies in stipple effects, with no true gradations from light to shade—only a series of large or small points of light. For this reason—and, as I have already shown, because no two points can be said to accurately correspond except the central focal points of the pair of pictures—the "process" copy of a stereographic picture looks like a *real* scene or object viewed through a dense wire screen (which indeed it really is), and this takes off much of the brilliancy, important detail, and realistic effect.

Nevertheless, a surgical or other work might well be supplied with a wallet of original photographs, numbered to correspond with certain diagrams or pages.

Fancy a few dozen good stereographs sold with books like Ward's Osteology, Grey's Anatomy, Ellis, Treves, Hart and Barbour, Guy and Ferrier, or Taylor.

I think half the bald descriptions in students' text-books on surgery, midwifery, anatomy, botany, zoology, medical law, dermatology, and a good many other subjects could well be done without, or at least cut down by half, and replaced by stereoscopic pictures which, in suitable subjects, give a concrete, realistic, and objective impression to be gained only in one other way, viz., by seeing the real thing itself. As to the possible objection of expense, publishers should be able to supply good stereoscopic photographs from negatives supplied by authors at sixpence a dozen.

THE OPERATIVE TREATMENT OF THE ENLARGED PROSTATE.

By H. L. Maitland, M.B., M.Ch.

In a paper I read before the New South Wales branch of the British Medical Association, which was published in the July (1903) number of the *Gazette*, I expressed my ideas as to the method of performance and value of perineal prostatectomy. That paper was based on a series of five cases. A few months later I published another series of five cases. The paper which

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I bring before you to-day is based on the experience of forty-four cases operated upon for the relief of symptoms due to enlarged prostate. The series consists of twenty-five cases enucleated by the perineal route; six cases enucleated by the suprapubic route; eleven cases of perineal prostatotomy; two cases of supra prostatotomy.

The series of cases extends over a period of five years. The first case of perineal prostatectomy I did in 1900.

The so-called senile enlargement of the prostate is due to a fibro-adenomatous growth, which is practically an overgrowth of the normal glandular tissue. Clinically the enlargement may be divided into a soft and a hard variety. In the soft variety the adenomatous tissue predominates, and in the hard variety the fibrous tissue.

The soft or adenomatous variety usually forms outgrowths of a larger or smaller size, which project into the bladder or towards the rectum .

The hard or fibrous variety is very small, hard, and tough, forming a lip rising above the level of the urethral floor, then dropping in a perpendicular behind, to form the anterior wall of a bladder pouch.

There are many gradations between the two varieties, and they merge into one another, but it is important that there should be a distinct recognition of these two varieties, because I maintain that their treatment is absolutely different.

I will first of all discuss the operative treatment of the adenomatous or soft variety; and this is briefly *enucleation*.

There are two routes by which the prostate may be enucleated: 1st, the suprapubic; 2nd, the perineal. Which of the two routes is the better? For many reasons I consider the perineal is the better.

(1) From an anatomical standpoint every fact is in favor of the perineal and against the suprapubic. When one considers the situation of the prostate—that it lies outside and below the bladder in the perineum, bounded below by the triangular ligament—surely it is evident that the prostate is more easily reached from the perineum than from in front, through the abdominal wall and bladder. And it is a reasonable surgical axiom that in operating on any organ the most direct route should be taken, provided important structures can be avoided. And the approach by the perineal route is through comparatively unimportant structures.

(2) *Drainage is more satisfactory.* We are accustomed to get perfect drainage of the bladder by a suprapubic opening. This I freely admit, because the superior wall of the bladder is kept opposed to the inferior by intra-abdominal pressure, and the urine does not collect; but when the prostate has been enucleated, you have quite another condition existing, viz., a cavity which is outside and below the bladder. It is not possible to drain this cavity suprapubically. By the perineal route you get drainage which is perfect. It is in the direction of gravity, and the drainage tract is a short one.

This question of drainage is an all-important one, and in the majority of cases in which I have enucleated the prostate through Freyer's suprapubic opening I have employed perineal drainage.

(3) There is less danger from sepsis, because there is better drainage.

(4) There is less shock, because the perineal is a less severe operation.

(5) There is less damage to the bladder: when the suprapubic route is employed the bladder is opened above and below. In the perineal route it is possible not to open the bladder at all, but I always incise the bladder wall as a matter of choice.

(6) The perineal operation allows the prostate to come more into view during the enucleation, that is if Kocker's transverse incision is used, and the prostate enucleated from outside the urethra.

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The suprapubic operation I admit allows of a visual examination of the visceral configuration of the prostate; but the perineal allows digital exploration, which is just as good in these cases.

(7) The patient can early be placed in the semi-recumbent position, perfect drainage taking place when in this position. This is of some importance, when it is remembered we are dealing with the aged.

(8) The shortening of convalescence. The aged patient can sooner leave his bed if he has no abdominal wound.

(9) The prostatic urethra is better preserved. Both lobes cannot be completely removed without some laceration of the urethra. The ejaculatory ducts, as well as the prostatic ducts, open into the floor of the urethra on each side of the veru-montanum, and, as they are torn through, the floor of the urethra must suffer. I am perfectly well aware that many cases have now been recorded where the whole prostatic urethra has been removed, and the urinary function re-established.

One of Reginald Harrison's cases early became strictured, and I have seen the same thing occur.

(10) There is less hæmorrhage: because I think it is better controlled from the perineum than suprapubically.

(11) There is less danger of secondary hæmorrhage. In two cases removed suprapubically, in which I did not drain by the perineum, most alarming second hæmorrhage occurred, the explanation being that there is more danger of sepsis from inefficient drainage in the suprapubic operation.

The main difficulty in the perineal operation is that of bringing the prostate down sufficiently low in the perineum so as to get round it with the finger.

Various suggestions have been made to overcome this difficulty, viz.:—

(I.) Pressure from above, through a suprapubic wound, or through Retzius space.

(II.) Traction downwards by means of a dilatable bag introduced through the prostatic urethra into the bladder.

(III.) By traction downwards made by fingers of left hand in rectum.

(IV.) By introducing the left index finger through the prostatic urethra, and hooking the prostate down while enucleation is done extra urethrally by the right index.

(V.) By such an instrument as Gouley's or Young's depressor.

(VI.) By traction downwards with a volsella forceps.

DESCRIPTION OF THE OPERATION OF PERINEAL PROSTATECTOMY.

The steps of the operation which should bear Gouley's name are as follows:—The patient, placed in the lithotomy position, has a catheter introduced, and the bladder well irrigated. Sufficient of the irrigation fluid normal saline is left in without producing undue distension, usually from four to six ounces. A median lithotomy staff is then introduced. I have had several specially made, with a broad groove and a prostatic curve.

The finger is then introduced into the rectum, and placed on the apex of the prostate. A median incision, beginning about a quarter of an inch in front of anus, is then made with a plunge of the knife, with the blade forwards—the point of the knife is made to enter the urethra at the apex of the prostate, and is then withdrawn, enlarging the incision upwards while withdrawing the knife. A probe-pointed bistoury is then introduced into the groove on the staff, and the floor of the prostatic urethra divided and prostate urethra as far as the capsule; a gorget is then introduced, the right

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index finger follows. The bladder is explored, and the prostate is palpated between the two index fingers, one being in the wound and the other in the rectum.

It can then easily be ascertained whether the prostate is suitable for enucleation or cauterisation, and whether there is a line of cleavage or not. If enucleation is decided upon, the left index finger is introduced and swept round laterally, and on either side beneath the capsule. This frees each half of the prostate, which is then drawn down by a volsellum; the attachments of the gland to the roof of the urethra are then divided by a scissors, so as to preserve as much of the urethra as possible; it is far better surgery to preserve the urethra, and leave a small portion of the gland behind, than to remove the whole gland as well as the prostatic urethra. The bladder is irrigated, a perineal drain introduced, and the wound plugged—the after treatment is that of an ordinary external urethrotomy.

The operation which I have here described can be very rapidly done, and the saving of time in an operation on the aged is an important factor. In a favorable case I have done the operation in four and a half minutes.

If more room is required, the median incision can be converted into the inverted Y incision by two lateral incisions.

CONDITIONS INFLUENCING THE CHOICE OF OPERATION IN ENUCLEATION OF THE PROSTATE.

When I published my first series of perineal prostatectomies in 1903, I expressed the opinion that in some cases it was necessary to have a suprapubic opening, but increased experience in the perineal operation inclines me strongly towards the view that a suprapubic opening is seldom required. If there be very offensive alkaline urine, then preliminary suprapubic drainage with bladder irrigation, followed by a perineal prostatectomy when the local conditions have improved, is the operation of choice.

TREATMENT OF THE HARD FIBROUS PROSTATE.

In a certain percentage of cases of senile enlargement of the prostate, the fibrous tissue greatly predominates, so that it is firm and unyielding, and very hard, intimately connected with its capsule, and quite impossible to shell out; in one case I remember the gland was as firm and as hard as muscle tendon. The gland in this condition is smaller. These are the cases in which Bottini's operation has given good results.

Many prominent surgeons differ widely as to the treatment of this form of prostate. The main indications in the treatment are:—

1st. To remove the mechanical obstruction to urination; and

2nd. To obtain diminution in the size of the enlargement as a result of cicatrisation, and the modification of its circulation.

There can be no question but that any form of prostatotomy which removes the mechanical cause of the obstruction will give relief.

Prostatotomy may be done in two ways: 1st, by cauterisation; 2nd, by removal of wedge-shaped piece of the prostate, followed by prolonged perineal drainage.

Cauterisation relieves the obstruction, and causes some shrinkage of the gland. I agree that the idea of cauterisation, which originated with Bottini, is in principle correct, but I condemn and reject his method of applying it.

Many remarkable statistics have been published of Bottini's operation, but nevertheless the question which presents itself to every surgeon's mind

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is—is Bottini's operation the most reliable and safest method of cauterisation? I am convinced that Bottini's operation is an unsurgical procedure, and I have, when the opportunity has been presented, expressed that conviction.

Some of the disadvantages of cauterisation as performed by Bottini:—

(1) The difficulty of diagnosing the pathological condition of the prostate.

The anatomic relations of the prostate prevent a perfect examination, either by sight or by palpation. One is unable to determine the form of enlargement accurately.

The two methods of examination we have at our disposal, viz.:—Palpation per rectum, and by means of the cystoscope, are both faulty. Palpation gives some idea of the consistence of the gland and of the condition of the enlargement towards the rectum, but every surgeon must admit that it gives us no idea of the intravesical enlargement.

The cystoscope at first sight appears to do away with this difficulty, but it is a matter of common experience that the lengthening and distortion of the prostatic urethra very frequently interfere with its introduction.

And, further, if it be introduced, the close proximity of the intravesical outgrowth to the end of the instrument materially interferes with its effective use, since neither the prostatic outgrowth nor trigonal pouches can be seen.

(2) The operation is performed in the dark.

It is quite impossible to tell where the beak of the instrument is; one may feel it per rectum, but that only gives an approximate knowledge, both of its position and the tissues within its grasp.

(3) Inability to gauge the length of incision.

As I have already stated, there are no means of ascertaining accurately the intravesical conformity of the prostate and the length of the prostatic urethra. How then is it possible to tell the required length of incision? You can tell from the instrument how far you have to cut, but you cannot tell how far you ought to cut.

These are the main objections to the operation, but there are others—such as the want of drainage and the dangers of post-operative hæmorrhage. These are real, and not theoretical, objections.

The operative measure I adopt in the treatment of the fibrous prostate has the advantage of being a simple procedure.

It is cauterisation through a perineal wound.

The operation was first described by Chetwood and Wishart, and the method has been followed by many surgeons who have been mindful of the deficiencies in the technique of Bottini's operation.

This method appealed strongly to me, because I felt that when the cautery was used upon the prostate the operation area should be under the direct observation of the operator; that opportunity should be given for digital exploration of the prostatic urethra, bladder, and visical orifice; and that bladder drainage should be secured. All these conditions are fulfilled in the operation I describe.

METHOD OF OPERATING.

A perineal section is done in the manner I have described.

A Bodenhumer's rectum speculum with long valves is then introduced, and a clear vision obtained by a head light on the operator.

A triangular piece is then burnt out of the prostate with a Pacquelin or, preferably, an electro-cautery. I have tried removal of a triangular piece with a knife, but the cautery seems to give the better results.

This method I also apply to the very old and enfeebled, instead of the more radical and severe operation of enucleation.

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DANGERS OF ENUCLEATION.

Prostatectomy is a capital operation, therefore it is accompanied by serious complications, and it is only by a proper selection of cases that good results will be obtainable.

Age is not a correct guide to follow in selecting a case, because it is not a correct index of the patient's physical condition. If his heart arteries and kidneys are in good condition, then he is a fit subject for operation.

The dangers to be apprehended from the operation are, I think, in the order of importance :—

Uræmia.
Sepsis.
Hæmorrhage.

More remote complications are :—

Injury to the rectum.
Incontinence of urine.
Perineal fistula.
Orchitis and epididymitis.
Stricture of the urethra.

There is one other sequela, and that is impotence.

This is of no importance in an old man, but is of grave consideration in a younger and more virile one.

We will consider these dangers and complications in their order.

Uræmia.—This, I am convinced, is the most frequent cause of death. And I believe that it should be almost a *sine qua non* that the kidneys are not seriously involved before prostatectomy is performed. Unfortunately the condition of the bladder often makes this a most difficult matter to ascertain. Bladder irrigation and the administration of urotropin for some time previous to the operation are aids in helping the aged patient over on to the safe side.

Careful examination of the urine from time to time during the preparation are, needless to say, the guide to the kidney condition.

Sepsis is a grave danger, especially in the presence of an infected bladder. The best safeguards are careful preparation by bladder irrigation and efficient drainage, which is only provided for by the perineal operation.

Hæmorrhage, I am of opinion, is a more common complication of suprapubic than of perineal prostatectomy. It has occurred in two of my cases of suprapubic prostatectomy, but I have never had it occur when the perineal route had been chosen.

It is best avoided by using the median incision as much as possible, and by tying all vessels, instead of controlling the hæmorrhage by packing

Injury to the Rectum.—The rectum may be injured directly, or the wound may subsequently slough and open into the rectum.

This complication can be avoided by keeping close to the urethra, and opening it at the apex of the gland, and then in the enucleation keeping well within the capsule of the gland.

The accident is more likely to occur if Rocker's curved incision is used, and also it is more likely to happen in an extra-urethral than it is in the intra-urethral enucleation.

Incontinence of Urine.—This is not an uncommon complication after the operation, but it only lasts for a short time, and is confined then to some little dribbling. It is due to both muscle and nerve injury, and most frequently happens when extensive injury is done to urethral muscles by opening it too far forward.

Perineal Fistula.—This may persist for some little time, but it is a complication that is easily avoidable. Too protractive drainage may cause it.

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I seldom leave the perineal tube in longer than a week, but I have drained the bladder for a month without a fistula remaining. An unnecessary large incision is also a factor in its causation.

Orchitis and Epididymitis are complications which sometimes occur, and arise often through the injudicious use of instruments.

I have placed the dangers and complications before you because they are real and exist, and have all happened in the series of cases that I present to you. I notice that as a rule they are carefully omitted from the literature on prostatic enucleation.

THE RESULTS OF THE REMOVAL OF THE OBSTRUCTION IN ENLARGED PROSTATE.

To understand these there must be a clear conception of the pathological changes which take place in the bladder.

In prostatic enlargement there are two conditions present: 1st, obstruction to the urinary outflow; 2nd, a circulatory obstruction.

The effects of urinary obstruction fall entirely on the muscular coat of the bladder, and the result is hypertrophy, with lessening capacity and distensibility. This is the condition in a stricture of the urethra, and removal of the cause is followed by an involution and partial disappearance of the hypertrophy.

But in enlarged prostate another condition is to be taken into account; it is one of the greatest importance, viz., a circulatory obstruction. This is produced in the following way:—The vesico-prostatic veins are valveless, and as the prostate enlarges they are pressed against the fibrous envelope, thus obstructing the venous circulation of the bladder, producing a venous hyperaemia of the bladder walls, which in turn produce trophic changes in the bladder walls. This leads to trabeculation, sacculation, atony, and distension. These changes are further modified by cystitis.

There is a third condition presented, and that is degenerative change due to senility, taking the form of an increase in connective tissue in the bladder walls.

Can these pathological changes be removed entirely by removal of the urinary and circulatory obstruction? My experience teaches me that as a rule they cannot be removed, and that such a thing as an absolute cure of all the symptoms which follow in the train of an enlarged prostate is rarely possible, despite what has been said and written to the contrary. Great relief can be given. If the cases be done before the advent of cystitis, and in moderately young persons, then ability to completely and naturally empty the bladder may be obtained; but if acute cystitis has supervened, and the patient is aged, then there is a lessening of the symptoms—the urine is not so alkaline, and there is less frequency, and the patient enjoys greater comfort.

WHEN IS OPERATIVE TREATMENT INDICATED.

Since the operative measures are accompanied by less danger than is entailed by habitual catheterisation, they are indicated when the enlarged prostate produces symptoms which render habitual catheterisation necessary. The indications for operation become more imperative when catheterisation becomes painful and cystitis supervenes.

This not a hard and fast rule, because there are certain conditions which modify it—such as the physical condition of the patient. One would be inclined to allow catheterisation in a cultured educated man who is capable of being taught the necessity of surgical cleanliness; on the other hand, you would advise against catheterisation in one who may be in a similar condition, but who does not possess the same mental advantages.

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RESULTS OF THE CASES OPERATED UPON.

1. *Enucleation by the Perineal Route.*—Up to the present date I have operated upon twenty-five patients by this route. I did the first case nearly five years ago.

Mortality.—In this series two cases died (numbers IV. and X.), both previously recorded. No. IV. died of sepsis; he developed acute mental symptoms two days after operation, and removed and fouled his dressings. No. X. died of uræmia on the seventh day.

Of the twenty-three cases all were improved after operation.

I have been able to ascertain the subsequent history of seventeen of these cases, and find that four of them died of subsequent urinary trouble.

Nos. V., VIII., XIV., and XVII., aged respectively 68, 70, 71, and 69 at the time of operation. All were improved after the operation.

Of the thirteen cases remaining, ten of them had had subsequent attacks of cystitis, in most cases mild.

In No. VI. the perineal fistula still remained, but he was more comfortable with that than with his prostate.

2. *Enucleation by the Suprapubic Route.*—Six cases. Five cases recovered from the operation.

No. III. died of uræmia.

No. IV. died twelve months after operation, of urinary trouble. He had frequent attacks of cystitis, which began six months after the operation. I was disappointed in this case, as he did very well for the first six months.

Nos. I. and V. both had secondary hæmorrhage after the operation, but recovered from it.

No. V. I have lately had under my care. He developed an acute attack of cystitis, and the suprapubic wound broke down; this has since healed.

Prostatotomy.—Eight cases cauterised: three cases wedge-shaped piece removed.

All eleven cases were much improved after operation; seven subsequent histories were ascertainable. Five of them, Nos. I., V., VI., IX., and XI., were in good urinary health. Nos. VI. and X. had suffered from moderately severe attacks of cystitis.

Prostatotomy by Suprapubic Route.—Two cases: both cauterised. Both these cases did well. No. II. has had one attack of cystitis since the operation.

Summary of Paper.

1. Clinically there are two forms of enlargement—the soft or adenomatous, and the hard or fibrous—but there are many gradations between the two varieties.

2. The operative treatment of the soft variety is by enucleation—preferably by the perineal route.

3. The operative treatment of the hard variety is by cauterisation through a median perineal incision.

4. A median perineal section should be the first step in any operative measure on the enlarged prostate.

5. That if suprapubic enucleation be performed, the perineal section lessens the risk, since it provides the necessary drainage.

6. That it is necessary to digitally explore the prostate before being able to decide on the proper operative measure.

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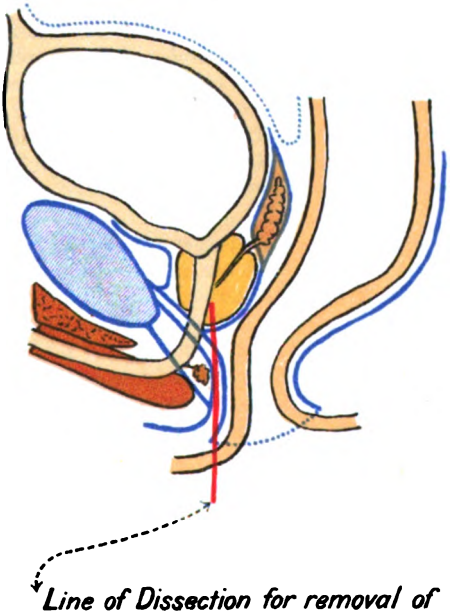

Line of Dissection for removal of Prostate by the Perineal Route.

SURVEYOR GENERAL'S OFFICE, ADELAIDE. A. Kingston, Photo-lithographer.

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CRITICISM BY DR. HINDER.

That the shock of the operation depends not so much on the route adopted as on the time taken, and the blood lost. Suprapubic drainage can be efficiently carried out by a tube introduced through the wound, and a catheter tied in the urethra, by means of which a septic bladder may be washed out by the nurse without exposing the patient. I am sorry to hear the Bottini operation condemned, because it gives, in selected cases, extremely good results, and has a very low mortality. I am inclined to think, from my experience of my own cases and that of my friends, that the ultimate result of the suprapubic operation is better than that of the perineal method.

DR. MAITLAND, in reply, said: I have endeavored to make clear that there are two forms of enlarged prostate, in which the treatment is entirely different. If enucleation is attempted in some forms of the fibrous prostate, disaster will result.

I have also tried to show that a median perineal section should be part of any operation for the relief of symptoms in an enlarged prostate.

Dr. Hinder has evaded the point at issue with regard to Bottini's operation. I advocate cauterisation—the principle of Bottini's operation—but I object to the cauterisation being done in the dark, and it is evident to every surgeon that if there are two methods of cauterising the prostate, one of which is done in the dark and the other which is controlled by vision, surely the latter—which I advocate—should be the one of choice.

It must not be forgotten that there are many methods of performing perineal prostatectomy, but the one I advocate is quickly performed, and free from hæmorrhage.

Dr. Hinder was not just to the segregator. Some four years ago I advocated its use, and then said that it must be used in conjunction with the cystoscope. These two instruments serve different purposes: the cystoscope gives a view of the bladder, the segregator collects the urines separately from each kidney. Dr. Hinder suggests that an ulcer in the bladder renders the segregator valueless; but that ulcer should be first diagnosed by means of the cystoscope, and then such an end is obviated. Both instruments are aids to diagnosing obscure kidney diseases, but have entirely different objects in view.

SURGERY OF ENLARGED PROSTATE.

BY HENRY M. O'HARA, F.R.C.S.I.

Mr. President and Gentlemen—I see I am listed to read a paper on Prostatectomy; but it is my intention to give you my experiences in the treatment of enlarged prostate, a subject which has become of great interest to surgeons. If my paper seems disjointed I must crave your indulgence, as it was written during the intervals of a busy practice. Let us hope that, like many other great innovations in modern surgery, prostatectomy will not be overdone and fall into discredit. The dissection of the male perinæum was, to me, in my student days, a difficult *crux;* and after twenty-four years as surgeon to a large metropolitan hospital, and having my share of private work, I must confess I still consider it a most complicated region. It is only a small triangular space bounded by a base line drawn from tuberischium to tuberischium, and the sides of the triangle made by lines drawn from those points to the pubes; but its contents are exceedingly intricate. To understand the supports of the pelvic viscera, it is necessary to know the position of the

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pelvic fascia which go to form the floor of the pelvis: these are the obturator fascia, the pyriformis fascia, and the recto-vesical fascia. The most important structure in connection with the surgery of the prostate gland is the recto-vesical fascia. It comes off the inner surface of the obturator fascia forming the white line, with its concavity upwards. It blends posteriorly with the pyriformis fascia. It passes downwards, covering the levator ani and coccygeus muscles, and beneath the bladder prostate and rectum, where it splits into two layers—one being reflected on the rectum, the other, or lower layer, sends a lamina downward to enclose the prostate and forms its capsule, and becomes continuous with the deep layer of the triangular ligament. This fascia completely closes the pelvic outlet and supports the pelvic viscera. My reason for giving this description I shall explain when comparing the different routes in the operation of prostatectomy. Chronic enlargement of the prostate gland has been looked upon as a necessary disease of old age; and, realising the fact that atrophy is the usual accompaniment of senility, I doubt whether every unfortunate old man is condemned to spend the winter of his days in the tortures of enlarged prostate. The idea seemed unnatural to me, and I therefore determined to find out by an examination of as many cases as presented themselves in private and hospital practice. I have now the result of 200 examinations, in patients ranging from 30 years to 82 years, and I come before you to-day to refute the long-established theory of senile hypertrophy of the prostate gland. It is not necessarily a senile condition, and I have discovered a greater percentage of enlarged prostates in men between 35 years and 50 years than in those over 70 years. I have always wondered why the prostate gland should be the only one in the human organism to undergo hypertrophic changes as the result of senile decay. In other glands we expect atrophic changes to accompany old age—particularly in the uterus, which is looked upon as the analogue of the prostatic utricle. As the result of my examination of 200 cases suffering from urinary troubles I found the following results:—

Between 35 years and 50 years, eighty-six cases, with eighteen enlarged prostates; fifty-one had suffered from gonorrhœa, one had tubercular disease.

Between 50 years and 60 years, sixty-two cases, with twelve enlarged prostates; five had marked atrophy, thirty-four had had gonorrhœa.

Between 60 years and 70 years, sixteen cases, with three enlarged prostates; two admitted gonorrhœa, one had atrophy, one had malignant disease, two fibro-myoma.

Over 70 years, twelve cases, with two enlarged prostates; one had atrophy, five had had gonorrhœa.

Thirty-two of these cases required prostatectomy. Of these, eighteen admitted to having had gonorrhœa; five of them had had syphilis also. Only the most serious urinary cases come into a general hospital; hence the large percentage requiring operation. In the healthy adult, the prostate gland is a sexual organ composed of muscular and glandular tissue. In its normal state it plays no part in the act of urination, but its function is to secrete a fluid for the dilution of the semen and nourishment of the spermatozoa. The verumontanum, a vertical elevation on the posterior wall of the prostatic urethra, is its most sensitive part, and becomes intensely congested during the sexual act. This region is a fertile ground for the development and subsequent lodgment of the gonococci. The inflammation extends from this point to the middle and the two lateral lobes, and these in their turn become engorged and swollen. Should the condition go on to abscess, and discharge its contents, the gland will contract as disintegration takes place; and, unless a cicatricial contraction of the urethra at the neck of the bladder

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is produced, an atrophied prostate may be the only result. Gout, trauma from instruments, and inflammation extending from cystitis, the result of stone, may also cause inflammation of the prostate gland; but this form generally subsides without suppuration. A very different condition, however, takes place if the acute gonorrhœal congestion, left untreated, goes on to the chronic inflammatory stage. The muscular and glandular tissue may remain permanently enlarged and eventually become a mass of fibrous tissue. The fibrous capsule of the gland also thickens in the inflammatory process, and becomes firmly attached to the recto-vesical fascia, causing great difficulty in separating it from the latter. A gonorrhœal attack invading the prostatic urethra spreads to the substance of the gland; and this condition, being rarely treated, produces a change in the muscular fibres and glandular tissue, and, in a strumous subject, probably it renders the gland liable to become secondarily affected with tubercle. I invariably examine the urethral discharge in every case of chronic prostatitis, and I have discovered gonococci in very old standing cases. It is an easy matter to express any fluid from the posterior urethra with the finger in the rectum. In acute cases, having assured myself of the gonorrhœal affection, I adopt the following treatment:—

Absolute rest in bed, when possible: a bland non-stimulating diet: leeching the perinæum: free action of the bowels, encouraged by salines: the local application of a 25 per cent. solution of argyrol to the prostatic urethra: hot hip baths and hot douching to the posterior portion of the gland, per rectum (this can be very well done with Bozesman's uterine irrigator): suppositories of icthyol. Should the condition not yield to this treatment after a fair trial, it may be necessary to drain the bladder in the suprapubic region, and give the urethra phyisological rest. I have never seen massage to the prostate do any good. From the data obtained from my patients, I feel satisfied that the enlargement of their prostate glands commenced with the later stages of their gonorrhœa, and had these been treated surgically at the time I think the majority of the cases could have been saved the enormous hypertrophic conditions that I found. The treatment of gonorrhœa is much too lightly undertaken, and it is my invariable custom to warn every patient what the direful results may be if neglected. The symptoms of chronic prostatitis may be very slight, and you will often find it difficult to persuade your patient that there is any need for care or treatment. You are all aware, gentlemen, of the complications that may arise from an attack of gonorrhœa; but, as I am only dealing with prostatic enlargements in this paper, I will confine myself to that gland. If the lateral lobes only are enlarged, only slight symptoms may continue for years; but should the middle lobe become affected it bulges backwards and upwards into the bladder, and carries the urethral opening up above the water line, forming a valve-like condition which eventually partially or completely blocks the flow. Before advising a patient to undergo the operation of prostatectomy, except in most urgent cases, I invariably give paliative treatment a fair trial: by this I mean daily irrigation of the bladder with some mild antiseptic lotion—preferably weak solutions of nitrate of silver, one in ten thousand, followed by a wash-out with normal saline solution; the passage of large sounds, curved at first and gradually becoming straighter, so as to bring down the prostatic opening of the urethra to the level of the floor of the bladder. This treatment will sometimes break in a patient to adopt catheter life on his own account, with a certain amount of comfort and safety. Men of placid temperament endure catheter treatment for years without injury to their nervous system. So long as they can attend to strict asepsis, and keep the bladder clean, they are best left alone. Operation is demanded when, from the retention of urine—either partial or complete—these conditions cannot be fulfilled, or where the patient's life is jeopardised through

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loss of sleep or general nervous irritation, the result of constant attempts to empty the bladder; where there is any indication of beginning renal disease; also, in certain occupations where it is absolutely necessary to hold the water for long intervals—such as coachmen, enginedrivers, &c. There are two forms of myomatous prostate very similar to those found in the uterus, the hard fibro-myoma—the discreet tumors—and the soft œdematous myoma. They grow slowly and insidiously, and, being of a non-inflammatory nature, do not affect the capsule of the gland, which merely stretches, leaving the recto-vesical fascia intact. We find enucleation from their fascial surroundings is therefore generally easy. The other causes of enlargement of the prostate gland, tubercle and carcinoma, I have only met with on two occasions; but I am inclined to think a small percentage of senile prostates become malignant. Of the thirty-two operations performed, two died—one, a carcinoma, of shock, on the third day, in which I was associated with Dr. Clayton: the other was a large fibrous prostate, the condition being complicated by chronic nephritis. The patient lived for three months, and eventually died of renal disease. One can hardly attribute his death to operation. I now show you his bladder and the prostate, and you can judge for yourselves the result of the operation. Where the condition of a patient is so serious as to require operative interference, several methods of treatment have been advocated of late years; but I will merely mention these, as I have had very little experience in all but prostatectomy. The American and English surgeons originally advocated castration, and I have adopted the plan in three cases. One was certainly relieved, but the other two developed acute mania and never recovered. After an operation for the radical cure of hernia in a man of 55 years, with Dr. Weigall of Elsternwick, acute cellutitis developed in the scrotum. The parts sloughed, and the testicle on the left side, was destroyed. It is ten years since the operation, and, on examination, Dr. Weigall assures me the lateral lobe on the side of the missing testicle is now completely wanting, and the lobe on the sound side is enormously hypertrophied. I think this is a good test that White's operation had something to recommend it.

Vasectomy is too uncertain in its result. It can be done under local anæsthesia; but I would not recommend it, unless the patient refused the more radical operation. The division of the bar at the neck of the bladder by the galvano cautery, or electric knife, as recommended by Bottini, is to my mind a dangerous procedure, even with the aid of a cystoscope, and I cannot recommend it. The most satisfactory operation at the present time is prostatectomy, either by the suprapubic or perineal method, and each has its advocates. Having performed the superpubic operation thirty-two times, with but one death, I must, for the present at any rate, pin my faith to that method. My colleagues at the Alfred Hospital, Messrs. Cooke and Hamilton Russell, have had equally good results.

In nine of my cases I had some post-operative complications; two elderly gentlemen, aged 77 years and 69 years, respectively, became temporarily demented, but have completely recovered. Three had urinary fistula for four and five months: two have completely recovered, but one is still unhealed. One patient developed thrombosis in the deep femoral vein, with much œdema of the leg: he has now quite recovered. Three are obliged to empty the bladder with catheter, having no muscular power in their large sacculated atonic bladders. So that, out of thirty-two prostatectomies, twenty-six are quite restored to health; one, the malignant case, died as the result of operation; and four are relieved. I have not found the help that other surgeons seem to get from the cystoscope: in fact I have given it up in prostatic cases, and rely on digital examination to form my diagnosis. I find with my finger in the rectum, and, having emptied the bladder, I can

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always map out the condition. If the urethral groove can be clearly defined per rectum, the trouble is invariably in the middle lobe. Before giving a prognosis as to the probable result of the operation, it is well to find out the amount of muscular expulsive force the bladder has. This can be discovered by passing a catheter, filling the bladder with some solution, and then directing the patient to force the fluid out. If the stream is a fairly vigorous one, the muscular wall must be still active: if the stream merely dribbles out, it is an indication of atony, and the prognosis is bad. However, by removing the enlarged gland, catheter life is made bearable, and the outlet of the urethra having been lowered to the floor of the bladder the decomposing urine that had previously lain in the bas fond can be evacuated.

I am well aware of the brilliant results of perineal prostatectomy in the practice of other surgeons; but the advantages which I claim for the suprapubic operation are:—

1. Rapidity of performance. Since writing this paper I removed an immense prostate at the Alfred Hospital, on September 1st, in the presence of a number of surgeons, two of whom took the time; and the operation took exactly four minutes. The prostates that require removal are generally inside the bladder, and easily accessible from the front. With the bladder filled, and the patient placed in Trendelenberg's position, an incision about three inches long is made in the mid-line, beginning at the pubes. Having separated the recti, divided the transversalis fascia, and exposed the bladder, I fix it with an instrument I now show you, which assists in holding it in position to be opened. I then fix, with a suture on either side of the upper part of the incision, the bladder to the skin: this prevents injury and leakage into the tissues. I expose the gland by an incision through the mucous membrane on its anterior superior aspect, and enucleate with my finger, working from above downward; another finger in the rectum will help to push up the prostate, taking care to avoid tearing the urethra if possible. I have found it a good plan, when the bladder is in a foul condition, to keep up constant irrigation for the first few days after operation, and I have devised this irrigator, which I now show you. It is made of metal, has two tubes—a small one for inflow of saline solution, and a large outflow tube. It prevents the lodgment of any septic material in the raw cavity left after removal of the gland. I think it also helps to control hæmorrhage.

2. It is practically impossible to avoid wounding some branches of the prostatic flexus of veins, and unavoidable bleeding can be controlled by sponge pressure on holders. This is where the value of the facsia on the posterior surface of the bladder can be realised. The fascia and ligaments on the floor give a firm resistance to pressure from above, which you cannot obtain in the perineal operation.

3. No important structures are injured on the route to the gland if the incision is made in Retzius' space, which is above the attachment of the recto-vesical fascia to the pubes. I have always thought that splitting the valuable supports to the pelvic viscera—that is to say, the fascias lying below the prostate—must interfere with the action of the muscles and ligaments about the rectum and neck of the bladder, and to reach a middle lobe or any tumor bulging into the bladder from a perineal opening is a matter of extreme difficulty, and increases the danger of injuring those important structures—the seminal vesicles and vasa deferentia.

4. In cases of very old men it is an advantage to sit them up soon, thus avoiding the danger of hypostatic pneumonia. This can be done with suprapubic drainage; but, with a perineal tube, a patient cannot well sit up.

5. The suprapubic wound can be more easily kept asceptic, the proximity of the perineal opening to the anus causing great risk of infection.

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Sixteen years ago, before Mr. Gill published his operation, I performed prostate-myomectomy in two cases, with Dr. Weigall, and they are at the present time in good health: the remaining portion of the prostate seems to have shrunk.

EXHIBITS.

I have brought with me five specimens of enlarged prostate, to illustrate the different forms met with:—

A.—An adenoma of middle lobe, from a patient of 70 years, which caused complete retention. The three calculi accompanying were found in the bas fond. I had no difficulty in shelling out this lobe. There was no history of gonorrhœa.

B.—A typical fibroid from a man of 39 years, who had suffered from gonorrhœal prostatitis at the age of 28. This was a difficult gland to enucleate, owing to the dense fibrous adhesions set up between the capsule proper and the recto-vesical fascia.

C.—A soft œdematous myoma from a man of 52 years. It is very similar to those found in the uterus. He had a history of gonorrhœa.

D.—Multiple isolated myomata, complicated by over 100 prostatic calculi, from a patient aged 77 years, who had history of gonorrhœa.

E.—Large prostate removed on Friday last (September 1st) at the Alfred Hospital. Patient aged 77 years. No history of gonorrhœa.

F.—My bladder irrigator, made for me by Messrs. Mayer & Metzler, London and Melbourne.

G.—My instrument for fixing the bladder before opening. Made from my design by Dietrich, Melbourne.

H.—A bladder from the patient who died three months after operation.

The deductions I draw from my examinations are:—That the hard, fibrous, adherent prostate, in whatever lobe it may be, is invariably the result of a gonorrhœal infection, and may develop at any age. That a mixed fibro-adenoma probably develops its excess of fibrous tissue as the result of gonorrhœal infection, and may develop at any age: that the so-called soft œdematous myoma, which attains such an immense size, is probably the result of a blocking—through some catarrhal condition, probably gonorrhœa—of the lumen of the gland ducts, so that the prostatic secretion is confined in tubules which rapidly become distended: this is the form of hypertrophied prostate, irregular in shape, that so quickly develops, and may involve only one or all the lobes, and is more often seen after 50. It does not become adherent to the recto-vesical fascia, and is therefore easily shelled out. That the simple adenoma is a rare condition, most frequently found in the middle or third lobe, and may develop at any age, has a smooth capsule, and is easily enucleated. That cancer is a rare condition. That tubercle of the prostate is generally a secondary condition.

Prostatectomy might aptly be called the "Finger operation." The diagnosis is made with the finger, and, having exposed the gland, the rest of the operation ought to be done with the finger. It requires a long strong finger, with good tactile sensibility, to accomplish the enucleation satisfactorily; and I cannot imagine a more pitiful spectacle than a little man with a short finger attempting to dig out a firmly embedded fibrous prostate. Scoops and forceps of any kind can only be a menace to the patient.

In conclusion, I must express my appreciation for the kind and skilful assistance I have received from Drs. Cuscaden, Joske (who completed one of my operations), Power, Lang, Anderson, Clayton, Weigall, Shortt, McKeddie, Harris, Hearne, Pitt, and my house surgeon, Dr. Lind. Their help has been on every occasion a great factor in the success of the several operations, and I take this opportunity of offering them my sincere thanks.

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DISCUSSION ON PROSTATECTOMY.

DR. STEER BOWKER: I was greatly interested in what Dr. O'Hara stated as the result of his examinations, that the prostatic enlargement did not necessarily belong to a senile change; for I have often noticed the enlargement in comparatively young people.

The first prostaticalism I did some seven years ago, and was by the perineal route: it resulted in a fistula between the bladder and rectum. This rather put me against the operation. But I have seen such good results at the hands of my colleague, Dr. Maitland, that I am not now so sure as to which is the best method. But of this I am sure, that the operator who always does the same operation is wrong; for judgment must be used, and in each case the most suitable operation chosen.

Dr. Maitland is rather too sweeping in his condemnation of the Bottini operation. I have had some excellent results, and this operation can be done without a general anæsthetic by the aid of adrenalin and cocaine, and as shock and the effect of ether and chloroform in these old subjects should be avoided in certain cases, a Bottini might be done when another operation would probably end badly. Shortly before leaving Sydney, I did one on an old man who had 17 ounces of residual urine. It relieved his frequency, but I was disappointed on trying a week after the operation to find he had still about 8 ounces of residual urine.

I had also some difficulty with a suprapubic operation. It must be a very easily shelled-out prostate which would allow of removal in four minutes, as Dr. O'Hara did, or four and a half minutes, as recorded by Dr. Maitland; for though I am not a weakling, and have fairly strong hands, it took a considerable amount of force, which left me with aching hands for the rest of the evening, to remove the prostate. He had complete retention about a week after, and my house surgeon could not get a catheter in. I had to open the bladder again and do retrograde catheterisation, and left a cathether in. Eventually he became quite proud of his powers of micturition. I think Dr. Hinder is rather rough on the segregator, for no doubt it has it uses, though it may have its errors. To listen to what he says would lead one to suppose that catheterisation of the ureter was quite an easy matter: this it most certainly is not. This method also has its errors and dangers peculiar to its usage.

PERSONAL EXPERIENCE OF THE OPERATIVE TREATMENT OF SENILE DISEASE OF THE PROSTATE DURING THE LAST FIVE YEARS.

BY THOS. FIASCHI, M.D., M.CH., ITALY.

The following account covers the cases of senile disease of the prostate operated on by me since my return from South Africa in December, 1900.

The only palliative operative procedure which I adopted was suprapubic drainage; this was resorted to in four cases in which there was a great deal of prostration, cystitis, pyelitis, &c., rendering these cases unfit for the time being for any serious operation. In two of these cases the prostate was afterwards removed successfully. In the third, death took place from heart failure, whilst under an anæsthetic and about to have suprapubic prostatectomy performed. In the fourth case death occurred from uræmia, fourteen days after a stone was removed and a drain placed above the pubis. This patient had almost died of hæmorrhage before the drain was introduced; he had

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previously been operated upon as follows :—Lateral lithotomy (in Melbourne), litholapaxy (in London), suprapubic lithotomy (in Sydney), and Bottini (on the Continent of Europe). This would have been a most suitable case for perineal prostatectomy at an earlier stage, before his kidneys got small, granular, and septic. No. 1 in the list of cases had a drain left above the pubis two years before, since no catheter could be passed per urethram. He passed two years in comparative comfort wearing a urinal, but as the prostate enlarged it pressed on the rectum to such an extent as to cause trouble; I therefore removed it.

The operative procedures adopted by me for the removal of the prostate were :—(1) The "combined operation," (2) suprapubic, (3) perineal alone.

In the cases where the combined operation was made use of, the perineal incision was a crescentic one, in front of the anus. A careful dissection was made to the apex of the prostate behind the bulb, followed by incision of prostatic sheath and enucleation with the fingers, aided by pressure from above. Draining was by the perineum, and for two or three days by the suprapubic incision also. In only one case was the suprapubic route alone made use of. In this case the difficulties of enucleation were considerable. Although the result was fairly satisfactory, this method did not appeal to me as a general procedure for the removal of the prostate. Latterly I have come to the conclusion that the suprapubic incision is quite unnecessary, and that the prostate can be removed with greater precision—and under direct view—through the perineal route alone, and that is the operation to which I now give preference.

The following is a short description of the perineal operation which I adopt :—For a few days before the operation I administer urotropine and plenty of water to drink, the dose of urotropine varying with the amount of cystitis or pus in the urine—from 5 gr. to 15 gr. thrice daily. At the same time the bladder is washed occasionally. Immediately before the operation the bladder is washed out with boroglyceride solution, and a soft catheter is placed in the urethra—either a Coudie or a Jacques. The patient is then placed in the lithotomy position, with a sandbag under the sacrum, so as to elevate the pelvis.

The parts having been previously shaved, an inverted Y-shaped incision is made in the perineum. This incision is deepened into the ischiorectal fossæ behind the line of the superficial perineal nerves, and in the middle line it passes through the central point of the perineum, so that the anterior end of the sphincter ani can be pulled back, and the accelerator urinæ forwards. The further dissection towards the apex of the prostate can be facilitated by placing the middle finger of the left hand, protected by a finger-stall, in the rectum. The incision is carried back in the middle line to the apex of the prostate, carefully avoiding the muscles connected with the bulb and the compressor urethræ in front, and the rectum behind. The dissection is then carried towards the apex of the prostate between the anterior fibres of the levator ani muscle (levator prostatæ), and the under surface of the apex of the prostate is well displayed; the finger will then pass easily between the recto-vesical fascia (prostatic sheath) and the prostate. By separating the fibres of the levator ani by means of retractors, and pulling upon the recto-vesical fascia with a pair of forceps, the prostate comes well into view. The removal of the prostate should then commence after all bleeding is arrested. The floor of the prostatic urethra is opened well back longitudinally, and the finger is introduced so as to determine the disposition of the enlarged parts. In the case of a large adenomatous prostate, one lobe is first enucleated with the fingers or a blunt dissector, and then the other lobe is removed. The finger is then introduced into the bladder, and if there is an enlarged middle lobe,

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it is turned out and enucleated, or its posterior attachment divided with a pair of scissors. Search is then made with the finger for stones, which are frequently present, although not detectable beforehand. If the prostate is hard and fibrous, the whole substance of the gland can be removed with a tonsil punch, guided by the finger in the bladder through the floor of the prostatic urethra. Lastly the wound is washed, all hæmorrhage stopped, and a large drain tube fenestrated for a short distance from its inner end placed in the bladder. This tube is fixed in the bladder by first passing a stitch through the bladder wall at the posterior end of the incision that was made in the floor of the prostatic urethra, and then passing the thread through the side of the tube within three-quarters of an inch or one inch of its inner end. When the thread of catgut is tied it brings the tube into position. This stitch is made of catgut, which will dissolve in about six or seven days, when the tube comes away. If there is much oozing a light packing of gauze is placed alongside the tube. The superficial wound is then closed.

In the after treatment the bladder is irrigated once or twice daily, and the patient is kept on small doses of urotropine. If any gauze packing is made use of, it is removed after twenty-four hours. The average time for the perineal incision to heal is about twenty-one days.

In the majority of cases the floor of the prostatic urethra was removed, but the roof and sides were preserved. No attempt was made to save the *vasa deferentiæ*. On making inquiries of some of the patients I found they had good power of erection. I question if the seminal fluid is any use without the prostatic secretion, so that it might be useless saving the ducts.

The results, as seen from the accompanying table, were fairly good. There were no deaths, and with the exception of those cases which turned out to be malignant, relief was almost invariable. I kept no record of weights of parts removed, nor any photos, but some were very large. I refused operation only in a few cases where sepsis had invaded the kidney, and in cases of senile heart, and these cases only lived a few weeks. It was interesting to watch the improvement in the renal function that took place when the micturition became normal. Albumen disappeared from the urine, and the amount of urea excreted increased. The operation of perineal prostatectomy properly conducted I consider not as dangerous as Cheselden's operation, and, other conditions being favorable, so soon as cystitis commences from enlarged prostate, I think prostatectomy is indicated. Generally speaking, when an obstructing prostate is the cause of septic cystitis, it is as necessary to remove it as it is to remove a stone from the bladder.

As to the choice of operation, I prefer the perineal route alone, since one can get parts under direct view, and have more control of hæmorrhage and more efficient drain, and the largest stone can be removed safely by carrying the incision in the bladder backwards. The fixing of the drain tube to the wall of the bladder ensures the effective drainage of the urine, and does away with the necessity of pushing it into the fundus and causing pain.

Complications.—Hæmorrhage was not alarming in any of my cases, and it was much less and more under directed control in those cases removed by the perineal route alone.

Incontinence or dribbling sometimes followed the perineal method, but never to any serious extent, so far as I could ascertain. It amounted only to a loss of control when the bladder got a little full, and this invariably disappeared in a short time. I ascribed it to injury to the compressor urethræ muscle when the dissection towards the prostate was carried too far forwards.

Perineal fistula was a cause of annoyance in a few cases, due probably to the tube being retained too long a time, but it never amounted to more than the escape of a few drops of urine when micturating.

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Epidydimitis was the most common complication I had to deal with. It occurred generally when the urine began to pass by the urethra. In one case suppuration took place.

There is one other feature in this list, and that is the large proportion of cases in which the disease turned out to be cancer of the organ. The proportion was about 13 per cent. This condition was suspected only in two of the cases before operation. In one case (No. 24) the finger, when passed through the neck of the bladder, gave a sensation like as if it had passed through a carcinomatous cervix uteri from above. Some of these malignant cases got temporary relief, and one had no urinary symptoms for twelve months after operation.

In future I intend to keep a more careful record of the histology of the parts removed, so as to determine more definitely the effects of removal of cancer of this organ.

For the histological examinations I am indebted to Professor Welsh.

URINARY SURGERY.

By Critchley Hinder, M.D.

It is my intention to discuss with you various matters of interest which have cropped up during the past few years in connection with a branch of work in which we must all be considerably interested. I feel that I would rather do this than confine myself to one subject in urinary surgery, for in doing so I should hardly be able to avoid repeating matters which can be gathered from any text-book, and which would be little appreciated by such an audience as that which I have the honor of addressing to-day. As tersely as possible let me give you my own convictions, while mentioning cases which have appeared to me to be of special interest.

Foreign bodies are not found in the bladder very commonly, but they certainly give rise to a considerable amount of frequency of micturition and tenesmus. I met with two cases in women, where the familiar hairpin was passed into the urethra until it slipped beyond reach. In each case the hairpin was located with the cystoscope, and then, by securing the blunt end with a lithotrite, it was removed without damaging the urethra. On two other occasions women in the general hospital were having their bladders drained with glass catheters. The nurse who had charge of these catheters had boiled them until the pot was dry, and the catheters had probably cracked: at all events about 3 inches of glass catheter were left in the bladder. An abler and more careful nurse was placed in charge, and there was no further trouble, except that one other patient—jealous of the interest that was roused—actually broke her catheter in her bladder herself. In each of these cases I located the broken piece with the cystoscope, worked it into position with a small lithotrite, and removed it per urethra. A boy of 5 years passed a pin into his urethra. It disappeared into his bladder. By the act of micturition it was passed into the lower end of his urethra, and slipped back again. With my finger in the rectum I was able, with the help of a long urethral forceps, to secure the pin at the neck of the bladder and draw it out. I removed a stone by lithotrity from an old man of 80: the nucleus was a piece of gum elastic catheter, which had broken off four months before, and which the patient estimated to have been about an inch and a half long. The old fellow said that he had missed the broken piece, but had no idea that it

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could possibly have been left in his bladder. In fact, with a cystoscope and a lithotrite, practically anything which can be passed into the bladder can be removed without a cutting operation. I have removed fairly large stones from the urethra on two occasions. In each case the stone was about the size of a pigeon's egg, and had formed in a pocket in the urethra immediately behind an old stricture. I have twice found stones in the substance of the prostate gland, once in the prostatic urethra, and once in a man of 42, who had a slight median lobe enlargement. The lobe was so developed that it formed a little pocket on the distal side.

During the past eight years I have only been compelled to cut for stone three times. One stone weighed 8 ounces, and was so very hard that the lithotrite was powerless. This patient was a flower-seller in the city, and only 21 years of age. In another case a man of 72 years had an hour-glass stone so firmly held by the lower and larger end that it could not be dragged out of the hole in which it was pocketed; in fact, it was a difficult matter to do so after a suprapubic opening had been made. On another occasion a flat dark stone was localised in a little pocket in the bladder wall. This stone appeared to exactly fit the cavity, and could not be dislodged with sound or lithotrite. A suprapubic opening revealed the fact that the stone, flat and smooth, exactly fitted the depression. In crushing a stone it is better to use a lithotrite, about number fifteen, and a flushing catheter about the same size; larger instruments are rarely necessary, and they are inclined to tear the urethra.

The cystoscope should always be used after the operation has been completed, in order to see if any fragments remain. The old rule, that we may consider the bladder cleared if we hear no further rattling against the evacuating catheter or if we feel no fragment with the sound, is a fallacious one, as on four or five occasions the cystoscope has revealed the presence of a fragment which had escaped detection, or another stone which had formed on the bladder wall in a limpet-like fashion, notwithstanding the fact that an ordinary examination had convinced me that I had left the bladder clean. On one occasion another stone as large as a pigeon's egg was discovered at the end of four months after operation, showing how remarkably quickly stones are formed. Lithotrity should be performed with the greatest gentleness, and, though it may not be possible to avoid the production of a little hæmorrhage from the prostatic urethra in old men, the bladder wall need not receive the slightest damage. If the patient is kept in bed for a few days, so as to diminish the irritability of his bladder wall, small stones may be crushed without an anæsthetic. I have operated in this manner on three occasions in feeble and phthisical patients. The lithotrite should be introduced once only in the majority of cases; frequent introduction of instruments, and the use of large instruments, is accountable for a great deal of the discomfort which lithotrity patients experience after operation. The pelvis should always be tilted, so that the stone may be crushed away from the sensitive trigonal region. Very frequently patients are able to get about on the third day, or even the following day if the stone is small.

I have on four occasions been able to detect with the cystoscope the presence of a stone in the lower end of the ureter. The history of the case, the puffy appearance of the lower end of the ureter, together with a slight bulging of the bladder wall immediately above the orifice, made the diagnosis fairly certain. A good skiagraph under such circumstances is invaluable if further verification is needed—though, in one case, the cystoscope revealed a small stone which two or three photographs missed.

Stones in this situation are best reached by placing the patient in the Trendelenberg position, and, through a suprapubic opening in the bladder,

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slitting up the ureter; or the orifice of the ureter may be dragged down, a probe passed up it, and when the stone is reached the stone and the probe are pinched up in a pair of forceps and cut down upon. In this manner stones may be removed at least one and a quarter inches from the urethral orifice.

The free abdominal incision adopted by some men for the removal of stone in the lower part of the ureter is sometimes followed by the most disastrous results. Owing to the necessary interference with the nerve supply the muscles waste, and a large ventral hernia may be formed. It certainly is much wiser to make all abdominal incisions away from the median line, parallel to the line of the intercostal nerves. Thanks to the excellency of recent skiagraphic work, stone in the kidney is diagnosed with much greater ease than was formerly the case. In removing a stone from the kidney I draw the kidney on to the loin, if possible; and we all know how often this manœuvre is simply impossible. A small shallow incision is made in the middle line and my finger does the rest. This is safer than making a large deep incision. On one occasion I met with dangerous hæmorrhage, which I was unable to control without removing the kidney. It was owing to the fact that the renal artery had bifurcated, and the lower branch had been torn across. Under ordinary circumstances simple pressure and mattrass sutures will be found sufficient to stop any renal hæmorrhage occasioned by a median incision. In cases of suppression of urine, associated with stone, it is safer to incise the kidney and its capsule, allow it to bleed, and leave the stone until the patient has recovered somewhat. These patients have very little reserve power, and usually collapse suddenly. Primary growths of the bladder form but a small proportion of the urinary cases which fall into the surgeon's hands. A portion of the bladder wall should be removed with the growth in every case. None of my cases lived more than twelve months without recurrence.

Villous papillomata are rarely single. I have only met with one of this kind. There are usually two, or perhaps several, patches to be found. They should be removed as early as possible, partly because one can never tell whether the growth is wholly simple and partly because these growths have a strong tendency to degenerate and become carcinomatous. As an example, let me quote the case of a man of 45 years of age, who had been ill for five years with occasional attacks of slight hæmaturia with frequency, and a little cystitis. These attacks appeared to be so slight that for a long time he paid little attention to them. When I examined him with a cystoscope a villous-covered growth could be seen, which, when removed, had a basal attachment of the size of my forefinger nail. An examination by Professor Welsh showed it to be a villous papilloma with carcinomatous degeneration at the base. Another patient had large villous patches—in fact, I might say masses of growth scattered all over the bladder. His history extended over three years, and it was only during the past few months that the hæmorrhage and frequency had increased to a marked extent. On introducing the cystoscope it felt as if it was thrust into a soft loose sort of seaweed. I opened suprapubically, and was astonished at the large masses which were present. A small portion removed for examination purposes was reported by the pathologist, Professor Welsh, to be a carcinomatous degeneration of a villous papilloma. The long history would be apt to induce us to presuppose that the growths were originally benign. Many malignant growths appear to become covered with villous processes. Our knowledge of these cases, then, convinces us of the necessity of advocating the early removal of villous papillomata—or I might go further, in saying that it forces us to the conclusion that every case of hæmaturia, however slight, should be submitted to cystoscopic examination early and while the blood is present in the urine.

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About four years ago, towards the end of summer, I saw a man of 28 years of age. He told me that he had had slight hæmaturia and frequency during this and the previous summer, but that during the winter he was perfectly well. The cystoscope showed an irregularly nodular and superficially ulcerated surface, which appeared a little like tubercular disease; but, per rectum, there was a density that I could hardly understand, in fact I only put aside carcinoma because of the patient's youth and the history of recovery during the winter. I drained through the perinæum; but, although he seemed to improve at first, he was soon about as bad as ever. He was going to England, so I asked him to see Hurry Fenwick. Fenwick was at first in doubt, but after a short time came to the conclusion that the condition was carcinomatous. Henry Morris concurred. The man died eight months after of extensive malignant disease, so that he had symptoms for about two years and eight months. He had a strong tubercular history; and I often wonder whether he had a tubercular bladder with superadded carcinoma. At the same time, Fenwick has reported two cases of early malignant disease very similar to this.

Unfortunately, malignant disease may advance considerably without any sign or symptom. I once saw a man who had a sarcoma as large as a walnut situated above and to the right of the urinary meatus. Four days before he was sent to me he had a profuse hæmaturia: previous to this he was absolutely unaware of any indication of illness. My experience of malignant disease of the kidney has been a sad one. It is difficult to detect until well established, and has invariably recurred within twelve months of the removal of the organ. No doubt all hope of success must depend on early recognition. Malignant disease of the prostate appears to me to be very difficult to recognise. I have had very many cases of enlarged prostate sent to me for removal which have proved to be malignant; and, again, I have seen many cases where I found it impossible to say whether the case was malignant or inflammatory until the patient had been under observation for two or three weeks. These latter cases were usually cases of slight hypertrophy of the prostate, in which inflammatory conditions of a low type had been set up—either by the presence of stone or by too frequent and at times altogether unnecessary catheterism. Slight irritation and frequency in early enlargement of the prostate is more easily set right with a dose of saline aperient than with a catheter. Malignant disease of the prostate is very common, and it frequently intrudes itself in cases of hypertrophy of the gland; but my own personal observation has shown me that its chronicity is often a very marked feature. As an example, let me quote a case. A man well over 60 years of age was sent to me. I stated that he had undoubted malignant disease of the prostate, and operative treatment was out of the question. He did not feel very ill, and was anxious to go to Queensland in connection with a mining matter. He came back at the end of twelve months, a stone heavier than when he left, and with about the same amount of slight frequency. The patient was sent to me again, and a rectal examination showed that the growth had spread across the pelvis to a marked extent. Within a few weeks he suffered more pain and went down hill, dying within six months. This case is an example of a statement which I have frequently made, namely, that malignant disease does not *per se* give rise to loss of weight, but that the loss of weight is always due to interference with the body functions, or the mental worry occasioned by pain, or the knowledge of its presence. If ulceration takes place the consequent illness is due to mental worry and septic absorption, and not to the mere fact that the disease is malignant. I have operated upon the prostate for non-malignant hypertrophy one hundred and four times. In forty-seven cases by partial

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prostatectomy, twice by simple section and drainage, twenty-four times with Freudenberg's modification of the Bottini instrument, and thirty-one times for the complete removal of the gland.

Simple perineal section and drainage I found to be of little permanent service. Partial section was done on all sorts of cases by the suprapubic method, and perineal drainage was adopted so that the tube might maintain a free opening while scarring was taking place. The mortality was 12 per cent.; but some of the patients had to be relieved, and the additional re-section hardly aggravated the shock attending the cystotomy, which gave them their only hope of relief. Some of these partial re-sections were done eight and nine years ago for absolute retention, and are perfectly well now. I find that partial re-section relieves impotence and need not necessarily cause it. In order to get a high percentage of recoveries from prostatectomy great care must be made in the selection of cases. Freyer is reported to have said that he has operated upon only one out of every twelve cases sent to him. I have been somewhat unfortunate, having lost seven cases out of thirty-one. I say unfortunate, because I could hardly prevent their deaths; three died uræmic a little over a week after operation. One of these came to me with profuse hæmorrhage: even after suprapubic cystotomy I was unable to stop the blood which oozed up from his prostatic urethra, so that at the end of three days I was compelled to remove the gland; it weighed eight and a half ounces. The patient was 79. The hæmorrhage ceased, and, so far as his bladder was concerned, he was well, but hiccoughing set in and he died of uræmia on the eighth day. As an evidence of what will happen at times, my friend Dr. Clubbe tells me that he had a patient a short time since who hiccoughed continuously for a fortnight after prostatectomy and then recovered. Two others died of pneumonia. Another at the end of the sixth day raised himself on his elbow, to speak to the nurse, and fell back dead. Another man, who suffered badly from prolapse of the rectum before operation, strained when the nurse was administering an enema on the fourth day to such an extent that the nozzle of the Higginson enema tube penetrated his bowel, for he experienced sudden pain and died in twelve hours. I found the enema in his peritoneal cavity: since then I have vetoed enemata in these cases. It is hardly right to refuse operation to an old man if he is prepared to take the risk. He knows too well what a miserable existence he is leading, and we know what a miserable death he is likely to die if left alone, apart from the by no means uncommon incidence of malignant disease. I urged an old man to submit to operation for hypertrophy of the prostate: he objected. About six years after he came under my care with great frequency and bladder spasms. He asked for operation, but I refused, because I felt that his condition was almost hopeless. He died within two weeks in abject misery, with intensely painful and almost continuous spasms up to the time of his death. The manner of his death made a great impression upon me. Ever since that time I have simply operated with a view to cure, if possible; and if not, to give the patient a maximum amount of comfort.

A train of accidental circumstances has given me a poor percentage of recoveries, but notwithstanding this I feel that I acted in the best interests of the patients in operating. The actual enucleation of the prostates took from two and a half to eight minutes. I prefer the suprapubic method, because there is no chance of interfering with the sphincter, the patients are more comfortable, and can be easily kept clean by the nurse. I operated perineally once. I saw no advantage in the operation, and the patient used to get dirty—his scrotum hung into the wound, and he resented the necessary meddling with his genitals. All my cases who lived recovered with normal

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micturition. I do not decry the perineal operation, for I feel that a careful operater could get as good results from this method as by the suprapubic method. The Bottini operation I have performed on twenty-four cases, and without a death. Only small prostates were attacked, and some of the patients had very dirty bladders. In five of them there was foul urine, and tenderness on pressure over the kidneys; whether it meant pyelitis or not, I cannot say, but they gave me the impression that they would stand very little in the way of operative treatment. On four occasions a stone was crushed and removed, and the prostate dealt with at the same sitting. Only small prostates were operated upon though the residual urine varied from one and a half to thirteen ounces. On one occasion when I attacked a prostate which, I should say, would have weighed about two and a half ounces, the final result was by no means equal to that of the others, and the recovery took some time owing to the many necrotic pieces which of necessity had to be passed. My experience has convinced me that the Bottini operation is well adapted for small prostates, which, although small, may give rise to marked or even complete obstruction to micturition, or to irritable and frequent micturition—disagreeable symptoms, which appear to annoy most prostatic patients. The risk attending the operation is certainly small, for I have never refused this operation to any patient with a small prostate whose symptoms have demanded relief. If the operation falls into disfavor it will be because it has been used indiscriminately, without due thought as to its action and its power to give relief,

Tubercular disease of the genital organs is fairly common. It is astonishing how often a tubercular deposit will be found in the testes of a man who is the picture of health and vigor, and this certainly occasionally leads the practitioner astray. Re-section of the diseased part is attended with good results in early cases, and one is not averse to such a procedure because any recurrence is so easily detected.

Operative interference in the case of tubercular disease of the prostate and the vesiculæ seminales should not be lightly undertaken, for we all know what a remarkable effect climatic and dietetic treatment has upon tubercular conditions, and how dangerous it is to import the septic element into such cases; so that, unless old inflammatory tissue gives rise to pain and discomfort, they are much better left alone.

Tubercular ulcer of the bladder almost always occurs in the vicinity of the urethral orifice. If the irritation is marked, drainage and careful frequent washing out with weak solutions of silver nitrate is of service. In all cases open-air treatment and rest is invaluable. In tubercular ulcer there is a marked tendency to recurrence, even after a lengthy period without symptoms. The simple ulcer is nearly always solitary, and very like a gastric ulcer: bladder drainage and washing out three or four times daily gives excellent results. I should like particularly to call your attention to the ease with which the ureter can be felt bimanually, especially in women. Even in thin normal subjects the ureter can sometimes be felt. In suspected tubercular disease a thickened ureter, which can often be felt bimanually, is a valuable diagnostic aid.

Re-section of a part of a tubercular kidney is to be condemned: either remove the whole, or leave it alone. In desperate, or, I might say, hopeless cases, the kidney might demand drainage for the relief of pain, but I am under the impression that the patient will usually live longer if left alone.

There is no reason why the whole of a tuberculous ureter should be removed, though some advocate it. The incision required would be very large, and such huge rents are a grave menace to the integrity of the abdominal wall: besides, deposits would still be left in the bladder wall. On the other hand, I have myself had cases in which a distinctly tubercular ureter was left and

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tubercular deposits were to be seen in the bladder, and yet these evidences completely disappeared after the removal of the affected kidney, and the building up of the patient's health.

Some might say that recurrence is certain. That is not altogether correct; for, although the probability of recurrence is great, still the same can be said of tubercle in other parts of the body.

I have absolutely no experience of tuberculin in urinary surgery. Urethral discharges are a source of great worry to patient and practitioner. In gonorrhœal urethritis the very best treatment is to put the patient to bed, give salines, plenty of diluents, and encourage him to return to a full diet, free from alcohol, as soon as possible. Locally there appears to be no special virtue in any antiseptic. The silver salts are probably the best, but all injections must be used in weak solution and frequently, say about every three hours.

My experience has been more closely associated with chronic cases of urethral discharge, and, though I use the urethroscope freely, I must confess that ulcerative patches necessitating prolonged treatment do not come under my notice: in fact, in a large number of instances, the patients are suffering from the effects of too much treatment. They will not allow their medical attendant to practise a masterly inactivity, but worry themselves and their adviser until, acting against his better judgment, he treats the urethra. At the same time there are cases where a distinct cause for a chronic urethritis may be discovered. Let me quote two or three cases of chronic prostatitis, where there is a little excess of the normal mucus in the urethra and little whitish casts of the prostatic ducts are found in the urine. These patients often complain of an uncomfortable ache in the perinæum, aggravated by erections or by connection, or by a loaded rectum. Per rectum, the prostate feels somewhat hard and toughish. A linear prostatotomy usually gives great relief, and will almost invariably get rid of all symptoms. It is a pity that more active measures are not adopted with the object of bringing about the early relief of acute inflammatory conditions of both prostate testis and sacculæ seminales, all of which appear to me to be often followed by impotence. I feel convinced that many cases of supposed sterility in women are due to impotence in men; but women bear the brunt of the trouble more easily than men. I have seen patients with purulent urethral discharges which were due to tubercular disease of the vesiculæ seminales, and they have been much annoyed by being told that it must be due to gonorrhœa. A suspicious discharge may be caused by malignant disease, but such a condition is not often seen in malignant disease.

Undoubtedly the most difficult cases to handle are those in which the patient refuses to believe that he is well, and, by persistently squeezing and manipulating his urethra, he irritates the mucus glands so that he is able to express a little glairy material, which the quack trades upon. The consultant is able to deal with this class of patient by giving him a thorough examination, and quietly winning his confidence. The patient receives the same advice from his family physician, but the weight of authority assists the consultant, so that his expressed opinion conveys greater conviction, and consequently the hopeless malady gradually disappears.

Friends of mine have very often asked whether it would be worth while for them to get a cystoscope, or a segregator. The segregator has recently caused a stir among British surgeons, although the instrument has been in use in America and on the Continent for some little time. Probably after a time its use will not be so strongly advocated. The instruments of Cathelin or Luys are the most complete, and act the most efficiently as separators of urines. The segregator is of use only if the urine is perfectly clear. We

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know that experimentally, under favorable conditions, it has been shown that it does separate the base of the bladder into two parts; yet, under most circumstances, we can only conjecture that it does do so, for the simple reason that we meet with cases which the cystoscope will show are positively inadmissible for the use of the segregator. At all events, in cases where the urine is clear, we may by examination discover a considerable difference in the character of the two samples obtained. We may, for instance, discover the presence of a unilateral nephritis, or certain details which would considerably affect the prognosis of the case. If the urine is not clear the segregator, unassisted, gives no definite information. If pus is obtained from one catheter, why may it not come from a patchy cystitis—from a tubercular or simple ulcer? Suppose the segregator acts so as to completely shut off one side of the bladder, and blood-stained urine flows from one catheter only, the blood may come from a ruptured varicose prostatic vessel; I have seen this on more than one occasion. Or it may come from a papillomatous or malignant growth, or from an ulcer near the orifice of the ureter. The fallacies of the segregator must be evident to anyone who will give the subject a moment's thought. If, on the other hand, a cystoscope supplement its use and clearly defines the source of that which alters the appearance of the urine, then the segregator may be of some service as a rough collector of the two urines, inasmuch as we can never swear that it shuts off one side from the other with accuracy. I would like you to clearly understand, therefore, particularly in cases where the urine is turbid from any cause, that the segregator used by itself is apt to be a delusion and a snare.

On the other hand, let us examine for a moment the capabilities of the cystoscope, particularly with reference to those points which the segregator is expected to clear up. Let us deal with a case of proposed nephrectomy. The surgeon's great desire is to know the condition of both the unsound and the apparently sound kidney. Kidneys are usually removed for growth, tubercle, or advanced suppurative disease. Suppose we suspect the presence of growth, the cystoscope will indicate the side from which blood is coming—and renal hæmaturia is very commonly associated with the presence of growth in the kidney. It is extremely rare to find a growth in both kidneys, at any rate in the early and operable stage of its existence, so that if clear urine were seen to be regularly emitted from the other ureter there need be no hesitation in removing the affected kidney. Suppose we are dealing with a suspected tubercular kidney. It is almost invariably to be found that the orifice of the corresponding ureter will show some sign of tubercular infection, and perhaps pus or *debris* will be expelled with the urine. If, at the same time, no pus is seen to be expelled from the other ureter, and if its orifice and the adjacent bladder wall are healthy, one can infer with tolerable certainty that this kidney is unaffected; nor is it likely that any number of examinations of the urine collected from this kidney will show any positive indication of the presence of tubercle if the visible signs I have already mentioned are not present. Again, if advanced suppurative disease is present and an exploration is made with a possible view to complete removal, it is an easy matter to wash out the bladder and watch the other ureteral orifice, in order to see whether clear urine only is discharged.

I very much doubt whether the collection of the separated urines can supply information of greater practical value than that which can be obtained from the careful examination of the bladder and ureteral orifices. A careful use of the cystoscope has enabled me to remove the kidney for malignant, suppurative, and tubercular conditions seventeen times during the past eight years, without a death. Any surgeon, with the same information, would probably get equally good results.

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The measurement of the renal capability is always a difficult task; and every now and again suppression of urine, or a uræmic condition, will be established when we least expect it. On the other hand, the recuperative power of a kidney will occasionally astonish us. We know of instances where one kidney will have been completely destroyed, while the other is a mere pathological remnant, and yet the patient has lived with it. I once operated on a man for a suppurative kidney and drained it. I knew that it was secreting very little urine, as the whole of it came through the loin. After three weeks he had a sharp attack of pain in the opposite kidney and suppression. I cut down on this kidney, removed a stone from the cortex without any difficulty—or I should certainly have left it for a later operation. This kidney secreted nothing for three or four days, but the opposite kidney, hitherto secreting a little pus and urine, roused itself, and urine poured away fairly freely, until eventually both wounds closed and the man recovered.

The accurate collection of the separated urines, whether by means of the ureteral catheter or segregator, is beset with such difficulty in those surgical cases where we most need it that I fear that its efficacy as a practical working method must be very limited. In theory the idea is charming, but as a working method which can be readily adopted I fear it will be found wanting.

TREATMENT OF CEREBRAL HYDATIDS—WITH NOTES OF A FURTHER CASE.

By Alex. MacCormick, M.D.

In the transactions of the third session of the Intercolonial Congress held at Sydney, 1892, Dr. Verco, of Adelaide, in a paper on Cerebral Hydatids, brought the literature of this subject up to that date. According to Verco, six cases of cerebral hydatids had been operated on and published before that date, and out of the six only one had recovered, viz., the case published by Drs. Graham and Clubb. Since 1892 I can find record of only seven more cases, and I append notes of another case, which will make eight cases recorded since 1892. Of these additional cases six are reported to have recovered.

Summarising all the fourteen cases, three methods of operation have apparently been adopted, viz.:—

1. Trephining and removal of a moderate-sized disc: emptying and removing the cyst.

2. Trephining; emptying hydatid cyst with aspirating needle; replacing disc, and, at a subsequent period (about a week or ten days), re-opening the wound and removing the cyst, after opening the dura mater.

3. Osteoplastic re-section of a large portion of bone, fully exposing the suspected area of cerebrum; opening the dura mater and removal of cyst.

It is impossible to estimate the true death rate of the operations for cerebral hydatids, as the successful cases are the more likely to be published; but, in any case, it has been very high. According to the published cases the last two methods are equally successful, but, with further experience, I am more convinced of the correctness of the conclusions which I recorded (*A.M.G.*, November 20th, 1904), viz.:—

1. That, as the diagnosis of hydatids is generally uncertain, osteocutaneous re-section enables the surgeon to explore a larger area of brain surface.

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I would further suggest that in cases where the hydatid is not at once evident on reflecting the flap, or where there is much shock, the flap be replaced and the operation completed in a week or ten days.

2. That this method has the advantage of leaving the skull whole.

3. That the cyst can be emptied without so much risk of infecting the surrounding tissues. It might even be turned out whole, and therefore there would be no need for much handling of the brain for lavage.

In the case here reported I used a guttapercha drain internally, *i.e.*, within the cavity left by the cyst, and a gauze drain outside the dura mater. I used the film of guttapercha tissue internally with the idea of easing tension, should the cavity fill up with cerebro-spinal fluid, as it subsequently did: the gauze outside the dura mater was to drain the serum, which must inevitably accumulate under the bone, between it and the dura. This was apparently a wise thing to do, since the temperature remained normal for five days; and subsequently, when it did rise, it generally came down after a free escape of cerebro-spinal fluid. Apparently it is wise to limit the escape, but not to block altogether the flow of cerebro-spinal fluid—to stop it gradually.

The case is as follows, taken from the House Physician's notes:—J. W. H., aged 12, male, was admitted into the Royal Prince Alfred Hospital on February 17th, 1905—In a "delirious" state. According to the account given by his friends, he had been complaining of headaches for the last fifteen months. They were not severe at first, and did not prevent him going to school; lately they have been more severe. For three weeks he has been blind of one eye, the left, and at the same time a weakness in the left leg has been noticed; one week ago he became blind also in the right eye. Has fits of screaming, and complains of very severe headaches and vomiting. There is marked paresis of the muscles of the left arm and leg. Fundus examination showed marked changes in both discs and retinæ—the discs were blurred, and the vessels indistinct. The head was much larger than normal, but the enlargement was uniform.

Dr. Scot Skirving and myself agreed that there was probably a hydatid in the right Rolandic area, and that an attempt should be made to remove it.

For the following notes I am indebted to Dr. McKelvie, my house surgeon. On February 28th, under chloroform, a large osteoplastic flap—about four inches in diameter—was turned back from the right Rolandic area. The dura mater bulged through the opening in the skull, and showed no signs of pulsation. A whitish area showed through the dura, as if a cyst were underneath it. An incision was made in the dura just inside the margin of the bone, when a hydatid cyst immediately protruded in an hour-glass-shaped fashion. The head was tilted over, the cyst punctured, and the contents emptied into a basin, the tissues being protected from contact with the fluid. The cyst was then lifted out in one piece. The space that was left would more than accommodate a cricket ball. The sub-dural space seemed to be obliterated at the point where the cyst was opened, so that there was no apparent escape of cerebro-spinal fluid at the time of the operation. The dura fell away from the bone around the opening in the skull, so as to leave the bone overhanging all around so much that altogether there seemed to be about a quarter of the space inside the skull empty. The opening in the dura mater was closed with fine mattress catgut sutures, and a fine guttapercha drain placed leading to the cavity occupied by the cyst. A gauze drain was placed in the wound leading to the space between the bone flap and dura mater, when the flap was replaced. The fluid removed contained little white bodies, which were scolices in a living state, as demonstrated by the microscope on a warm stage by Dr. Buchanan. March 1st, 1905—Passed a good night, pulse 108, temperature normal, no vomiting, drain removed.

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March 2nd—Getting on well, pulse 108, temperature normal. A good deal of clear fluid is escaping from the wound, the dressing on which has to be frequently changed. No vomiting. Complains of headache occasionally. Had convulsions on left side of face when being dressed. March 5th—Doing well, temperature normal, pulse between 98 and 100. Occasionally has pain in vertex. Can move his arm and leg much better, sight not much improved, wound looking well. A good deal of cerebro-spinal fluid escapes. March 6th—Temperature suddenly shot up this morning to 103; no vomiting. From the 6th March to the 20th, temperature very variable. On occasions patient dull and lethargic—paresis entirely gone. The temperature generally comes down after a more profuse discharge than usual of cerebro-spinal fluid.

About this date the discharge of cerebro-spinal fluid ceased, and then the wound appeared to be healed. Gradually his intellect improved, but he could only distinguish light from darkness. One month after the operation he was walking about. A few weeks ago—about the end of July—he was reported to be well, but there was no improvement in his eyesight.

INTESTINAL ANASTOMOSIS.

By Alex. MacCormick, M.D.

The following three cases, in which I established an anastomosis between different parts of the intestine, are, I think, of sufficient interest to place on record, as illustrating some of the uses to which this procedure may be put.

Case I.—Mrs. E. J. W., age 25, was admitted into the Royal Prince Alfred Hospital, April 28th, 1897, complaining of a fœcal fistula on the anterior abdominal wall above the pubes. Dr. Brook Moore, of Bathurst, who sent the patient to me, gave the following interesting history:—"I first saw her on February 17th, at her own home, twenty-four miles from Bathurst, in a tent on a mining camp, at 4 p.m. She had, the previous night, been delivered of a dead six-months fœtus: for a month previous to this she had been feeling ill. An unqualified practitioner was called in to remove the placenta. In doing so he evidently pushed his hand through the uterus, and pulled down a quantity of small intestine. When I saw her she was suffering intense pain, and looked desperately ill. I found a large rent in the uterus extending into the vaginal vault, and several coils of intestine hanging into the vagina through the rent: one coil had its mesentery detached for about 8 or 9 inches. I douched the intestines and vagina with hot saline solution, and then reduced the intestine—with the exception of the latter coil, which would not stay back, so I thought it best to leave it where it was, hanging out of the vagina. I expected she would die before next morning. I visited her daily for a week. A large piece of small intestine sloughed off, leaving a fœcal fistula through the torn cervix uteri. She was very ill during this time. I had her removed to the Bathurst Hospital, with the idea of performing an intestinal anastomosis. She had horrible eczema of the skin of the vulva and buttocks. On March 22nd I opened the abdomen, and found many recent adhesions, which bled freely on separating them. I was able, however, after a very tedious dissection to trace the small intestine from the duodenum to the fistula; but the patient began to feel the shock so much, on account of her already weak state, that I decided to hurriedly divide the intestine, leaving a stump of about 3 inches connected with the uterus. I closed the intestine rapidly with a row

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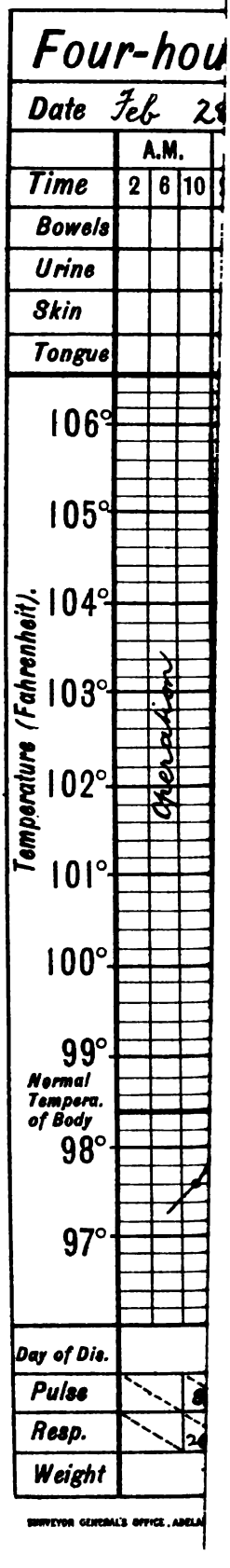

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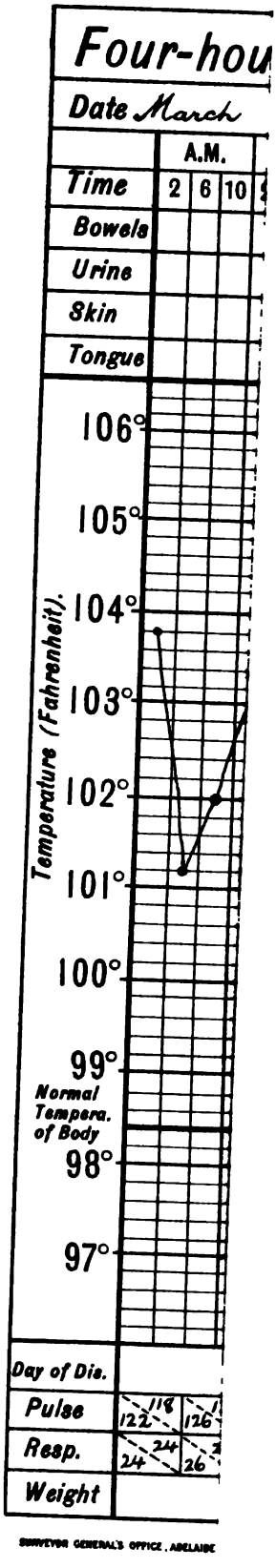

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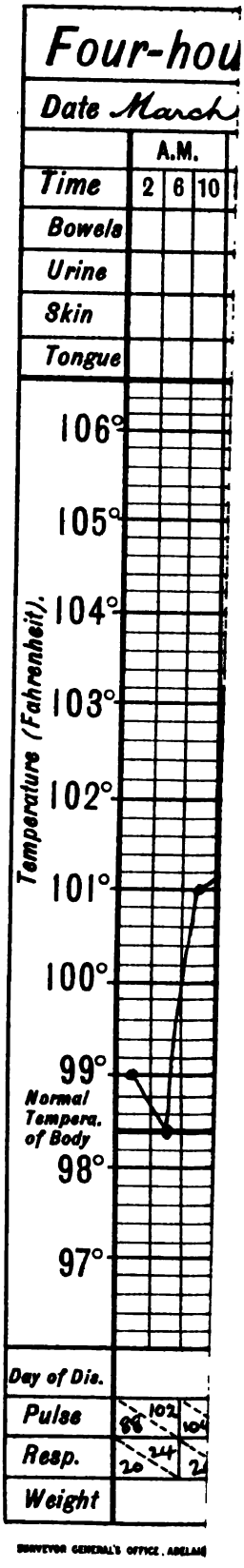

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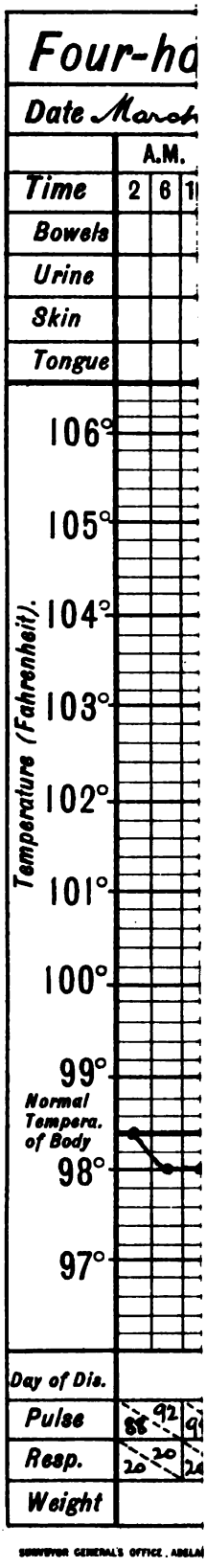

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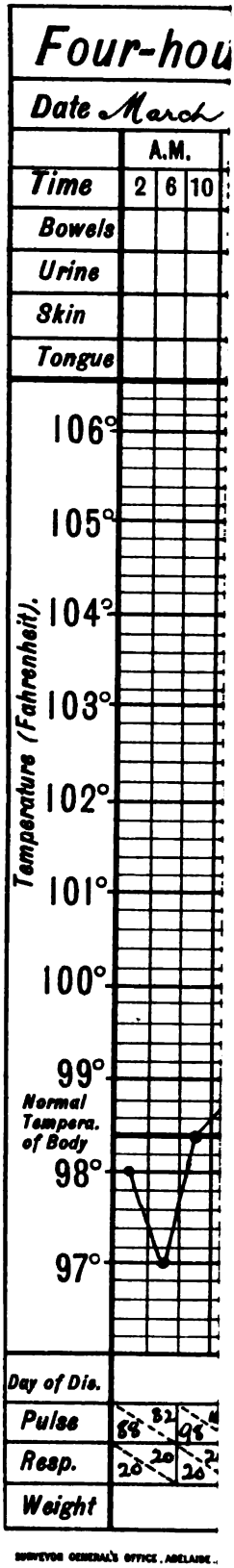

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of Lembert sutures, and dropped it back. The upper end was attached to the skin to form an artificial anus. I recongised that I was doing so in the most unfavorable position; but one had no choice, as it was urgently necessary to cut short the operation. I intended subsequently to perform an anastomosis between the large and small intestine; but the patient left the hospital on account of some disagreement with the nurses."

On examination at the Royal Prince Alfred Hospital, there was a fœcal fistula in the middle line between the umbilicus and the pubes. The liquid contents of the small intestine were constantly escaping, causing a horrible eczema, which extended to her knees. The cervix uteri was transversely torn. On syringing fluid into the rectum, none returned by the vagina. In spite of all this, the patient was fairly well nourished, and appears to have been a fine handsome woman. She was kept lying on her left side, so as to get the right side of the abdomen free of eczema, and, on May 20th, under chloroform, an incision was made over the cæcum, and on opening the abdomen the large intestine was seen to be pale and shrunk, the lower segment of small intestine was short and stretched from the cæcum to the uterus. On passing a bougie into the fistula, the upper segment also was seen to dip down into the pelvis, and therefore could not be readily brought into apposition to the large intestine. One had, therefore, to seek for a freer loop higher up, which was apposed to the front of the cæcum at its junction with the ascending colon, and an anastomosis completed by three tiers of suture, in the usual way. I was anxious about the atrophied and contracted condition of the large intestine —that it might not functionate readily; but I was soon relieved of my anxiety, for flatus passed per rectum within twenty-four hours, and fæces soon followed. The patient left the hospital, wearing a truss over the fistulous opening, in comparative comfort; but subsequently she came back and had the opening closed. She now menstruates regularly, and is apparently in good health.

Case II.—Mrs. B., aged 56, was operated on at "The Terraces" Private Hospital, September 20th, 1903, for a uterine fibroid and double ovarian cyst, and the uterus and ovaries were removed. On account of extensive adhesions, the whole of the raw surfaces could not be covered effectively with peritoneum. A vaginal gauze drain was inserted. Convalesence went on uninterruptedly until October 9th, when the patient complained of abdominal pain and sickness after walking back from her bath. This was followed by free vomiting, by constipation, and increase in pulse rate. On October 11th, all the symptoms of intestinal obstruction became intensified; and, as the usual medicinal remedies failed to relieve the obstruction, and the pulse was getting weaker, I determined to open the abdomen without delay. Under chloroform, the abdomen was opened through the old cicatrix. Coils of intestine were found adhering to the front of the rectum, and as each coil was separated bleeding was free, and had to be stopped by the application of hot saline swabs. Recognising that the patient could not stand too much handling, and that there was no actual strangulation of the intestine, I determined to effect a lateral anastomosis. I traced the small intestine down from the commencement of the jejunum, and found that, after passing about 6 feet through my fingers, it got engaged in the adhesions in the pelvis. I then lifted the lowest loose loop across, and effected a junction between it and the ascending colon—there was only about 5 feet of small intestine above the seat of anastomosis. The abdomen was then closed. The relief was immediate, and the patient was discharged from hospital on November 2nd, and has since remained well.

Case III.—H. T., age 21, was admitted into the Royal Prince Alfred Hospital, February 1st, 1905, suffering from an appendiceal abscess. On the same date the usual "gridiron" operation was performed. There was 1 ounce of foul pus, with a perforation and a fæcal concretion, found. The appendix

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was removed; the septic cavity washed, and packed with gauze. February 6th—Pulse 86, temperature normal, but patient complaining of pain in the left side of the abdomen, high up—no vomiting. February 7th—Pain in the left side is worse, and seems to come in paroxysms, pulse 104, temperature 99°, bowels slightly opened. February 9th—The pain in left side getting worse; general condition not so good; enemata failed to open bowels; no flatus passed; vomited a little clear fluid. Abdomen became greatly distended during the night, and pain all in the left side below costal margin, and going round to the back. As there was evidently intestinal obstruction, and the patient would soon be *in extremis* if not relieved, immediate operation was determined upon. Under ether, the abdomen was opened. The large intestine was empty, but nearly the whole of the small intestine was enormously distended and dark-colored. There were some adhesions in the cæcal region, which were easily broken down, and were evidently the cause of the obstruction. The upper half of the distended small intestine was emptied, by bringing forward a distended coil and inserting a Paul's tube, and, by manipulation, emptying its contents through the tube into a basin. The opening made for the tube was then closed, the intestine washed with salt solution and returned into the abdomen. The lower half of the small intestine was similarly dealt with—the Paul's tube being inserted into an area which was denuded of peritoneum from the latter having given way. When the whole of the small intestine was emptied, enterostomy was performed, and the remainder of the abdominal wound closed. The patient made a steady convalesence; but, as usual in these cases, suffered horribly from eczema of the skin of the abdomen as soon as the Paul's tube came away. On several occasions he had severe attacks of pain in the lower abdomen, probably due to a band of adhesion. He was kept lying on the left side for some weeks, so as to get the skin of the abdomen on the right side into a fit state for operation for lateral anastomosis between the small intestine, above the fistula, and the large. This was carried out under ether on May 11th, the anastomosis being made between the commencement of the transverse colon and the small intestine, 9 inches above the fistula. May 25th—The last abdominal wound is now healed, and patient has been passing flatus and fæces per rectum. Under ether the fistula was closed, and in twenty-six more days the patient left the hospital. July 24th—He has put on condition, and has no abdominal trouble.

The first case illustrates well the power of endurance of a woman during the puerperal period, and Dr. Brook Moore is to be congratulated upon the success of his treatment at the time of the accident. Case No. 2 shows the great advantage of short-circuiting in cases where there are extensive adhesions which one cannot be satisfied of completely separating, so as to get rid of all chances of obstruction. When the bowel is healthy, and the patient in good condition, this method has many advantages over an enterostomy, for which it should be generally substituted. In this case it is possible that the intestine beyond functionated partly. In case 3 the patient was so collapsed that the operation had to be got over quickly. In this case the emptying of the intestine of its contents, so avoiding fæcal poisoning, probably saved the patient's life.

In cases of fæcal fistula with eczema, and a condition of skin around the fistula which will not allow of its being rendered even moderately clean, a short-circuiting operation is very useful; and there is not the same necessity afterwards for trying to ensure a patency of the intestine when closing the fistula. In the first case the blind end of the upper segment of small intestine was closed, and no inconvenience followed.

The method of anastomosis which I make use of is the same as I have been in the habit of using in gastro-enterostomy, viz., three tiers of continuous

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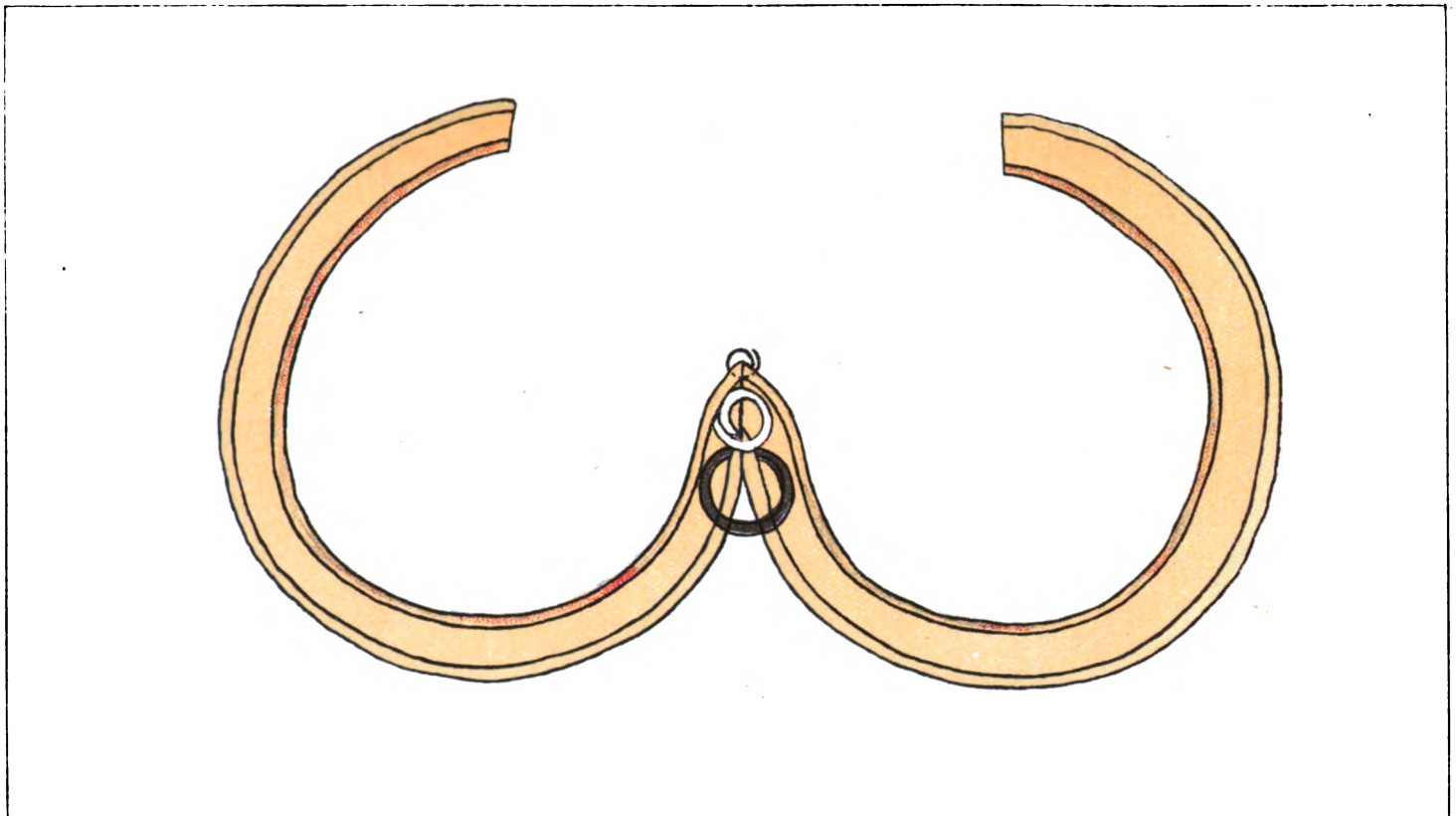

SURVEYOR GENERAL'S OFFICE, ADELAIDE. J. Kingham, Photo-lithographer.

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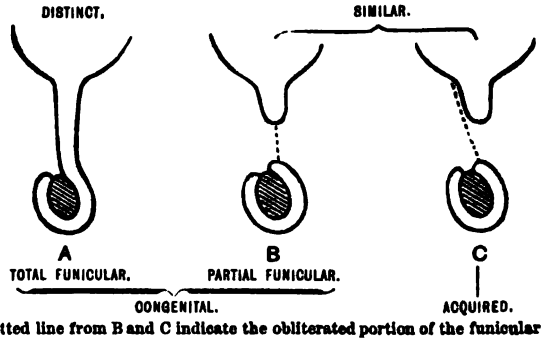
suture—(1) sero-muscular, (2) sero-muscular cut edge, (3) mucous membrane only. The first and second are composed of silk, and the third of catgut. Reference to the diagram will illustrate this method. In case of great hurry, two tiers of continuous suture might be sufficient, but the introduction of a third tier will not take more than five minutes, and it will give much greater security.

THE ETIOLOGY AND TREATMENT OF HERNIA IN ADULTS.

By R. Hamilton Russell, F.R.C.S.

These three diagrams, A, B, and C, represent the three main groups into which oblique inguinal hernia is usually classified. The first two owe their origin to the processus vaginalis. In A there has been complete failure of the obliterative process throughout, so that, should a hernia descend into that, it will pass into the tunica vaginalis; this we call a total funicular hernia. In B there has been incomplete obliteration of the processus vaginalis, so that when a hernia descends it is debarred from passing into the tunica vaginalis;



The dotted line from B and C indicate the obliterated portion of the funicular process.

Fig. 1.

this we call a partial funicular hernia. The obliterated portion of the processus vaginalis may be long, leaving a small hernial sac ready prepared and a normal tunica vaginalis; or it may be short. It may be high, leaving a small sac and a large tunica vaginalis; or it may be low, leaving a large sac and a small tunica vaginalis. It is very often sacculated, and a secondary sac may be placed subpertioneally, when the resulting hernia is called properitoneal; or it may be inserted between the abdominal muscles, giving rise to interstitial hernia; or it may be placed between the skin and the abdominal wall, in which case the resulting hernia has not been dignified by any particular name, but we have been content merely to wonder why it should behave in so unusual a manner. These latter varieties are frequently associated with imperfect descent of the testis, and the ingenious view has been advanced that the retained testis is to be regarded as the cause of these hernial divagations. It would be much nearer the mark to call it the result. The truth is that the retained testis and the laterally expanding hernia are common results of a developmental accident of frequent occurrence. These variations in the saccular arrangement of the funicular process that I have named are only samples; the actual varieties that come under our observation from time to time are almost unlimited in number. The last of our three varieties (C) is called *acquired* hernia; this is quite independent of the funicular process,

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and is caused by weakness of the abdominal wall, which allows the hernia to escape through the inguinal canal, pushing the peritoneum in front of it to form the sac. Thus C is, in origin, quite distinct from A and B; it is produced by a different cause, and will call for a different remedy. Strangely enough, however, as I have pointed out once before, while A and B, which are identical in origin and nature, are easily distinguished clinically, B and C, which are quite diverse in origin and nature, are yet virtually indistinguishable from one another; so that the surgeon, every time he meets with a case of hernia in which the sac is distinct from the tunica vaginalis, is quite unable to tell whether he should be guided by the assumption that its cause is a congenital sac (B) or a weak abdominal wall (C). It is therefore not surprising that a large number of devices should have been instituted for the cure of hernia, all of them designed to meet either contingency, somewhat in the manner that has been not altogether unknown of putting several drugs, each appropriate to some special indication, into a bottle, and labelling it "The Mixture." This, I need hardly say, is not a very good plan, nor is it likely to yield results that are altogether satisfactory. I would further point out that all the operations that have been devised have been, in the main, prompted by the supposed necessity of meeting the case of class C; and there is not the slightest doubt that the prevailing opinion has been that the predominating type of inguinal and of all other forms of hernia is the acquired type, and that the congenital forms, A and B, although quite well recognised, are merely of the nature of pathological accidents peculiar to the inguinal canal. Thus it has come about that all operative procedures have comprised two elements—first the removal of the sac; and, secondly, some device for strengthening the abdominal wall by stitching it up in various ingenious ways. Now, with respect to these two factors in the operation, I wish particularly to put to you this: The first step, the removal of the sac, has been regarded as a very simple matter, an obvious and necessary incident in the operation; whereas the matter of chief importance is the method of closing the canal so as to restore its valvular mechanism (whatever that may mean), and upon this project much thought and ingenuity has been expended. I wish to-day to review this matter of the "Radical Cure of Hernia," and perhaps I may succeed in arresting your attention with some considerations not altogether unworthy of your notice.

The only place in my opinion in which the etiology of hernia can be effectively studied in the first instance is a children's hospital. In an adult hospital it cannot be done; nor do the deadhouse and the dissecting-room help us very much in our primary investigations, though they will often be found to afford interesting and valuable confirmatory evidence. I shall not attempt on this occasion to re-state the arguments that I have already adduced in favor of the views as to the etiology of hernia that I believe to be correct. Suffice it for me to state simply that in children acquired hernia (class C) does not exist.

Six years have elapsed since, at the Brisbane Session of this Congress, I first pointed out that acquired hernia in childhood was a myth, and a myth that had proved highly prejudicial. The view was put forward that inguinal hernia in a child is invariably dependent upon the presence of a congenital sac, which, in the great majority of cases, is provided by some portion of the processus vaginalis. It was contended that if the sac be completely removed from the inguinal canal, there will be no return of the hernia; that, moreover, by this operative procedure alone can the hernia be cured; that the so-called curing of hernia by the use of a truss is a deceptive misnomer; and, as a practical corollary, that operative removal of the sac should be advised and carried out in every case of hernia in a child. It must be confessed that these views seemed rather sudden and shattering at the time; nevertheless,

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I think that their correctness has, in these intervening years, become established in the opinion of so very many of those best qualified to judge that they will henceforth need no further advocacy from me. The views which six years ago seemed strange and provocative of a very natural feeling of incredulity are strange no longer. That which does appear to me strange when I now look back at the occasion to which I have alluded is that the above views should have appeared, at that time, only appropriate to one form of inguinal hernia—the oblique inguinal of childhood; for the oblique variety of inguinal hernia was deliberately contrasted with all other forms, such as the direct inguinal, the femoral, obturator, and other varieties, all of which I, at that time, supposed to be of the acquired type. Subsequent opportunities for observation have, however, shown, as it seems to me, quite unmistakably that this view was erroneous; and in a series of papers that have been published during the last few years—one of which papers was read at our last session at Hobart—I have advanced reasons for believing that acquired hernia, other than traumatic hernia, has no existence, and that the presence of some sort of congential peritoneal pouch is an essential condition for its occurrence. The correctness of this view, in respect at any rate of the hernia of childhood, is, in my opinion, no longer open to question. By this I by no means imply that it is universally accepted; I am, however, absolutely confident that it has already acquired so strong and increasing a hold on influential surgical opinion that it may be left now to take care of itself. I, accordingly, do not intend to deal other than incidentally to-day with the hernia of childhood; my subject is the hernia of adult life, its etiology and operative treatment. As you will naturally expect, however, I shall approach the subject from the standpoint afforded by the information we have only recently acquired as to the etiology of hernia in childhood.

The first great question that arises, and that I shall endeavor to answer, is this—What etiological difference, if any, is there between the hernia of childhood and that of adult life? Or, to put the question in another way—Is the hernia of adult life always, as in the case of children, dependent on incomplete closure of the funicular process, or some other form of pre-existing sac; or do we meet with genuine acquired hernia in adults? How does adult hernia answer in the tests that have sufficed to satisfy us as to the congenital nature of hernia in childhood? The evidences of the congenital origin of the herniæ of young children are redundant and overwhelming; but there is one test that far outweighs all others, and that would be absolutely incontrovertible even if all the other evidences were withdrawn. This is the evidence afforded by the operation of simple removal of the sac, as proving effective in curing the hernia. I repeat that, if there were not another particle of evidence forthcoming as to the congenital origin of hernia in childhood instead of the abundance of it that we have actually found, the evidence afforded by the results of operative removal of the sac would be all-sufficient, and could not be successfully opposed by any line of reasoning that I can conceive. Can we, however, employ this test in the case of adults? It is obvious that a grave complication is introduced in many cases of hernia in adults where the hernia has existed for a long time, through the stretching and permanent weakening of the hernial opening. It is scarcely necessary for me to remark that an acquired hernia will develop at any situation in the abdominal wall, provided the musculature be sufficiently damaged and weakened at that spot. We should, therefore, expect, *a priori*, that if we were to remove the sac in a large number of cases of adult hernia, indiscriminately collected, guided by the same principles that have served us so well in the case of children, we should, in our adult operations, have a very large percentage of recurrences.

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It is therefore clear that, should any surgeon treat a large number of adults by simple removal of the sac, on the assumption that the etiology of adult hernia is identical with that of childhood, he will, in his adult work, be carrying a very heavy handicap, which he escapes in his work among children. On the other hand, should he succeed, even in spite of this handicap, then its imposition will have only availed to enhance the value of the evidence, and to render the proof of the correctness of his principles the more convincing. Before giving you, in very simple form, the statistics of my operative experience, we must first come to some agreement as to what we are going to accept as evidence of success. After what lapse of time may we conclude that all fear of recurrence is past, and write a case down as a successful operation.

I am in entire agreement with Deanesley, and I think most surgons are, in respect of this matter. Recurrence after operation, when it occurs at all, does so, in the vast majority of cases, within six or eight months from the date of operation. I have never met with a case of recurrence supervening after a longer period had elapsed, and I am sure that such an occurrence must be quite rare. I therefore propose that we should take a year's immunity from recurrence as our standard of success, always recognising that, even although possibly not an infallible standard, it will, for all practical purposes, satisfy our demand for a fair working estimate of the value of any particular method of operating. I therefore include in my statistics the cases of adults who were operated upon more than one year ago. Of eighty-nine such cases, I have lost sight of twenty-six, whom I have failed to trace. The others—sixty-three in number—I have recently seen or heard of, or otherwise known about. Of these sixty-three, recurrence has taken place in three. One was a woman with an ordinary inguinal hernia, which recurred almost immediately she got up after the operation. Evidently I had blundered badly in some way. The second was a man with a very unusual kind of hernia, which I wished to examine with much deliberation. Unfortunately, he took the anæsthetic badly, and I was unable to satisfy myself that the operation had been efficiently done, and I predicted that recurrence would take place; this happened in about five months. The third and last case was specially interesting. A man, *æt.* 62, of poor physique, in whom a left inguinal hernia appeared for the first time, and became at once strangulated. He was promptly operated on, and the sac removed. Some five months later he came back to me with a commencing recurrence. I remarked at the time that this case appeared to be really one of genuinely acquired hernia. The hernia had appeared at the age of 62 for the first time; it had been at once operated upon, and the sac removed; the abdominal wall had suffered no damage from the prolonged existence of the hernia, and yet the hernia began to recur a few months later. He was a flabby, pallid individual, and I could come to no other conclusion for the moment than that we had to deal with a case of real acquired hernia, the only instance that I had ever seen reason to regard as such. Nevertheless, I was wrong, for on referring to the notes of the operation I found that my careful house-surgeon, Dr. MacKeddie, had made a special note to the effect that the case was one of total funicular hernia, *i.e.*, hernia into a totally patent funicular process. The explanation of the recurrence is, doubtless, that, under the circumstances surrounding the operation, I was less careful about getting to the upper limit of the funicular process than I ought to have been. In addition to these three cases, I must mention that two other of my earlier cases had a transient recurrence, which was cured by the use of a truss for three months. Both of these cases occurred more than five years ago, when I had but few opportunities of operating on adults, and when my methods were not so thorough as they are now. On one of these two cases I operated

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a second time, and found only a portion of the bladder in the canal. Nothing was done, and the hernia never came down afterwards; and, after wearing the truss for three months, it was discarded, and the patient has remained perfectly well for the last five years. Those are, in brief, my statistics of operation in adults. In all cases the operation has consisted solely in thorough removal of the sac; in no case has the canal been closed by sutures of any kind, except in one or two of the earliest ones. The list includes subjects of all ages, and herniæ of various duration. The oldest was a man of 75, with a large irreducible hernia of thirty years' standing. One fatality must be chronicled. An old man, with a very large double irreducible hernia, who gave his age as 74, but it subsequently transpired that he was probably over 80. It will be noted that, while the principle of operation adopted in all these cases of adult hernia has been identical with that which we have followed in the case of children, the results have been, to all intents and purposes, identical, in spite of the heavy handicap to which I have alluded. I would further state, in general terms, that the various signs of congenital origin which I have been accustomed to note in the case of the herniæ of children have been noted with equal frequency and consistency in the case of adults. Nevertheless, it must be admitted that the stretching and alteration in the structures that is displayed in herniæ of long standing will conceal these evidences unless they are very carefully looked for by a practised eye. It must also be remarked that we meet with occasional borderland cases, which begin with a very small sac, in which the clinical history and the appearances observed are, perhaps, indistinguishable from those that would be presented by a genuinely acquired hernia. It has been a matter of surprise to me that such borderland cases are not more common than they have proved to be in my experience. If now the question to be put as to whether the hernia of adults is identical in mode of origin with that of childhood, or whether, on the contrary, adult hernia is sometimes acquired, I think I shall be held justified, and shall be assuming a sufficiently guarded attitude, if, in the light of the facts I have detailed, I answer the question in the following manner:—

I have never met with a case on the operation table that I have seen any reason to regard as being a case of genuine acquired hernia; all my experience in the surgery of adult hernia has been such as to force upon me the conclusion that there is substantially no difference between the hernia of adult life and that of childhood. This being so, however, I quite recognise that the possible occurrence of rare exceptions is not excluded thereby, and I am by no means unmindful of the immense operative experience which, at the present day, has fallen to the lot of a few favored surgeons, contrasted with which my opportunities for observation have been relatively trivial. It may be that, in the case of elderly people who have put on much adipose tissue, hernia is occasionally acquired. From the practical standpoint, however, I would urge that such rare exceptions, assuming that they exist, may be disregarded, for the conditions of their occurrence would be such as to contra-indicate an operation of expediency, and any such operation would be sure to fail. We shall, on the other hand, not be misled if we take, as a working hypothesis for all forms of inguinal hernia, the view that it is dependent on the presence of a pre-existing sac. Assuming then, as I believe we may do with perfect safety, that the underlying cause of hernia in the adult is the same as that which is efficient in the child, the practical question arises—"Is the treatment also identical; and will the same method of operating suffice for the adult that is found to be efficacious in the case of young children?" My answer to this question is that the principle holds good, and that simple removal of the sac in adults of any age, and in hernia of any duration, is very nearly, if not quite, as efficient and satisfactory as it has proved to be in the case of

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children. But it has to be noted that there are very important anatomical and clinical differences presented by adult hernia when contrasted with that seen in children; and these differences, while in no way affecting the principles that govern our operative treatment, demand important modifications in detail. We will therefore briefly consider these differences, and the methods that seem appropriate to their management. In the first place, by reason of the length and obliquity of the canal in adults, it is not possible to get at the peritoneum at or above the region of the internal ring without first slitting up the external oblique; and this should always be done in adults, although in children it is not usually necessary. This, then, is the first great difference. The second is dependent also in part on the length and obliquity of the canal, in conjunction with the results entailed on the structures forming it by the prolonged existence of hernia in the case of elderly people. These results are progressive stretching and dilatation, until the canal becomes converted into a large hole in the abdominal wall. The kind of thing referred to is represented in Fig. II. We all, I think, recognise the appearance as that presented when

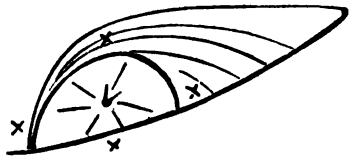

Fig II.

the sac has been tied off in the ordinary way prior to the completion of the operation by Bassini's or some other method of suturing. I used to think—in fact, I expressed the opinion at our last meeting of Congress—that the best way of operating in such a case as this is by Bassini's method, by which the muscles are brought down by sutures to cover the large exposed area of peritoneum. I have since then altered my mind; and I would point out now, what I must confess it took me a long time to discover, that the presence of an exposed area of peritoneum and fascia, like that represented here, is, *ipso facto*, proof that the sac has not been properly removed, and for this reason: The mouth of the sac is situated normally under cover of the muscles, and any portion of the peritoneum that is seen to emerge, and come to light below the muscular border, must be regarded, for operative purposes, as a portion of the sac. It will therefore follow that the whole of the area of exposed peritoneum in reality belongs to the sac, and should have been removed. On the other hand, if our ligature, instead of being applied in the way that is here represented, had been made to encircle the exposed peritoneum in such a way that, when tightened, the periphery should have been made to converge centripetally into the knot, it is obvious that the area of peritoneum would have disappeared almost entirely, and the opening in the abdominal wall would have been closed, or at least so nearly closed that we should no longer experience any pressing desire to close the opening by sutures. The actual technique employed in applying the ligature is a matter in which some choice is open to us. A purse-string suture will doubtless answer very well; or we may boldly cut away the entire area of peritoneum and suture, taking care, in any case, to include the fascia. The method I personally prefer is a special form of knot of my own devising. This knot has four fixation points, each point being indicated in Fig. II. by a cross. Each cross indicates a spot under cover of the muscles where the ligature fixes the peritoneum and fascia. When the ligature is tightened, each of these four points is brought convergently into the knot.

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There is one other matter of technique to which I must allude, and that relates to the clearing of the sac preparatory to the application of the ligature. Where the hernia has been of long standing, the peritoneum of the inguinal region becomes lax and voluminous, and the redundancy should be included in the ligature and removed. This is done in the following manner:—The left index is passed through the mouth of the sac into the abdomen, and the abdominal wall lifted up on the finger and pushed back from the sac. Externally, it is pushed back while the first step in the fixation of the ligature (at the external fixation point) is effected; above, the curved fibres are drawn well up, while the ligature is passed through the fascia and peritoneum at that spot. The most important part of this stage of the operation relates to the inner part of the mouth of the sac. Here the peritoneum is very lax; it is lifted well up on the finger, and the epigastric vessels and subperitoneal fat are pushed inwards. What appears to be a secondary sac will now make its appearance on the finger. This is really the peritoneum of the internal inguinal fossæ. Frequently the bladder will appear, and some of the peritoneum can be readily stripped off that viscus. The whole of the peritoneum thus isolated may be now included in the ligature and removed, and the operation concluded by suture of the incision in the external oblique. It will be obvious that this method of operating relies for its efficiency, first, on the removal of all lax peritoneum from the inguinal region; and, secondly, upon closure of the opening in the abdominal wall by concentric traction upon the peritoneum and fascia, as distinguished from the usual method of drawing down the muscles by suturing them to Poupart's ligament. I venture to think that no one present will have the least hesitation in arriving at a decided conclusion as to which is the more correct principle of the two. But it will doubtless be asked, and with a great show of reason—Why not close both the internal and external layers? Why not add the suturing of the muscles to the tying off of the peritoneum in the way I have advised? I am far from saying that there is any particular harm in putting one or two sutures into the walls of the canal in the way that has been customary; nevertheless, I think that if surgeons conclude that the principles I have enunciated are sound principles, and that the practice I have described as based upon those principles is to be trusted, there will be but little inclination to suture the walls of the canal. Personally, I do not put any stitches into the muscles, because I am convinced that they are unnecessary, and that the principles of treatment point altogether elsewhere for the means of preventing recurrence. And if sutures are unnecessary, their employment cannot be justified on the ground of their being harmless.

There are several reasons, none of them being perhaps of great individual weight, but collectively forming a sufficiently strong indictment against the use of sutures in the muscular wall to at least give us pause. Here are two of them—

(1) In the event of suppuration occurring (and such an accident will occur in the practice of every surgeon occasionally), I believe the sutures will do great damage to the musculature, and will greatly tend to promote recurrence; whereas if sutures are not used, the accident of suppuration will in no way impair the result; some surgeons are inclined to think it might even be improved thereby.

(2) In the event of recurrence taking place after any of the suturing operations commonly employed, any subsequent operation will be greatly hampered, and the chances of success greatly diminished by the altered arrangement of the parts brought about by the suturing; whereas, on the contrary, where the procedure adopted has been that which I advocate, the conditions for a second operation in the event of recurrence will be in no way affected prejudicially.

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It is obviously impossible for me, in the time at my disposal, to attempt to discuss in detail the management of the numerous varieties of inguinal hernia that we meet with. There are, however, one or two features of the subject to which I should wish to allude. When we say that the hernial sac is congenital, or to use the term I prefer, "of congenital origin," this does not imply that the whole of the sac, as it appears at the operation, was present at birth. A small sac is always susceptible of being rapidly enlarged, up to a certain point, by virtue of the looseness of the peritoneum in the inguinal region. The hernial sac will therefore be composed of the original congenital pouch, plus an added portion. I have illustrated this point by these two diagrams (Fig. III., A and B). This "added portion" is of great importance

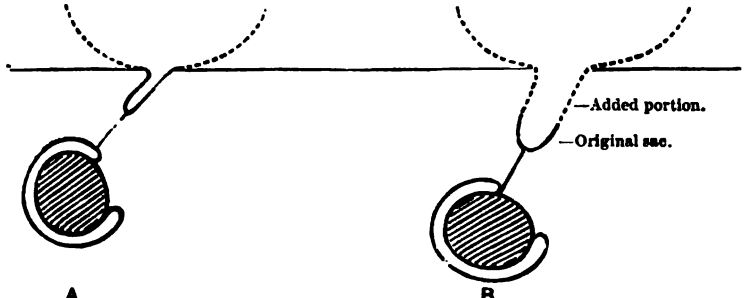

FIG. III.

from two aspects—first, it incidentally provides some remarkable and very important varieties of inguinal hernia; and, secondly, it plays a prominent part in facilitating the manipulations of the operator engaged in removing the sac.

Let us consider this latter aspect of it first. When the sac has been exposed, and the separating and isolating process is being carried out, we are enabled to increase the length of the sac very materially by pulling down some of the peritoneum which, prior to our proceedings, was in the abdomen. If now we apply the ligature to this "added portion" and cut off the sac, the ligature will spring back into the abdomen well above the muscles of the abdominal wall. We, shall in fact, have applied the ligature intra-abdominally. Now, that which we are enabled to do, by pulling on the sac while we are operating, may also be effected by the pressure of a hernia so soon as it has completely filled the sac. So that if the sac be small in the first instance, a process of pulling down the abdominal peritoneum so as to enlarge it will be rapidly accomplished, and the sac will be rendered much larger than it was originally. This power of adding to the original sac, by inducing the descent of a portion of the abdominal peritoneum proper, is not only of extreme utility to the operator, but, when it occurs as the result of hernia, it may be, and frequently is, fraught with exceedingly important and interesting results. It must be remembered that the peritoneum forming the funicular process is, virtually, a portion of the same structure that lines the pelvis, and has intimate relations with the pelvic organs; and it is in complete accord with all our experience of developmental accidents that, in the formation of the funicular process, it should sometimes happen that contiguous pelvic and abdominal organs should be implicated in its formation, and included with it in the inguinal canal. In this way a portion of the bladder may enter the canal on either side; while on the right side may be found the cæcum, and on the left side a portion of the colon and sigmoid flexure. In the female, the ovary and Fallopian tube, and even the uterus, may be drawn in. Any of

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these organs may be actually present in the canal at birth; or, short of that, they may be congenitally disposed in such close proximity to the mouth of the sac that, should hernia occur, they will be at once drawn down into the canal with the "added portion" of peritoneum. When this occurs the particular viscus implicated—cæcum or bladder or colon, as the case may be—may make its initial entry into the canal at any period of life. It is obvious that when this condition is brought about, the operation will be complicated, for the application of the ligature to the neck of the sac will be interfered with. There are, as usual, two ways of meeting the difficulty, a right and a wrong way. The method which, I think, is frequently employed is to ligature the sac as high as pos ible (*i.e.*, below the point of fusion of the intruding viscus with the sac), then to close the canal by suturing according to the surgeon's fancy. This is the wrong way, and is pretty certain to be followed by recurrence. The right way is to cut out from the sac the portion of, say, the large bowel that interferes with the application of the ligature, return it into the abdomen with its attached portion of the sac, and then ablate the remainder of the sac by ligaturing it at the highest accessible point. If the sac be treated in this way, the chance of recurrence will be exceedingly small. I have adopted this expedient in five cases, of ages ranging from 5 months to 68 years. In no case has recurrence taken place, although all, with the exception of the youngest, were operated upon more than three years ago. Where the bladder is the viscus in the hernia, the manœuvre I have just described is unnecessary, as the peritoneum can be readily separated from it.

Another class of complication that may occasion perplexity is caused by variations in the anatomical arrangement of the sac—varieties of sacculation. These are most commonly, but by no means exclusively, seen in association with imperfect descent of the testis.

I have already referred to the most notable varieties of hernia that arise in this way, the interstitial and properitoneal forms, chiefly remarkable for their having been the joint heroes of the most romantic etiological stories that even the voluminous literature of hernia is able to show. Another not uncommon form of sacculation is that represented in Fig. IV., where a pouch

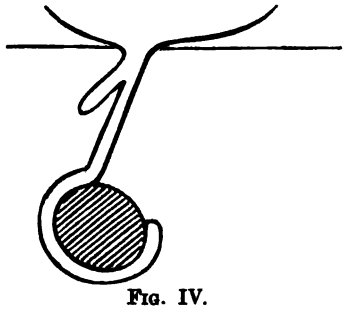

FIG. IV.

appears high up on the inner aspect of the funicular process. This will closely simulate a direct hernia if the bowel should descend into it. I have twice seen hernia occur in manhood into such a sac while the funcular process was open, but empty. Again, doubtless the funicular process might be closed, leaving the pouch open, giving rise to an anomalous form of hernia, apparently non-funicular in origin. I have never met with such a case, however. The varieties of pattern displayed in the sacculation of the processus vaginalis are unlimited; they will, however, but rarely cause any great perplexity if the surgeon is imbued beforehand with right views as to their origin and

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nature. If the funicular process is ligatured in the proper place and removed, all the sacculations, of whatever particular arrangement, will be removed also.

Time has permitted me only to indicate here and there a feature of this very wide subject, while many others I cannot even allude to. I should have liked to allude to some of the better known and more popular methods of operating, such as Bassini's and Kocher's. On the whole, excellent as is Kocher's method of treating the sac, I am inclined to think that the method of removal that I have described satisfies the requirements that, to me personally, appear most important more completely than any other plan. I do not wish to appear intolerant about the practice of suturing the walls of the canal; it cannot, as a matter of experience be so harmful, because such excellent results are obtained by (or in spite of) it. There are, however, certain theoretical considerations which I can find no sufficient reason for disregarding. One great object that we wish to attain is to strengthen the musculature enclosing the inguinal canal. It has even been suggested that hernia should be treated by local massage and exercises with this aim in view, a suggestion which is absurd, because it ignores the predominant part played by the sac in the causation of hernia. The strengthening of the musculature, however, being an obviously desirable end upon which we are all agreed, I fail to see how the stitching of the muscles to a tendinous structure is likely to strengthen them. The principle is, in my humble opinion, entirely fallacious and wrong, although I must own to a great disinclination to express myself thus about a proceeding which is systematically followed by a number of surgeons for whose individual ability and judgment I have far more respect than I have for my own. If, however, the principle is wrong, can the practice be accounted at least harmless? If it can, then it furnishes the only instance known to me, either in the domain of surgery or in any other of our mundane affairs, in which the systematic following of a wrong principle yields results as good as the systematic following of a right one. Furthermore, I would urge, what I know from observation to be no mere theory, that the surgeon who holds what I may term the generally accepted views as to the causation and treatment of hernia will remove the sac in a very different way from the surgeon who is convinced that the sac is the cause, that everything depends on its efficient removal, and that the abdominal wall has nothing to do with the matter, and may be disregarded. I am therefore unmoved by the contention that removal of the sac is a feature in all operations for hernia; it is not sufficient that it should be a feature of the operation—it must be the operation. I am sometimes asked some such question as this—"Is it true that in operating for hernia you simply remove the sac?" Simply removing the sac sounds very easy; as a matter of fact, it is by far the most elaborate and difficult operation for hernia that has ever been devised, and all other methods are child's play in comparison with simple removal of the sac, if it be efficiently and thoroughly carried out.

Finally, I would observe that a correct view of the etiology of hernia is everything, and, unless we can attain to that, a secure pathological basis for our work will be lacking. An insecure pathological basis for our theory and practice will mean, in the future as in the past, a multiplicity of operations, all of them more or less successful, none completely trustworthy; it will mean a most pernicious acquiescence in failure, so that when our operation fails we excuse ourselves by saying that the patient has a congenitally weak abdominal wall, that it is impossible to strengthen it, and that it is an error in terms to speak of the "radical cure" of hernia.

Turning from the practice to the literature of hernia, we find that an insecure pathological basis has been productive of the most extraordinary looseness and inaccuracy. We have not even taken the trouble to adopt an

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ordinarily rational nomenclature, as witness the custom of naming these two elementary varieties A and B, Congenital and Funicular; whereas they are, of course, both equally congenital and both equally funicular. Furthermore, an insecure pathological basis has conferred exemption from criticism, for a critic will rarely be found ready to find fault while conscious that his own pathological basis is insecure. Hence the custom has grown up among authors of writing things that would certainly never have seen the light but for this security from criticism of which I am speaking. On the other hand, a secure pathological basis will mean a single operation or, at any rate, an established and immutable principle for the guidance of the surgeon. It will mean a refusal to acquiesce in failure, so that when recurrence takes place after operation, instead of comforting ourselves with the patient's congenital muscular weakness, we shall put to ourselves the question—"What mistake have I made?" While, if we turn again to the literature of hernia, we shall find ourselves suddenly subjected to restrictions unknown heretofore, and we shall no longer enjoy the privilege of romancing—a privilege of which we have been accustomed to avail ourselves rather liberally. To aim at these results is, at least, a high and worthy aim, and I shall be abundantly gratified if the few facts and suggestions I have been permitted to offer should seem to you to contribute, however modestly, towards the right kind of progress in this important department of surgery

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DISCUSSION ON HERNIA.

H. S. MAITLAND: I have listened with very great pleasure to Dr. Russell's views, more especially so as they coincide with my own. I regard obliteration of the sac as the main factor in the cure of hernia in children and the young adult when the obliquity of the canal persists. In the older man, with protuberant abdomen, where the conjoined tendon is atrophied, I do a Bassini's, but I only operate on these older people if for some reason they are unable to wear a truss.

Dr. Russell speaks of a hernia in which the bowel forms part of the sac, and suggests dividing the sac on either side, and returning the gut: but I would like to sound one word of warning against treating the sac so in a hernia par glissament or sliding hernia, because such a method might possibly cut off the blood supply to the bowel, with disastrous consequences. I have no doubt that this is sometimes the cause of those cases of fæcal fistula which are recorded after hernia operations.

DR. STEER BOWKER: I have been greatly interested in Dr. Hamilton Russell's paper, and will certainly on some future occasion try it, and will get him to show me how he places his puracotomy, though I do not consider one year is nearly long enough to judge of the risk of recurrence.

For my own part I am well pleased with the results of the operations that I have been doing since I was assistant to Dr. McCormick some years ago, the first idea of which I got from him, and have added to and altered since, when I have assisted at the operation afterwards. It is exactly the operation described in Bryant's book, recently published, and called "Halstead's operation." Halstead's operation used to be after the manner of a Bassini, both of which operations I have always considered to be bad operations, and have always dropped the cord in and not displaced it outwards; for if there is a recurrence, it always takes place where the cord has been brought out in either a Bassini or a Halstead. The operation I do, and which is now claimed by Halstead, differs absolutely from the old Halstead or Bassini, and is made up from points which I considered good in various operations—even to the imbrication of the external oblique at the close of the operation.

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APPENDICITIS.

By Critchley Hinder, M.D.

Appendicitis is an extremely well-worn subject; but, none the less, it would certainly be a very extraordinary thing if we were all of one mind in respect to many matters in connection with this common and dangerous malady. In the limited time at my disposal I shall endeavor to deal with those aspects which appear to me to be likely to excite the greatest amount of interest.

First, let us deal with the cause of appendicitis. Every case of appendicitis is, as far as we know, associated with the presence of organisms, and these organisms are similar to those which normally inhabit the intestinal canal. The degree of severity of the attack depends, in a large measure, on the virulence of the particular organism.

In order that any organisms may be able to run riot with the body cells it is necessary that they, as an attacking force, should be of greater power than the defending cells. In appendiceal infections it happens very rarely that the organisms already within the intestinal tract have acquired an increased degree of virulence, although such cases will occur in different forms of enteritis. I think, then, that the cause of the beginning of the trouble lies in that which diminishes the tissue resistance of the appendix itself. In a very large percentage of cases in which the appendix is removed for appendicitis, one or more concretions will be found within its lumen. I have only on four occasions found a foreign body, nor has an examination of the appendix disclosed the presence of any massive pieces of undigested food stuffs which might have passed in from the cæcum. The concretions varied in consistency, and some have been too hard to be broken in the fingers, and, in fact, appeared to be gall stones, but a careful examination showed that this was not the case; and the impression conveyed by the varying degree of solidity is that they, like gall stones in the gall bladder, have been gradually formed within the appendix.

When operating for the removal of an appendix about the third or fourth day, when the clinical symptoms induced one to believe that the attack was subsiding, I have noticed that the appendix gave me the impression that it had recently been distended. Again, every appendix which is removed during the middle of an acute attack will be found to be either greatly distended or ruptured. I think, therefore, that we would hardly be wrong if we were to assume that an inability of the normal secretions of the appendix to escape into the cæcum is the cause of a very large percentage of cases of appendicitis. Under normal conditions the secretion escapes readily: under abnormal conditions the secretion escapes with difficulty, and the residuum becomes more solid and forms a concretion, even as a concretion is formed in the gall bladder or in the urinary bladder when it is unable to completely empty itself. Directly the concretion comes into existence it acts as a foreign body, and excites inflammatory changes in the wall of the appendix—catarrhal at first, then ulcerative, and finally perforative. The perforations are very often indeed right opposite the concretion. The gangrenes are often similarly situated; but, at times, a considerable area is affected, and this can be readily explained by the great tension which exists within the appendix or by a thrombosis or arteritis being occasioned by the intense activity of the organisms, which have gained in power and virulence owing to the marked diminution in vitality following upon the primary increased tension. It is possible that an attack may be occasioned by thrombosis or arteritis of the mesentric vessels, for such a rare condition occurs in connection with intestinal vessels; still

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those who operate largely on these cases must have been impressed with the very overwhelming proportion in which an increased tension is found within the appendix which is about to be removed during an acute attack.

To sum up, I maintain that obstruction to the outflow of the appendiceal secretion is most usually the cause of acute and chronic appendicitis. Certainly, to the clinician, the many different kinds of appendicitis so often spoken of are simply unrecognisable.

Next let us inquire into the probable cause of the obstruction. Kinks and twists are said to be common. These kinks and twists must be either congenital or acquired. If the kink is congenital it is possible that it may become more marked as the child grows and its abdominal contents alter in character. If the kink is acquired it will be found associated with inflammatory features in its immediate neighborhood. If in the pelvis, in women, pathological changes occurring in the genital organs will be quite sufficient to account for the kinking of the appendix. Typhlitis or peritphlitis is not to my knowledge particularly common, so that I should not like to say that it could be the cause of more than a very limited number of attacks. In fact a great number of the kinks and twists found in an appendix are caused by previous attacks of appendicitis.

We have now got rid of the probable causes which act from without. In examining appendices which have been removed during acute or chronic attacks it will be observed that, although there are no twists or kinks present, nevertheless a constriction exists—or maybe there are two, or even three, constrictions. The most constant constriction will be found at, or very close to, the cæcal extremity. These constrictions are congenital, and may be looked upon as evidences of a similar condition existing in the lower animals. It would perhaps be absurd to assume that this alone is the cause of an attack, but where the lumen of a mucus canal is narrowed it is surprising how little it will take to effectually block it. We have evidence of this in the ease with which an extra glass of alcohol will block the urethra of a man suffering from stricture, or hypertrophy of the prostate. We know that the constriction and the retention of the appendiceal contents is remarkably common, so that the least amount of swelling of the mucous membrane at the neck of the appendix defines the onset of an attack. It is probable that constipation, or diarrhœa, will cause some swelling of the mucous membrane of the cæcum, and produce sometimes a partial and sometimes a complete closure of the canal. I am inclined to think that dyspeptic attacks, obscure abdominal pains accompanied by malaise and a general feeling of unwellness, are very frequently due to a chronic appendicitis and consequent absorption of septic products. It is said that constipation is a common cause of appendicitis; but constipation is such an extraordinarily common complaint of modern life that he would be a rash man who would contend that it alone is the cause of appendicitis. If it were the case there would be an enormous proportion of us eligible for an attack of appendicitis.

I shall now proceed to discuss the question as to when we should operate. We have a scattered population, and medical men are in places few and far between. If a man is unaccustomed to operate, or if he is unable to obtain the assistance he feels necessary, he will certainly save more lives by abandoning all thought of operation and freely purging his patients in the early stage, with the object of depleting the mucous membrane and allowing the appendiceal contents to escape. Speaking from my knowledge of adhesions, I think that the damage likely to be done by the supposed breaking down of the adhesions by the administration of aperients is mythical. If an abscess forms adjacent to the abdominal wall, or the rectum, it may be opened without great difficulty through the abdominal wall, the vagina, or rectum; but if it

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occur at any depth, and away from the abdominal parietes, a good assistant and the greatest of care and experience is required in order that the abscess may be opened without rupturing the bowel or spreading infection over the peritoneal cavity. A patient in an early stage, or with a localised abscess, may be transported for great distances without injury. If the patient is in the neighborhood of skilled assistance there is not the slightest doubt but that he should be submitted to operative interference immediately after the attack has begun, for no one can say, with any degree of certainty, whether the attack is likely to be perforative or accompanied by an acute peritonitis, or whether it will resolve. The pulse and the temperature are some guide, but frequently, by the time the pulse rate has increased, the favorable moment has passed, and the surgeon is invited to operate upon a patient who has a considerable amount of dirty fluid in his abdomen and a progressive peritonitis. The fallacy of waiting for a rise in the pulse rate is exemplified by the following case. A man, of 33 years, was taken ill with severe abdominal pains during the night. He was seen by his medical attendant on the following night. I saw him, in consultation, the following morning thirty-four hours after the onset of his illness. He had pain and some tenderness over the lower abdomen: his pulse was 84, his temperature 99. On opening his abdomen his appendix contained two concretions, and a perforation directly opposite one of the concretions. Offensive, thin, yellowish fluid, to the extent of a pint, was found in the pelvis, and a thin layer of it over the left loin. He recovered. This is a very unusual history, but I have seen at least one other precisely similar, and several whose symptoms were certainly hardly indicative of the grave condition within the abdomen.

If the patient is first seen after he has been ill a few days, and a small mass can be felt, careful inquiry should be made as to the course of the illness. If the pulse and temperature are low there is a fair probability that the mass does not contain pus, and that the inflammatory exudate will subside. To attempt to discover and remove an appendix from the midst of an inflammatory exudate is a difficult matter, and the operation is much more easily executed, and the patient's risk much diminished, if the removal is effected at a later stage. On the other hand, if the mass is accompanied by a rising temperature, or if a slight temperature persist, pus is almost certainly present. I would most emphatically condemn the advice given to any patient with an acute attack to wait until the acute symptoms have passed before submitting to operation. In the first place, no one, however great his experience, can say at the onset whether an attack will subside or end lethally. When a mass has formed after the fourth day we know that adhesions have been set up, and the appendix has been shut off, so that there is less risk in waiting for a short period to see if resolution is likely to take place.

When the appendix is discovered matted down in the pelvis, the Trendelenberg position should be used, and an incision made just outside the middle line. If the incision parallel to Poupart's ligament is adopted, I think it of very great importance not only to avoid as far as possible the actual division of the muscles, but also to preserve from injury the dorsal nerves and their branches. I have certainly seen cases where the union of the muscle along the line of incision has been perfect, but a bulging and laxity of the abdominal wall in that region will appear, which is very characteristic of atrophied muscle: on cutting down upon the part, the muscle fibres will be found to be pale and thin, and without tone. I do not think that many surgeons sufficiently recognise the importance of maintaining the integrity of the nerves of the abdominal wall.

Drainage through the abdominal incision is another fruitful cause of hernia. In some cases it is of paramount importance that free drainage

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should be established in order to save the patient's life; but very frequently, where the case is sufficiently septic to need drainage, a tube containing a gauze wick may be passed through a simple stab wound about an inch from the original incision. In doing this I usually make a short cut through the skin, and then, with my left forefinger inside as a guide, push a sharp-pointed scissors in an oblique fashion through the abdominal wall. This practice I have adopted for the past two years, and I have never seen it followed by hernia. A piece of gauze, changed three times daily, may usually be substituted for the tube after four or five days. I adopt the same stab method in the drainage of other abdominal conditions, and strongly recommend it to your notice.

In removing the appendix, in some difficult chronic cases, I have for the past two or three years adopted the following method:—The appendix is tied off at the cæcal end, and the stump held on one side. A forceps is attached to the distal cut end, securing the mucosa and submucosa only. Keeping a steady pull on the forceps, the muscular and peritoneal coats may be pushed off with a blunt-pointed scissors, or forceps, until the whole of the submucous and mucous portion comes away like a long earthworm. By this means the appendix may at times be removed from the most difficult situations with ease and safety.

There are many methods of dealing with the stump. Simply clamping a portion of the cæcum and cutting away the appendix with scissors, and sewing a double line of suture, as in suturing a piece of bowel, is an excellent method. However, the appendix is often so awkwardly situated that some of the more finished suture methods are impossible unless a large opening is made, and this is always to be avoided. It is not possible to ligature the base of the mucosa and submucosa, and push it within the cæcum: this is sometimes attempted, and a purse-string suture closes in the peritoneum round the inverted stump: it simply amounts to pushing the stump between the coats of the cæcum in that situation. Even though the stump has been treated with cautery or carbolic acid, it is not easy to be positive that all septic organisms have been destroyed. Returning the unligatured stump of mucosa and submucosa into the cæcum is unwise, for I have known considerable hæmorrhage to take place from it. In this, as in all other operations, the method which allows rapidity of execution combined with safety has much to recommend it. What is, I believe, known as the "cuff" method is both scientifically sound and effective. After ligaturing the base of the appendix, a cuff of peritoneum and muscle coat is stripped down for about an inch and a quarter. The mucous and submucous coats are cut off as short as possible. This stump is treated either with the actual cautery or with pure carbolic acid. The cuff is re-drawn over the end of the stump, and left covering it. There is really no need to ligature the cuff again, because if any oozing should take place, it would be preferable that it should escape and be dealt with than that it should remain bottled up where the increased tension would be likely to interfere with the resisting power of the normal tissues. Such a trifling raw stump has never, to my knowiedge, caused adhesions which gave rise to any symptoms. The stump can usually be left lying against the parietal wall, so that should any trouble take place the abscess would be in a safe position. If so placed it would probably remain there for some time, owing to the paralysis of the bowel from exposure and handling. Drainage should be adopted just as often as the surgeon thinks it is necessary; there can be no hard-and-fast rule except it be the rule that, when in doubt, drain.

I believe there is a considerable difference of opinion as to whether the appendix should be removed even when an abscess is present. The appendix can be removed at any time by any man who is determined to do it, but the

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question is, is it wise to remove it on all occasions? It would undoubtedly increase the patient's risk—perhaps not a great deal, but I am quite sure that one would be compelled to make a larger opening, and this is what should be avoided at all costs. If an abscess is present, a small opening is all that is usually needed: at a later stage this opening may be closed after the appendix has been removed, and, by means of a stab drain, if it be considered necessary to drain again, the abdominal wall is left as sound as it is possible to do so.

I do not think that any patient recovers from a true general peritonitis where the whole of the peritoneal surface is inflamed. In fact if, on opening the abdomen, the intestines are found matted together to any great extent with lymph, and more than one pocket of pus is found, the chances of recovery are small. I have seen these patients do well for a few days, and then there would follow a rise of temperature and a sudden break-away of pus in some part of the abdomen, and death would ensue. In severe early conditions there is a good chance of recovery if the dirty fæcal-looking fluid be thoroughly washed out of the abdomen. In such cases where free dirty fluid is found on opening the abdomen, I immediately find and remove the appendix, then make a cut in the left flank, and another in the right flank, and flush out through the left flank opening; so that the current tends towards the opening over the appendix. At the same time the finger is introduced into the pelvis, and recent adhesions are broken down so as to clear any pocketed collections of fluid. Gauze drains are admirable, but they act much more satisfactorily if introduced within a rubber tube, as there is a tendency for the gauze to become choked. The renewal of the gauze through the medium of a tube gives the patient very little pain as compared with its renewal without the tube. In connection with these cases I have often remarked two outstanding factors which are of immense service to us in saving the lives of our patients: firstly, the intra-abdominal pressure which tends to establish a flow towards the opening in its wall, a tendency, by the way, which is greatly assisted by the capillary attraction of the gauze; and, secondly, the disinclination of septic organisms to advance up stream. We often open into an abdomen and remove a dirty appendix, with infected material in its immediate neighborhood, but, provided a virulent blood infection has not taken place, the patient is almost certain to get well if a gauze drain is introduced so as to establish a current in the right direction.

Very free drainage must be adopted in some of these septic cases; and, partly because of this and partly because the peritoneum is already irritated, there is always a considerable risk of adhesive peritonitis giving rise to intestinal obstruction. If the pulse remains about the same as when the operation took place—it may be 100 or 120—and if there be distention, frequent vomiting or belching of flatus, accompanied by pains in the abdomen, it is more than likely that the patient has intestinal obstruction. At times, too, the bowels will be induced to act slightly; but still the patient does not sleep, and his abdominal pains persist, and his abdomen remains distended or becomes distended very soon after he expels a slight amount of flatus or fæces; he is still suffering from obstruction, and will probably die if not relieved. Many of these cases are apt to be looked upon as due to acute peritonitis, and nothing is done to relieve the patient; but with peritonitis we get a much more feeble running pulse, the vomiting is effortless, and the abdomen is usually more superficially tender all over. Nothing is easier than the relief of such a dangerous condition: and, in fact, the method I am about to mention is to be recommended in all cases of intestinal obstruction in the acute stage, for it is not advisable to attempt to discover and relieve an obstruction in a depressed patient with a distended abdomen. Make an incision one and a half inches long about half an inch to one side of the

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middle line, low down; split the rectus and open the peritoneum for about half an inch, pick up the first piece of distended bowel which presents; see that it comes freely and easily, and is not likely to drag when made fast. Suture the parietal peritoneum to the peritoneal surface of the intestine with a curved round needle and fine catgut, so as to leave a small oval of intestine exposed, In doing this, hold the centre of the oval piece of bowel with a pair of forceps. Before opening the bowel, tuck a narrow strip of gauze round the sutured edge, so as to catch any escape of fæcal material; then, with a sharp-pointed scissors, open the bowel and pass in a piece of tube as large as a number twelve catheter. If the fæcal fluid runs satisfactorily, let it drain away; if not, inject a packet of salts dissolved in a little water into the tube, nip the end of the tube with a pair of forceps, and leave it there for about twenty minutes before releasing it. In almost every instance the bowels will begin to act of their own accord in a few days. The tube may be taken out and the fistula allowed to close: the opening through the muscle will greatly assist the closure. Never make the incision through the middle line or the semilunar line, and be satisfied to stitch together peritoneal surfaces only. This will greatly facilitate the sinking back and closure of the fistula later on. I have had fifteen out of eighteen recoveries after adopting this method in cases of obstruction following free drainage of extensive fæcal extravasation.

Dr. Davies and Dr. McEncroe, two of my house surgeons, have been at considerable pains to collect details of the cases operated upon by me at the Royal Prince Alfred Hospital during the past five years. These, together with those I have operated upon in other places, amount to 452. The death percentage in hospital amounted to 9.5 per cent., whereas the other series gives me but 5 per cent. of deaths. The only patient who died when operated upon during the interval died about the fourteenth day, from intestinal obstruction. Her intestines were much matted from additional pelvic disease, and the intestines were so constricted that drainage of the bowel was of little service. I have seen the same multiple constriction occur in two other cases of intestinal obstruction. Both died. Note carefully why these patients died. Every one who died had marked adhesive peritonitis, extravasation of fæcal fluid, or extensive abscess formation. On two occasions a foul sloughy appendix had so infected the patients that even after their appendices had been removed, and careful drainage had been established, they made no improvement whatever. I think it is fair to say that almost every case would have recovered if the patient had been operated upon directly the earliest symptoms had become manifest.

This record will make it plain to the greatest sceptic that immediate operation is the true safe treatment for appendicitis. Besides those patients who died, there were many other cases of abscess and general extravasation with rupture which recovered, and many who would never have reached such a dangerous condition had immediate removal been recommended as the proper scientific method of treatment. I have known cases recently where experienced surgeons have, even as early as the second and third day, recommended patients to wait until the interval before having the appendix removed, and these same patients have developed abscess, or have gone on to rupture. I can hardly be accused of possessing the *furor secandi* of a young man, nor have I blindly followed any man's teaching, but I give you the results of my own experience; and I have demonstrated repeatedly to others the utter impossibility of saying what the future progress of an appendicitis will be. I think many of the more experienced physicians in New South Wales entertain the same views with regard to the necessity for immediate operation. Undoubtedly many attacks of appendicitis do resolve, but it is

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no difficult matter for a surgeon of any experience to obtain 1 per cent., or even a smaller mortality, for operation for the removal of appendices which have not created serious systemic disturbances.

It is only natural that a larger number of deaths should occur in a general hospital, simply because among the poorer folk illness is often disregarded for a longer interval, and the patients are admitted to hospital in a much more serious condition. In private work, it is the practitioner's duty to plainly lay before the patient what should be done to give him the best chance of recovery: if he elect to take his chance, there is no help for it; but it certainly is hard upon the operating surgeon who is called in to operate upon a patient who, through delay, has but a very much-diminished chance of recovery. So long as the fault lies with the patient we must put up with it; but if the medical attendant is foolish enough to take the responsibility of the unknown future upon his own shoulders, he must make up his mind to bear the blame which will certainly be his if his patient dies.

This subject is such a very large one that I have purposely only touched upon a few points which, I hoped, would interest you; and, before closing, I should like to caution you that appendicitis in very young children may very easily escape notice. One of my cases was 2 years of age. He had an abscess, and recovered after a few weeks. The other was 3 years of age; he had a ruptured appendix, with general peritonitis, and died a few hours after operation. The oldest patient I have seen suffering from appendicitis was a man of 80 years: so that there is no age which is free from the possibility of an attack.

My contention is that the appendix can always be removed up to the third day after the onset of acute symptoms. That it should be removed in all cases where rupture or a loose extravasation of septic fluid is present. In cases of abscess, say, of a week or more old, where the abscess is shut off by firm adhesions, it will be safer to drain the abscess and remove the appendix at a later date. Large incisions have appeared to me to be too often followed by an atrophy of the muscle fibres on the lower side of the oblique incision usually adopted, so that a sound repair of the hernia cannot be brought about. I invariably use an incision coming close up to the deep epigastric artery, so that the pelvis may be explored at the same time.

ON THE TREATMENT OF APPENDICITIS.

By W. J. Long, M.B., Ch.B.

In my opinion the proper treatment of appendicitis is removal of the appendix as soon as possible after the onset of the disease. The main reasons for this opinion are:—(1) There would rarely be any complications, with their attendant risks; (2) there would be no recurrent attacks. This would mean that the death-rate from appendicitis would almost certainly be not more than 2 per cent.

The only reservations I make are:—1. Unfitness of the patient, owing to his having other illness, *e.g.*, typhoid fever, advanced tuberculosis, advanced heart disease, &c., or his own desire that he run the risk of the attack, after having this explained to him. In the latter case, the question of operation is left out of the treatment of the appendicitis and reserved for its complications: practically this is the stand taken by most practitioners and their patients at the present time. Further, the patient may not be seen until the attack is subsiding, or until some complication has arisen.

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2. Unfitness of surroundings owing to their poorness, and to the paucity of attendance, or owing to the absence of a surgeon well versed in aseptic abdominal surgery. It is obviously unwise, on seeing a patient right away in the country, to operate at once unless there are grave reasons for doing so. It is safer to treat the case expectantly, and operate on any complication that arises, or in the interval if this do not happen. It is also obviously unfair, both to patient and practitioner, to operate when the latter is not in a position to be thoroughly well up in abdominal surgery.

Expectant Treatment.—After making a diagnosis, attempting at the time to localise the appendix and the nature of the disease in it, if the patient be in much pain I give one-third to one-half grain of morphia, apply heat to abdomen, and watch the patient. If the bowels have not been emptied I allow an enema: it is wise to abstain from purgatives. The patient has generally taken salts or oil and promptly vomited them. The aim of the treatment is to keep the inflamed parts as quiet as possible. The physiognomy, pulse quality and rate, state of abdomen, and temperature are carefully watched, and the decision made as to whether the case is recovering or going on to complications. No rule, such as a pulse rate of over a hundred, should guide the surgeon as to the patient's condition. It is the combination of high temperature and pulse-rate, with anxious expression and hard abdomen—or, still worse, the combination of high pulse-rate with low temperature and a beaten expression—that make the surgeon operate at once.

The Treatment by Removal.—The treatment by removal in the interval and early in the attack is the same. An incision 2 to 4 inches in length, with its centre corresponding to where the base of the appendix is expected to be, is made in the direction of the fibres of the external oblique, its length corresponding with the obesity of the patient. The fibres of the external oblique are separated and retracted widely. The fibres of the internal oblique and transversalis are then separated from the border of the rectus outwards to the ilium, and also retracted. The peritoneum is cut in the direction of the original incision. All bleeding is stopped by pressure, a ligature only being applied where necessary. The wound is retracted as required. The index and middle fingers, of whichever hand the operator is most expert with, are introduced, and a rapid careful examination is made, chiefly noting the extent of the adhesions to the surrounding organs, omentum, mesentery, bowels, bladder, pelvis, large blood vessels, muscles, &c.

In general it is best to find the longitudinal band on the front of the colon and trace it down: practically always, the base of the appendix is found at its end. The adhesions should be broken down in the inverse order in which they have been formed: this is usually determined by the ease with which they undo. Very little bleeding accompanies this as a rule. It must always be done very gently, and it is, at times, very difficult. Sterilised pads should be placed to prevent the soiling spreading from the operation area. If the omentum is tightly adherent it is better to remove the adherent parts with the appendix. In this way I have more than once removed the appendix with a surrounding abscess whole, without soiling the operation area. If there be an old fistula between the appendix and the bowel, the opening into the bowel must be turned in and sutured. Retention products, serum, pus, etc., in the appendix, must be not scattered in stripping it out. Should the serous coat of the bowel be injured the rent should be sutured, to avoid future adhesions or fæcal fistula. When the appendix is cleared of adhesions its mesentery is ligatured off, and the appendix is crushed at its junction with the cæcum. A purse-string suture is applied to the cæcum round the base, the appendix is cut through the clamped portion and inverted, care being taken to place a piece of gauze so that the area could not be soiled, and the purse-

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string suture is tightened. This method is practically never followed by fæcal fistula. If the operator choose, he may re-duplicate the purse-string suture, or he may place a few interrupted sutures. There is no need to ligature the appendix nor to turn down peritoneal cuffs. If the patient is bad and the surgeon is pushed, a ligature may simply be tied round the base of the appendix: as a rule this simple method gives perfect results. The mucous membrane left may be cauterised with pure carbolic acid, care being taken to remove any excess. The field of operation is then gone over for bleeding points, is cleaned with hot saline solution, and is sutured in layers. I always use silk for all buried sutures—fine Chinese twist; one or two points through the transversalis and internal oblique are sufficient—more for the external oblique, and horsehair for the skin. A sterile gauze dressing is applied.

Difficulties may arise during the course of the operation owing to the density of the adhesions, or the position of the appendix. If, after a reasonable time, the appendix is not delivered out of the wound, the incision may be enlarged by carrying it across the fibres of the internal oblique as far as may be necessary. The fibres must be sutured together carefully afterwards. The appendix may lie hanging into the pelvis, where it often is adherent to the iliac vein: this is fairly easily torn; or it may be adherent to the bladder or rectum. It may be turned back into a retro-colic pouch, and need stripping out from that position. If it seem impossible to excise the appendix, if any one part can be found it should be opened and followed up to the two ends, and the mucous muscular coats dissected out; the outer coats can then be sutured again, and the adhesion left alone. It must not be forgotten that the adhesions formed are often worse than the original disease.

When I am expecting the operation to be difficult I usually make my incision in the semilunar line. If pus is not expected, the incision can to some extent be made more secure against hernia by incising the different layers not immediately in the same line right through. If pus is expected, it is better to go straight through. If the abdominal wall be accurately sutured by interrupted sutures, picking up each layer to the same amount on each side and very little peritoneum, hernia very seldom results. This incision gives more room than the gridiron, especially for exploring the pelvis. The deep epigastric artery may be ligatured if necessary; but this is seldom the case. It has only once happened to me that I could not find all of the appendix: in this case I only got small pieces, and went on to undo the adhesions that I thought were causing most of the symptoms.

After-treatment.—This consists most largely in letting the patient alone. He must be watched for symptoms of hæmorrhage and peritonitis, and in either case the abdomen must be promptly opened. The hæmorrhage must be stopped, or the peritonitic area shut off with gauze and drained. Morphia may be given for pain; strychnine for shock. Hot water may be used to wash out the mouth, and it or tea may be swallowed as soon as the patient desires it: it does not increase the vomiting, but it makes it much easier, as there is something more than just a mouthful of bile to bring up. Mustard leaves or linseed and mustard poultice are to be tried to relieve the vomiting, and, if bad, brandy, or champagne and soda, warm draughts of soda bicarbonate, frequent minim doses of vin. ipecac. on the tongue may be tried. The patient may be turned on the side, or on back with knees drawn up. As a rule the bowels can be safely left alone for forty-eight hours or longer. If any distension occur, enemata of soap and water, with oil and turps, are the best. When a purgative is needed I generally give 5grs. of calomel, and, after a few hours, a Seidlitz powder, or repeated drachm doses of mag. sulph. until an evacuation is secured. I sometimes give other things, as best suited to the individual cases.

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The sutures may be removed from fifth to seventh day (in about ninth or tenth for through suture), and a collodion splint applied. The patient is allowed up in fourteen or twenty-one days, according to the case and to the suturing.

COMPLICATIONS.

1. *Hæmorrhage.*—This must be treated on ordinary lines, tying where possible and plugging where not. The plugs must be carefully placed, for they are liable to cause obstruction and fæcal fistula. A rent in the larger veins should be sewn.

2. *Abcess, or Localised Peritonitis and Diffused Peritonitis.*—This is the most common complication. It is most commonly caused by sloughing and perforation of the appendix; but there may be no perforation. If the inflammation is very intense and virulent, and the appendix is gangrenous, the patient is so prostrated that no barrier is thrown out, and he dies from general peritonitis. If not so intense, or if some adhesions have been formed by previous attacks, the peritonitis is localised and abscess (intra-peritoneal) is formed. In a few cases the appendix is retro-peritoneal, and then the abscess is extra-peritoneal.

Abscess.—This occurs where the perforation is, and, further, wherever the appendix is—unless transplantation by the omentum, or by gravity, has taken place: consequently its position varies very much. Many cases of abscess recover without operation: they either are absorbed, or burst into the bowel. Often the operation is simplified by leaving the abscess till it gets large, and is reached easily from the surface; but the proper treatment, undoubtedly, is to open it directly it is diagnosed. If the surgeon be fortunate enough to see the case early, he is generally able to remove the appendix as well as the pus; this is of great benefit. If left, it causes further attacks in some cases, and I am of opinion that in early operation for abscess one is more likely to get recurrence if the appendix is not removed than where the operation is done later. In the latter cases the appendix often sloughs away and comes out with the pus. Further, the drainage is less after removing the appendix, and so the patient has a better abdominal wall left. In cases not seen till late it is much wiser not to attempt to remove the appendix, unless it happens to come to the examining finger at once. Its removal is tedious and risky, as it endangers the continuity of the abscess wall. In deciding the incision to be made, the position of the previous pain and the position of the swelling must be taken into account. Unless the case is seen quite early (when the gridiron incision may be used), an incision cutting in one line through is the best, as giving the most room. When the pain and swelling are situated in the loin, and the leg is flexed and there is pain on moving the leg, and possibly mucus or blood in the stools, the appendix is behind the cæcum, and the incision may be the same as the lower end of the incision for nephrectomy, and it may not enter the peritoneum at all, or at any rate it may only enter it amongst the adhesions, and the free cavity is not opened. If the peritoneum cavity is opened the field of operation must be carefully packed off, so that the contamination will not spread.

When the pain and swelling are in the iliac fossa, and there may be some flexion of the leg, in this case, also, it is best to open well to the outer side. Often in this way the abscess may be opened without entering the free cavity.

When the pain and swelling are situated nearer to the umbilicus it is generally impossible to open the abscess without crossing the free peritoneum cavity to get at it. These are probably the most dangerous of the abscess cases. The main safeguard is in making a fairly large incision, so that a good

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packing can be placed all round the abscess. When this is satisfactorily done the pus is reached by separating the adhesions where they seem easiest to undo: the pus is mopped out as it wells up, and the cavity is thoroughly emptied. If the appendix is seen, it is removed: if not, the cavity is carefully and loosely filled with iodoform gauze; a rubber tube, without side holes and wrapped round with iodoform gauze, being placed in the centre of the pus cavity. After this the packing is removed, being replaced by a new and smaller one of iodoform gauze. The end of the tube must not rest on the bowel, as it is likely to cause fæcal fistula. The tube and gauze in the abscess cavity will need renewal before the surrounding pack: the latter should be left from four to six days, so as to get a good wall of adhesions to keep the pus away from the free cavity. The centre gauze may have to be changed daily, owing to the amount of the discharge. Meddle as little as possible with the gauze. Obstruction of the bowels may come on, and, of course, it is paramount in the treatment. The gauze must be removed; and if this prove insufficient, the wound must be re-opened and the bowel examined, or a small hole be put in the nearest piece of ileum and a fæcal fistula formed. This generally heals up in four or five weeks; if not it is excised, and the hole in the bowel sutured.

When the pain is deep in the pelvis, and there is pain on passing water and on emptying the rectum, and there is considerable distention of the lower pole of the abdomen, it is due to the abscess forming low down in the pelvis. These cases are often overlooked until the pelvis is full of pus. There is no tumor to be felt on abdominal examination at first, but there is on rectal examination. It is to be treated as in other abscess cases. If seen early it is almost always possible and right to remove the appendix. It is usually the tip that is in trouble. The base can be ligatured off, and the sutured bowel packed away from the field that is to be soiled: then the appendix is traced down, and the pus removed, and then the appendix (a fæcal concretion often being found at the bottom of the pelvis), and a rubber tube and gauze drain inserted, as described. When the abscess is larger, and, as is common, fills the pelvis, it may be drained by the posterior vaginal route in women: this gives dependent drainage. Occasionally it can be drained by the rectum in men: if not, it is well to keep as near the edge of the peritoneal cavity as possible, so as to keep within the adhesions, and simply empty out the contents and place a large tube in the cavity. If the free peritoneum cavity is opened, the packing off must be perfectly done. It is well not to make the tube too long, because the cavity contracts, and then the tube may press against the bowel. I do not believe in washing out these cavities. When the appendix is left, fæcal fistula often forms in these cases owing to the dependent position of the opening in the appendix. Sometimes these abscesses are fæcal, and contain gas at the time of opening them.

Diffused Peritonitis.—It is only a step from localised peritonitis, or abscess, to diffuse peritonitis. The worst form of diffuse peritonitis is that in which the infection is so severe from the start that no limiting adhesions are set up, and if an early diagnosis and immediate operation are not carried out the patient dies very quickly. In other cases there has been localisation at first, and, owing to some accident, *e.g.*, increase of tension through steady increase of the pus, too vigorous movements of the patient, or too vigorous manipulation on the part of the surgeon, the pus escapes from the abscess cavity and sets up further peritonitis. Even then it may localise again and a second abscess is formed, communicating or not with the original cavity. I have found quite different looking pus in the two cavities.

As soon as a diagnosis of diffuse peritonitis is made it is important to lose no time in preparing for operation. The belly can be prepared rapidly under

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chloroform, and a large incision made over where the abscess is believed to be. If the appendix can be found (and it generally can), it should be removed by simply tying it off. The pus must be mopped out carefully, and gauze drains with tubing inserted wherever the pus has been. If the operator is not sure about all the pus being got out, it is better to make another incision and be sure about it.

If the pelvis only has been filled with pus, two abdominal incisions will be enough. The pelvis must be mopped dry, and plenty of drainage instituted. If the pus has welled up further, the loins must both be incised, and large rubber tubes inserted, and gauze drains run from opening to opening. The main idea is to drain wherever there is pus, regardless of the number or position of the incisions. I do not believe in washing out the cavity, but if it is to be done it is best to put the irrigating tube in above where the pus has been, and wash out from a clean place towards the dirty part, so as not to soil the rest of the peritoneum. The pus may be washed out of the pelvis if necessary by keeping all the wounds well open, and holding up the bowels so that the irrigation does not spread amongst the bowels at all. Then the drains are placed, saline is put in subcutaneously, if necessary (and it seems to do good in these cases), and the patient is put to bed with the head of the bed considerably raised. Morphia has to be given on account of the great pain and restlessness. Saline enemata are given as soon as the patient can retain them. Stimulation is carried out as required. Here, as elsewhere, the patient must not be exhausted by over-treatment. All vessels and the appendix ligatured in these pus cases should have the ligatures left long: they generally come out in from seven to ten days, and save a great deal of trouble from stitch abscess later.

No sutures should be buried in the abdomen wall in pus cases. They are almost certain to suppurate, and everyone knows how much worry suppuration round a piece of silk can give.

Stitch abscess is best treated by curetting the wound with a small ear curette (fishing), with the object of removing the suture.

When these diffuse peritonitis cases recover it is wise to give an anæsthetic as soon after as seems right, according to the patient's condition, and either scrape all the granulations away or excise the wounds and suture them up properly. This also applies to long drained abscesses. Ventral hernia is usually saved by this.

3. *Fæcal Fistula.*—This generally occurs in abscess cases where the appendix has been left. The fistula is in the appendix, and generally heals as the wound granulates: in other cases it may be from the ileum or colon, particularly when much adhesion has been stripped—and I have seen it in the duodenum. The latter case wastes so rapidly that it is best to follow it up soon, and suture the rent in the bowel. Ordinary fæcal fistula usually heals spontaneously: if not, follow down the channel, dissect it right out, and sew up the hole left in the bowel after its excision.

4. *Ileus and Intestinal Obstruction.*—If the patient continues to vomit, and gets distended, it is wise to remove the drains; and if no benefit ensues, the wound should be re-opened, and any kinks or adhesions removed. Generally the best thing to do is to draw the nearest piece of the ileum into the wound and open it. It is wise, if expecting trouble, to leave a piece near the wound so that there will be no trouble in finding it.

5. *Septicæmia.*—This is treated just in the same way as in any other septic case. Quinine, antistrepto-coccic serum (if the case seems to be suitable), and stimulants being the best.

6. *Pyæmia.*—Treat on ordinary surgical lines. Open every abscess as it forms, and keep up patient's strength. Quinine is the best drug. Strychnine and digitalis and alcohol are required.

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7. *Pylephlebitis.*—This is noted by the patient becoming jaundiced wildly delirious, comatose, with widely dilated pupils, and death. I do not know what can be done.

8. *Abscess of Liver.*—There is generally tenderness over the liver, some increase of dullness on percussion, jaundice, sweats, and hectic temperature. An incision must be made into the abscess. Exploration with needle first is justifiable here.

9. *Subphenic Abscess.*—Increased dulness of the liver, the latter being pushed down; perhaps pain and cough, and sometimes jaundice, and sputa, and diaphragmatic pleurisy. It is best opened by removing a part of one or two ribs directly over where the pus has been got on exploration. The diaphragm is stitched to the skin, and then the abscess is opened and a large drain placed. There is often a great deal of bile drains away, and plugging the cavity with gauze is the best treatment for it.

10. *Pleurisy and Bronchitis.*—Treat on ordinary lines.

11. *Empyœma.*—This is not common, but is sometimes met with, particularly in cases of abscess behind the colon. It is treated by open drainage.

12. *Tubercle.*—This is a serious condition, as it is generally only in tubercular ulcer of the bowel and after a considerable time that the appendix is affected sufficiently to form tuberculous abscess. There are, generally, a large mass of glands, too extensive for dissection. One case went delirious after operation (apparently from general tuberculous dissemination), and died much in the same way as I have seen cases do after operations on tubercular hips, &c., many years ago.

13. *Foreign Bodies.*—Personally I have yet to find a foreign body in the appendix, unless fæcal concretions of all shapes and sizes are counted as such. The treatment is not altered.

14. *Pyosalpinx.*—This should be removed at the same time as the appendix. The treatment is the same, whichever organ started the mischief.

15. *Hernia of Appendix.*—When found in a hernial sac the hernia is usually of the sliding variety, and the utmost care must be taken in tying off the sac. If not, some of the blood supply of the colon may be taken up in the ligature, and death of that part of the bowel occur. If the appendix is unhealthy it should be removed; otherwise I do not think it necessary.

16. *Hydatid.*—I have seen hydatids growing in the serous coat of the appendix in a multiple abdominal case, and have removed the appendix for appendicitis caused by pressure of hydatids.

17. *Kidney Complications.*—Perinephritis is the commonest of these, and is usually seen in the retrocolic cases. Some degree of nephritis and pyelitis may occur.

18. *Malignant Disease* may occur here as elsewhere. The symptoms are the same as for cæcal cancer, and the only treatment is excision.

19. *Cystitis.*—Its best treatment is the removal of the abscess, and the administration of urotropin.

20. *Ventral Hernia.*—If the patient is very obese, and the hernia large, a well-fitted belt may be used; but, as a rule, it is best to completely excise the scar and suture the wound in layers. If it cannot be well brought together, a "grid" of hardened silver wire may be used to stiffen the wound.

21. *Pain in Wound.*—Patients at times complain of not being able to straighten themselves: this is generally due to adhesions re-forming inside after operation. They usually stretch or absorb, and the patient gets well. Occasionally a second operation is necessary to get rid of the condition.

22. *Omental Abscess.*—This seems to be formed by the omentum being affected from contact or adhesion with the appendix, and rolling up in itself in the same way as it occasionally rolls itself around the appendix. I have

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treated it both by complete removal with the appendix, and by incision into the abscess through a new incision. In the latter case the abscess was adherent to the anterior abdominal wall.

23. Injuries caused by retractions to blood vessels and bowels.

The above remarks are gathered from my operative experience of the last eight years.

APPENDICITIS: A CLINICAL REVIEW.

By Robert Scott, M.D., Ch.M.

In bringing under your notice the subject of my paper, and traversing ground that has already been so well and ably exploited by distinguished members of the profession all over the world, my only excuse must be that appendicitis, in one or other of its varied forms, is a disease of such wide significance, and one with which we are all—whether physicians or surgeons—brought so frequently face to face.

Being then of such frequent occurrence, there is more likelihood of an interesting discussion following; and, in that discussion, let me express the hope that we may gain some useful information that may enable us to combat the disease more successfully, and still further lessen the mortality in the acuter forms—which still persists, despite the splendid triumphs of modern aseptic surgery. There is no disease in the acute inflammatory stage which permits less of the "festina lente" method of treatment. Timidity must be put aside; diagnosis must be rapid, and a definite and determined line of treatment carried out. Many of us, who have been in practice for a number of years, have reason to look back with regret on the line of treatment which has for its refrain "He seems a little easier; we will see how he is in the morning," and the morning brings with it a perforative appendicitis with an acute peritonitis, which, in so many cases—even with an immediate operation—means death to the patient.

I had hoped to be able to collect all the cases treated by medical men in our city and district, whether operated on or treated without operation; but, finding it impossible to get anything like a complete list, I abandoned the idea, and give a list of all cases operated on in the Ballarat Hospital during the last three years, which was kindly supplied by the Resident Medical Officer (Dr. Langmore). I also give a list of cases seen by me in private—some of them in consultation. These tables cannot, however, be put forward as being of any statistical value in showing the actual prevalence of appendicitis in our district.

Date.	Total Number of Cases Operated upon.	Catarrhal Cases.		Suppurative Cases.		Remarks.
		Recovered.	Died.	Recovered.	Died.	
1902 .... (9 months)	Males, 3; females, 2	—	—	4	1	Peritonitis following operation
1903	" 5 " 4	8	—	—	1	Complicated with abdominal hydatid
1904	" 12 " 8	10	—	9	1	Peritonitis following operation
1905 .... (5 months)	" 3 " 4	3	—	3	1	Subphrenic abscess, which burst into lung

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In my case-book I have notes of twenty-one cases during the last two years, of which fifteen were operated on. Of these nine were catarrhal, and all recovered; four were suppurative, and all recovered; two were cases of general peritonitis, and died from toxæmia a few hours after operation. Of the six cases treated without operation three were catarrhal and two suppurative, and all recovered; one was a case of general peritonitis, which died from infection following perforation of the appendix. Of the twenty suppurative cases operated on, there were only two in which the appendix was removed, and these two both died.

Although some of the cases had prolonged convalescence, they all made a good recovery, and not a single case has come under observation again for any relapse or symptom necessitating further operation for removal of the appendix.

I do not propose to enter fully into the functions of the appendix. It appears to be a matter of conjecture as to its true use in the economy of nature. Sir William McEwan, in a Huxley lecture delivered by him, holds that the appendix has a distinct and definite function in digestion; and instances the case in which the anterior wall of the colon had been blown away by an explosion, enabling the condition of the mucous membrane of the cæcum and the orifice of the appendix to be carefully noted. It was observed that, shortly after food was taken, an exudate of glairy alkaline mucus was poured out from the orifice of the appendix, as well as from the mucous surface of the cæcum. It was noted that, after the reception of "bad news," which brought on an attack which he called "indigestion," the secretion in the cæcum became drier, causing smarting round the edges of the wound due to the acid chyme which was poured into the cæcum not being sufficiently neutralised by the "succus entericus," which, under normal conditions, was secreted by the cæcal and appendical glands.

A man 56 years of age was operated on by me for an appendical abscess, which was opened and drained without removing the appendix. On the twelfth day, on removing the gauze drain, about 4 ounces of glairy alkaline mucus welled up from the wound. This condition persisted for about ten days, sometimes being more profuse than at other times. Eventually the sinus healed up, and he has had no further trouble. Evidently this secretion came from an unobliterated part of the appendix which gradually became sealed up, for at no time was there any fæcal fistula.

Etiology.—Much has been written, and many theories advanced, as to the causation of appendicitis, and the alarming increase in the number of cases occurring of late years. Heredity, enteroliths, gallstones, rheumatism, gout, influenza (responsible for so many sequelæ), ectopic gestation, trauma, errors in digestion, and many other causes, have all their advocates.

I believe there is no more potent factor than digestive disturbances. MacEwan says that "present day man seems to act as if food should be thrown into the stomach, as a sandwich into a pocket, and the lid closed. Then he wonders that he has indigestion and appendicitis. Though, in a sense, digestion is independent of the direct control of the brain, yet powerful mental impressions exert an influence over the glands concerned in digestion, from the salivary down to the glands of Lieberkühn in the cæcum and appendix. The mental influence is such that, when the mind is engrossed in other affairs than the taking of food, those gland secretions are apt to be scanty, and indigestion is apt to follow. The standing lunch, eaten against time, with the mind otherwise occupied, is one of the best ways of not only producing indigestion, but of ultimately causing cæcal and appendicular mischief."

It is a recognised fact that appendicitis is most frequent between the ages of 10 and 30. It we contrast the amount of work put into those

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twenty years now with what was expected of an individual fifty years ago under like conditions, we will see that the greatly increased strain put upon the infantile and adolescent tissues must leave a weakened condition, less able to withstand the onslaught of any microbic invasion, and the first organs to suffer would be those of the digestive system. Now, if the appendix take an active part in digestion, would it not be one of the earliest to suffer, when we remember its anatomical construction—a cul-de-sac. What more likely than prolonged neglect of the ordinary laws of digestion in causing inhibition of the functions of the cæcum and appendix with subsequent stasis and inflammatory changes?

What is the ordinary condition of life in our States? Almost as soon as the child is able to think for himself, he is sent to school. The infant comes home from school, partakes of a hearty meal, and returns to school, where he sits—often in a constrained attitude—for two hours. All this time his brain is engrossed with the school work. Until about the age of 14 the child absorbs as much mental pabulum as possible, and if he shows any aptitude in a particular subject—such as music, or recitation—probably enters for some local "competition," which is a further strain on his mental capabilities. Then comes office work, or a university career, when the conditions of life are still strenuous; and, if anxious to succeed, has often to forego outdoor exercise for sedentary work, and has probably irregular meals. Even if exercise be taken, the boy often acts unwisely, in his ignorance—rushing from the office, or home, after a hasty meal, to play a game of tennis or football, or—worst of all—go for a long bicycle ride. All these conditions must, sooner or later, bring serious consequences in their train; the digestive system will probably earliest suffer, and the part that will soonest become disorganised, owing to its anatomy, is the appendix. I have gone fully into this somewhat homely picture, because, from a clinician's point of view, a timely warning to parents would do much to help physique and lessen disease. *Mens sana in corpore sano* never more urgently required impressing on parents. If the child were allowed to "run wild" till the age of 7, we would hear less of "post nasals" and appendicitis, and one would also find an improvement in the physique of coming generations in Australia. It is well known that appendicitis is rare among the black races, the reason being due to their mode of life.

Trauma is, no doubt, a very frequent cause of appendicitis; and possibly this is the reason that males are more frequently attacked than females.

A case bearing on this was a man who was thrown out of his buggy, and was so caught between the wheels that the back wheel pressed deeply into the iliac region. This happened about ten years ago. He was not laid up, nor did he complain at the time; but, on consulting me some weeks later, he stated that after a long drive or continued standing he had pain referred to McBurney's point with a sense of uneasiness and fulness over the cæcal region, which was always relieved by a night's rest. This condition has persisted up to the present. The accident probably caused a slight local peritonitis, and anything causing stasis of the bowel sets up a local hyperæmia and pain due to adhesions.

To enumerate the various factors which may have a bearing on the etiology of the disease would be tedious, but I may here cite the case of a woman admitted to the hospital under my care, with a history of pain in the right side for a fortnight, when she noticed a swelling in the right iliac region; otherwise the symptoms presented nothing of urgency. An incision was made over the swelling, but finding the parts matted together, a second incision was made in the mid-line. I then found the condition to be a tubal

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pregnancy adherent to the parietal peritoneum, with a matting of omentum and the tip of the appendix firmly glued into the mass. These were all removed, together with a parovarium cyst of the same side, and the patient made a good recovery. Apparently the ectopic gestation had been the exciting cause. Here I may remark that it should be a rule always to examine the uterine appendages of the right side when operating on a female, involvment of these organs being such a common occurrence.

From the clinician's point of view the various forms that appendicitis may assume fall under three great heads:—(1) Catarrhal appendicitis; (2) suppurative appendicitis; (3) acute toxæmic appendicitis.

The amount of literature that has been written about the treatment of these various forms rather appals one, and even yet no hard-and-fast rules can be laid down. Every case must be a law unto itself, and every medical man must exercise his own judgment and formulate his own rules for guidance, based on past experience and aided by knowledge gained from the experience of others.

In (1) catarrhal appendicitis, when one is called in early, and the patient complains of pain in the umbilical region, with a temperature of 99° to 100°, slightly furred tongue and nausea, with perhaps vomiting, but no thickening in the cæcal region, and no great rigidity of the abdominal muscles, the treatment consists of a saline purgative or calomel with small doses of morphia to allay the pain, and hot foments over the painful area, with liquid diet consisting of chicken or mutton broth and albumen or barley water. I always instruct the nurse to take the temperature and pulse every two hours, and at once report any rise above 100·5. At the same time the friends are informed that an operation may be necessary at any time during the attack, and, if not during the attack—which may prove mild—then during the quiescent period an operation should be performed. In mild cases I always keep the patient in bed until all tenderness has disappeared—a variable length of time. If operation then be declined, the responsibility rests with the patient himself.

(2) Suppurative appendicitis.—One is called in and finds the patient complaining of pain in the right iliac fossa, pulse and respiration increased, temperature 101 to 102. Tongue coated with a creamy fur, and the right leg flexed on the thigh; vomiting usually, and nausea always, present; on palpation, a rounded swelling is felt in the cæcal region, painful on pressure, but the rest of the abdomen fairly flaccid.

When confronted with these symptoms, what is the line of treatment to adopt?

During the time I have been in practice, no class of case has given greater anxiety. Before these cases were so well understood, and before asceptic surgery had attained its present brilliant position, one hesitated before advising operation, and waited for nature to effect a cure. How many lives have been lost by thus temporising! Some years ago I waited for four days, and if the symptoms did not improve, advised operation. Now I am firmly convinced that the only treatment admissible is *early and immediate operation*. If the case is being attended by a physician, he should call in a surgeon. When the case has reached this stage, it is no longer one requiring palliative treatment. When operation is decided on, what form should the operation take? On this point opinion is still at variance. Many leading American and Continental surgeons contend that the incision should be on the inner side of the swelling; others, just as eminent, that the first opening should be over the most prominent part of the abscess. It has always appeared to me that the incision should, in the first place, be made over the swelling. As the peritoneum is reached the incision is gradually deepened, using the finger to

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indicate by sense of touch whether the pus is nearly reached. When pus is reached, the opening into the abscess is very carefully enlarged, taking every precaution not to break down the retaining abscess wall. The contents are then very carefully swabbed out; if the quantity is great, I do not hesitate to use a saline douche, taking care not to exercise any great pressure. If the appendix is lying in view, I ligature and remove it, but waste no time looking for it. An iodoform gauze drain is then loosely packed in the cavity, and the ordinary dressings applied. Should the incision be larger than is considered necessary, one or two sutures are inserted.

Murphy, of America, in a paper published some months ago, advises that in these cases the operation should be directed to opening the abscess and draining, the appendix being amputated only if it is readily accessible, the greatest care being taken to avoid separating the agglutinated intestines. This appears the more rational course; the patient is not in the best condition for sustaining the shock of a prolonged operation. There is decided danger in breaking down the wall of the pus cavity, and in some cases the appendix will not be found lying in the abscess cavity. If the operation has taken place into the meso-appendix, the organ itself may really lie outside, or form part of the wall of the abscess sac, and in such cases any attempt to remove it would almost surely prove disastrous to the patient. In the majority of cases the appendix becomes a necrotic mass, which comes away in the discharge, or becomes so glued down and obliterated that it gives no further trouble. Should the appendix give further trouble, it can be searched for and removed at a second operation at a very slight risk to the patient.

(3) Acute toxæmic appendicitis. I have used this nomenclature because it expresses the condition of the patient; although it may not be strictly classical; still the appendicitis is the exciting cause. These are the cases which Mayo Robson aptly describes as being of "a peculiarly tragic nature." The attack comes on, in many cases, with startling suddenness. From being in a condition of perfect health the patient is, in a few hours, fighting for life against an army of malignant microbes. The diagnosis is comparatively easy if there is a history of prior attacks of right-sided pain, but in many cases not even this is present, and one is called in to find the patient taken suddenly ill with abdominal pain and vomiting, rapid pulse and temperature of 101 or 102, which, with the dusky color and anxious expression of the face, and the general rigidity of the abdominal muscles, make up a picture characteristic of acute infective peritonitis. In such cases there must be the least possible delay. Even if the diagnosis cannot be clearly made as to whether the case is one of acute intestinal obstruction, or appendicitis, or other cause, the treatment must be the same—early and immediate laparotomy. The mortality in these cases is very high; but a surgeon's duty is to give the patient the "only chance," even at the risk of incurring the unpleasantness that "a death from operation" always brings. Certain death without operation, and a "possible" chance by operating, must be the prognosis

This outlook, coming suddenly on patient and friends, makes it difficult for them to realise fully the gravity of the case, and after the almost certainly fatal result of operation some are sure to cavil at the wisdom of the surgeon. A case in point came under my care a few months ago. I was called to see a lady in consultation with a brother practitioner. She was 29 years of age, and in perfect health until thirty-six hours previously. When out in the evening she was seized with abdominal pain. Vomiting shortly after began, and was incessant. She was seen early in the morning by her medical attendant, but she steadily became worse. She was seen by me at noon the following day, when she presented all the symptoms of acute general peritonitis. At the urgent request of her husband, I operated three hours later, when I

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found the abdomen full of most offensive sanguineous fluid, and found also a perforated appendix. The patient only lived a few hours after the operation.

From a clinician's point of view, it matters little in these cases whether the infection be due to streptococci, staphylococci, or the colon bacillus; early diagnosis and prompt operation is the only possible hope, and this hope should, even in cases appearing almost desperate, be given to the patient, if consent be obtained. Irrigation with hot saline solution appears often to rally the patient, and gets rid of the offensive fluid more speedily than dry swabbing. I cannot see that the former method is more likely than the latter to harm the surface of the peritoneum, and so open up new avenues for infection. I have not tried continued irrigation after laparotomy, as recommended by La Place, but should be inclined to give the method a trial in a desperate case.

The after treatment of these cases hardly comes within the scope of this paper, for it falls under the heading of the general treatment of post-operative laparotomy.

As one writer remarks, the surgeon must (in these cases) always remember that even if he can save one life in fifty it is worth while making the attempt.

The method employed in making the incision is a matter of individual choice. The ideal method is the "gridiron" incision—there is less risk of subsequent hernial trouble; but I never hesitate to divide muscular fibres if desirous of more room, and if thorough technique is carried out and carefully applied, and buried sutures are introduced, the scar appears to be as firm in one case as the other.

I have not touched on the various methods adopted for amputating the appendix, for, like the incision, this is purely a matter of choice. The American method most adopted consists in ligaturing the appendix, and then inverting the stump into the cæcum, burying it there by a purse-string suture reinforced, if necessary, by a second row of Lembert sutures. Personally I have never found any trouble result from the amputated stump left in the abdomen after touching it with pure carbolic. The advice given by Treves is all-important—that the amputation must be made as close to the orifice as possible, so as to leave no cul-de-sac that may form a focus for future infection.

To sum up. Though many causes contribute toward the production of appendicitis, one of the most frequent is due to an aberration of the function of the appendix brought about by continued abuse of ordinary conditions of digestion. Therefore it is the duty of medical men to warn patients, and especially parents, to take all possible prophylactic precautions to ensure avoidance of attacks. When an attack comes on, it may assume a mild non-infective course which may pass off with palliative treatment, and give no further trouble, or may, either during this or subsequent attacks, pass into the more serious stage of suppuration. When it reaches this stage, either the condition known as suppurative appendicitis may be present, where the abscess forms slowly, owing to perforation being, for the most part, in the meso-appendix; or the fulminating attack known as acute perforative appendicitis may present itself, due to an overwhelming attack of microbic invasion and the bursting of the appendix into the general peritoneal cavity before there is time or (from the position of the appendix) opportunity to shut off the invading infection from the peritoneal cavity. But the treatment of these cases is the same, viz., *immediate operation.*

The ethics, as between physician and surgeon, in these cases may be summed up shortly. In purely catarrhal cases either physician or surgeon may treat the patient; but in suppurative and acute perforative appendicitis, the physician *never*, the surgeon *ever*.

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SOME OBSERVATIONS ON APPENDICITIS.

By R. Steer Bowker, M.R.C.S., &c.

Mr. President and Gentlemen—When I started to write this paper I thought I would be able to put all I had to say on a few pages, but by the time I had finished it I found that it was of such length that I could hardly expect you to hear me out, more especially as there are others to be heard, and the time at our disposal is not great, so I have asked to be allowed to read an epitome of what I have to say, and will mention the points I wish to bring before you without going further into detail than is necessary, and will only quote my authorities without giving their arguments.

My excuse for bringing so well discussed a subject before you is that there are so many erroneous views enunciated concerning it, such deep interest taken in it by the laity, and so many stupid and misleading statements made about it by the public press.

One constantly hears the question asked, in a more or less scoffing manner: "Why is appendicitis so common now?" And it is freely hinted, even by some members of our own profession, that appendix operations are too frequently and unnecessarily done. Gentlemen, the error is on the other side: even now, with our present knowledge of the disease, delays are too frequent, and operations too often deferred.

Causes.—Foreign Bodies.—The general public, and even many medical men, have been accustomed to look upon fruit seeds and other foreign bodies as the chief cause of appendicitis, whereas it is a very rare thing to find any such in an appendix. It is an accident when any such body gets into an appendix, and due to some accident if the appendix does not, by its peristaltic action, get rid of it again.

Anatomy.—With regard to the anatomy, I will not take up your time, except to ask you to remember that the blood supply is a poor one: that, in the ultimate distribution, the vessels ramify between the mucous and submucous coats, surrounded by a fairly dense muscular layer, and that the organ is one which nature, in her economy, is endeavoring to rid herself of: also the longitudinal muscular bands (or tæniæ) on the cæcum leading to the appendix, and the fact that, at the cæcal orifice of the appendix, the mucous membrane is thrown into folds: one crescentic in shape—just above it—is known as the valve of Gerlach, and another on the opposite side, but within the lumen, is that of Nanninga, more marked—the acuter the angle at which the appendix comes off from the cæcum, and that on distension of the cæcum these folds or valves are liable to close the orifice (Kelly, 1); that the nerve supply is derived from the superior mesenteric plexus of the sympathetic, which supplies also the small intestines, and to this fact is attributed the wide distribution of pain in early appendicitis (G. R. Fowler, 2); and that the veins empty into the superior mesenteric and on into the portal. Its lumen is narrow, the adenoid tissue encroaching on it, any catarrhal condition liable to still further lessen its calibre. Ribbut, Kelly, and, more recently, Woods-Hutchinson (3), have described how nature, in trying to destroy the organ, does so by obliterating the lumen—often only in parts, and so leaves strictures; and that the lumen is the habitat of various micro-organisms which may, on slight provocation, become virulent.

Definition.—What is appendicitis? It is as well to give a definition before going into the causes; and, for this purpose, I cannot do better than quote R. T. Morris (4). He says:—"Appendicitis is an infective exudative inflammation of the vermiform appendix of the cæcum, originating in any local cause for the production of an infective 'atrium,' or gateway in the

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tissues of the appendix, and progressing by bacterial invasion into the layers of connective tissue and the layer of lymphoid tissue, all of which are partially or completely disabled by interstitial exudate compression within the narrow muscular and peritoneal sheath of the appendix. The principal cause of appendicitis is mixed bacterial infection from the lumen of the appendix: the chief cause of bacterial infection from the lumen of the appendix is the formation of an 'infective atrium' in the *mucosa*, by force applied in any way."

So that appendicitis is the result of an "infective atrium" in the mucous lining of the appendix, the result of any cause.

Now, Van Zwallenburg (5) shows that the chief cause of infective atria is the cutting off of the blood supply; that the chief cause of the cutting off of the blood supply of the appendix is distention of its lumen, causing pressure on the vessels in the submucous coat, compressing them against the unyielding outer walls, de-vitalising the mucous membrane and allowing it to be attacked by the contained pathogenic organisms; that distention of its lumen is caused by any obstruction to its outlet, a constriction more or less narrow, with a contained concretion of some sort—not necessarily a hard body, a little fæcal matter (or even a plug of mucus) may suffice to plug the opening. Given these conditions, an excess of fluid may be forced into the appendix by some muscular effort past the obstruction, and, by its recoil, forces the obstructing material (whatever it may be) against the mouth of the stricture, blocking the opening, and so acting as a ball-valve; distention takes place. So that it may be said that anything which interferes with the blood supply is a direct cause of appendicitis. Given a good blood supply, the bacteria are inert; de-vitalise the tissues, and immediately the bacteria attack them. This supply may be cut off from the base by a twist, twisting the artery, by pressure of some other part, &c., or an obliterative endarteritis starting peripherally may occlude the main vessel, and cause sloughing of the entire appendix. But no doubt the description given by Van Zwallenburg (5) is the most common cause; and I will now trace the various factors which lead to that condition.

First and foremost, we have some former slight attack recovered from, the result of injury, typhoid ulceration, or in fact anything causing an abrasion of the mucous membrane, which has healed, with the formation of connective tissue, in which contraction has taken place, leaving a narrowing or stricture of the tube; or this may be caused in the process of natural obliteration which goes on from birth until death, causing destruction of the lymphoid tissue and connective tissue replacement—or even a thickening of the lymphoid tissue, or an increase in it, may block the lumen, or some peri-appendicular peritoneal adhesion may cause a kink in the appendix, and so arrive at the same result.

Now for the *causæ causarum*. The causes of stricture have been mentioned.

Lymphoid tissue in some people is excessive.

Catarrhal condition may be caused by any intestinal irritation, typhoid fever, influenza, some of the exanthemata—any hereditary malformation of the appendix, narrowing its lumen or distorting its shape.

Kinks caused by peritoneal adhesions from inflammation of the other organs. Kinks caused by peri-appendicular peritonitis, the result of intra-abdominal muscular traumatism, as described by Byron Robinson (6)—chiefly by the action of the psoas. Distention of the cæcum, the result of flatulence, may cause obstruction; and, if the appendix is already a maimed one, may precipitate an acute attack—and, to my mind, at any rate, accounts for those attacks of colic or appendicitis attributed to the eating of green fruit or some indigestible food, but not on the grounds stated by Rubin (7), of

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Rush Medical College, who attributes appendicitis to our civilisation causing man to retain gas in the large intestine for long periods of time; that he is most of his time in places and under conditions which render it undesirable that he should rid himself of it; that this gas distends the cæcum and appendical opening, and allows foreign bodies to get in—attributes to this the fact that savage people seldom suffer from the disease, and quotes Bland Sutton and J. H. Campbell, who have made a study of the appendix in anthropoid apes, as saying that they never came across an inflamed appendix in these animals, and in proof of his contention gives laboratory experiments which he conducted; but forgets that these were on a cadaver with no blood supply to the mucous membrane, and consequently no turgid valve of Gerlach or Nanninga to shut off the lumen: besides which, Wienberg (8), in the annals of the Pasteur Institute, relates a case of appendicitis in a chimpanzee. Distention of the cæcum is controlled, to a certain extent, by the muscular bands and the abdominal wall, especially behind, and so distends in a lateral direction more than antero-posteriorly, and approximates the edges of the opening; and where the appendix comes off from the cæcum at less than a right angle —as it generally does—the valves of mucous membrane very efficiently close it.

Now, to follow up the theory of Van Zwallenburg (5), some extra strain—a bicycle ride, some athletic exercise, or even a change of the position of the body during sleep—may set up this ball-valve action in the appendix, by causing an excess of fluid to distend it; peristaltic action is set up, which may overcome the obstruction, or may not—if not, the appendix still tries to get rid of the obstruction, and painful contractions of the organ take place, giving rise to "colic" (just in the same manner as we get hepatic colic). The obstruction, by the excessive effort of the peristalsis, may yet be overcome, and the attack end in colic only, for the time being; or the obstruction may persist, more fluid be exuded from the walls of the appendix, and exudation take place into its tissues, rendering it still more tense: distension increases until circulation is arrested, pain diminishes by reason of paralysis of the nerve endings, and perforation or gangrene takes place. Now to interpret these signs: when the pain lasts one or two hours, with sudden cessation, then the obstruction has given way. If there is no tenderness left, the attack has passed and left no material further damage; if tenderness is left, then some inflammation has been caused; if the pain lasts several hours, with vomiting with abrupt cessation, perforation has taken place; if the pain gradually diminishes after six hours to twelve hours, gangrene has resulted; and if it reappear after twenty-four hours, peritonitis has commenced. This account very accurately describes an ordinary acute attack; but there are other factors at work, which will have to be considered, causing the disease to run different courses; some to be acute or fulminating, some to run a chronic course, some to end in resolution, and others to go on to suppuration, some to be localised, others to cause general peritonitis, pyelophlebitis and portal pyæmia, vomito-negro, empyæma, or acute toxæmia.

The nature of the attack will depend on (1) the position of the appendix; (2) the nature of the micro-organism set free.

There are two chief positions of the appendix governing the nature of the attack—*Colonic* and *Enteronic* (Byron Robinson) (6)—*Colonic*, or benign area of peritonitis, being the outer side of a line drawn from the surface of the liver along the psoas muscle over the right pelvic brim to the pelvic floor: *enteronic* area, to the inner side of this line towards the centre of the abdominal cavity; an appendix pointing here will be amongst the enteronic coils, where the stomata vera of the peritoneum are vast in numbers, whilst in the colonic area they are limited.

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The enteronic area is the area of absorption (septic). Perforation in the enteronic area is liable to be followed by absorption and acute toxæmia before a limiting membrane can be thrown out. Whilst in the colonic area, the peritoneum has more time to throw up a defensive barrier, absorption being slower. The most constant micro-organism in the appendix is the B.C.C. Bacillus pyocyaneus is occasionally found, and streptococci more frequently.

All authorities are fairly unanimous in that the nature of the infection depends on the virulence of the organism which predominates, that the infection is generally a mixed one, that the B.C.C. is the only organism ever found alone, and that the streptococcus pyogenes leads to the most severe infection.

Though constipation, *per se*, has little to do with the causation of appendicitis, it has, I think, a deal to do with the numbers of the bacilli, for it would cause stagnation and favor the multiplication of germs, giving them a quiet corner to breed, surrounded by heat, moisture, and food.

An infection caused by the B.C.C. alone is generally of a mild form, the resulting peritonitis more limited and less severe, with the production of pus of a characteristic fœtid odor: whereas in a streptococcal infection in the enteronic area, highly virulent toxines are thrown into an area where absorption is very rapid, and the probability of death is very great. The question is not as to whether complete gangrene or perforation has taken place, for probably, as it would take longer for the whole appendix to become gangrenous than it would for it to perforate, the omentum would have a longer notice of impending disaster, and so have a better chance of coming to the rescue.

I do not think the nature of the micro-organism has anything to do with whether gangrene or perforation shall result; that is determined by three other factors—(1) nature of obstruction; (2) position of appendix; (3) number of former slight attacks.

Where the obstruction is caused by a hard concretion, and the distention and œdema is cutting off the blood supply, the organ will give way first where the pressure is greatest, viz., at the site of the stercolith; perforation will result, relief from tension will take place, and gangrene be averted. Again, where there have been many attacks of peri-appendicular peritonitis, causing the appendix to be buried in adhesions, sloughing seldom takes place on account of the greatly increased blood supply—more particularly where the appendix is attached to muscles—to the outer side of the colon. I have called this the region of "chronic or constant appendicitis," as distinguished from "recurrent appendicitis"; a sloughing appendix is generally found free in an abscess cavity. Complications from this class of cases arise from (1) absorption of pyogenic organisms into the circulation; (2) displacement and migration of venous thrombi; (3) infection through the medium of lymph channels; (4) direct extension of abscess.

There is another class of case where only slight symptoms are followed by an acute attack. Lejar and Menetrier (9) describe, in the *Revue de Chirurgie*, various diverticulæ of the appendix—small outgrowths from the lumen, having thin and easily ruptured walls, connected with the lumen by a small neck, the result of some abscess in the appendiceal wall; an adhesion between one of these and a muscle, or some other organ, could be easily ruptured by any exercise or strain, and set up an immediate and acute attack, and no doubt account for some of the attacks following immediately on exertion. These diverticulæ are prone to take place at the site of the meso-appendix, and may account for some cases of thrombo-phlebitis.

Recurrent Appendicitis I class those sub-acute attacks, either of catarrhal appendicitis or of obstructive appendicitis, from which a patient recovers and which recur from time to time until an attack with perforation occurs.

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Then there is another class of case of which I wish to speak, more common than is generally thought, and which I divide into two groups :—(1) Chronic, or constant appendicitis, the source of definite trouble and easy of diagnosis (as a rule); (2) unrecognised appendicitis, causing general ill-health with but vague symptoms, in which the diagnosis is difficult and conjectural. I think, in the latter group, there are more cases than in any other class.

Now to consider the milder attacks of the disease which prepare the way for perforation, or for chronic appendicitis and perpetual ill-health.

Appendicular colic will be severe, or otherwise, according to the difficulty the appendix experiences in ridding itself of the obstructions. This having been accomplished, the attack will subside. If there remains no inflammatory trouble there will remain no tenderness on pressure shortly after the attack, but if there remains for a couple of days tenderness on pressure, then there has been some damage done—a small infective atrium formed, probably, or some exudation into the tissue of the appendix, which will almost certainly lead to future trouble; so that the fact as to whether, after an attack of colic, there is or is not a resulting tenderness becomes a most important sign, and should guide the surgeon or physician in the advice given to his patient.

In the case of a smooth foreign body (viz., one which, by its sharp edges, does not perforate the appendix) its position in the lumen will have a great deal to do with the result; if it gets to the distal end of the appendix it will probably become fixed there, as peristalsis will not have much power over it, and the appendix will, after a time, cease to try to get rid of it; mucin and salts will be deposited on it, and it will in time become a veritable stone, and others may form above it: and if the appendix is situated to the outer side of the cæcum, as in these cases it generally is, their presence sets up a mild irritation, and peri-appendicular peritonitis—which will cause it to become adherent to the tissues around, probably the iliacus (or the sequence may be reversed and fixation of the appendix by peritonitis, the result of traumatism of the iliacus, be the first step), a chronic irritation will be set up, with but little pain, only occasional aching, interfering with the action of the intestine and nerves of the part, causing indigestion, flatulence, constipation, &c., without any very definite symptoms—only occasionally vague discomfort: the person is never very ill and never well, often only "that tired feeling" of which so much is made by the quacks. These appendices never slough and seldom perforate, on account of the extra blood supply that they receive from their situation and condition. They are generally large appendices with their mucous membrane more or less destroyed. They can generally be diagnosed—if thought of—and carefully searched for.

By *Unrecognised Appendicitis* I mean that class of case written about by Gibbons (10) in the journal of the American Medical Association, chiefly in children, and more recently I think by Fowler (11) in adults.

Gibbons says :—"Ailments referred to the intestinal tract in infancy and childhood, and misinterpreted as 'gastric difficulties,' 'cholera infantum,' 'colic,' 'gastro-intestinal,' 'intestinal and nervous dyspepsia,' 'liver ailment,' 'constipation,' or even diarrhœa—such 'difficulties,' 'disturbances,' and 'ailments' are nearly, if not always, due to a diseased appendix." He says :—"Watch carefully children who are pale at certain times without cause, who are yellow or of an earthy color, especially those who vomit easily without cause, or after driving or walking." There are also cases, in adults, in which it is difficult to realise that the appendix is the source of their trouble. My attention was first called to them by a couple of cases I saw in consultation with the late Dr. Angel Money, a few years ago, in which the operation proved the diagnosis and cured the patients. These patients are always in ill-health, though never very ill—constantly complaining—and have consulted in-

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numerable medical men, and have taken almost all known medicines, and gone through a course of what Gibbons calls "dietary juggling," and are told eventually that their trouble is "nervous," or the result of "excessive uric acid," and know all about "purins," &c.

Bearing this in mind one must look with suspicion on such a case—complaining of dyspepsia, flatulence, and constipation; no signs to lead one to a definite diagnosis, never any marked pain, perhaps an occasional attack of colic, forgotten until inquiry is made; no tenderness on pressure anywhere, but probably having a dilated cæcum. This condition of cæcum, together with a flabby whitish tongue, is often the only sign of chronic appendicitis; and if careful palpation of the appendiceal region is made some tenderness may be found, enough to warrant the probable diagnosis. Remembering that the appendix in these cases is generally to the outer side of the cæcum, and pretty firmly fixed, probably more or less obliterated and containing calculi, and interfering with the nerve supply to the cæcum by irritating the peripheral nerves—never, or rarely, ending in an acute attack, but always remaining a source of chronic intestinal irritation.

The symptoms of an acute or sub-acute attack are so well known that I need hardly go into that subject, except to mention that the pain may be complained of at the onset almost anywhere over the abdominal wall; but, as a rule, tenderness on pressure will generally be found over the seat of the appendix, or the pain will generally eventually settle there: though, in a case I had recently, the pain was complained of over McBurney's point, and there was resistance and tenderness there, but, on making my usual incision in the iliac region, only coils of small gut were found and no cæcum, but I could feel the cæcum higher up in the region of the kidney, and I removed through an oblique incision (as for a kidney operation) a large appendix on the point of rupture—a case of non-descent of the cæcum, the appendix being behind and in the kidney pouch. Another point to be emphasized is that during even a moderately mild attack the cessation of pain—the temperature and pulse dropping or remaining throughout but little above normal—may not mean that the appendix is well, but that it is dead; a fact I could illustrate by many cases had I the time. This is a condition which has lulled many into a false sense of security, with disastrous consequences.

"Why *is* appendicitis *so* common now?" It is due no doubt, in great measure, to more accurate diagnosis, and so the increase is more apparent than real; but there is no doubt that since 1889 there has also been an absolute increase in the number of cases, and Marvel (12), in a very able article, puts this down to what I consider the correct cause—a conclusion I had come to before reading his paper. He attributes this increase to the recent epidemics of influenza, which began in that year, and quotes the statistics of three large American hospitals for the last three five-year periods, also Continental opinions, among which are Winternitz, Pever, Faisans, and Sonnenberg, showing that the Pfieffer bacillus has been found in the appendix, in support of his views; and there can be no doubt that influenza, more especially the intestinal type, is responsible for many cases of appendicitis,

Now, as to treatment of these treacherous cases. All up-to-date physicians and surgeons the world over are in accord in thinking that all pus cases should be operated upon at once—except, perhaps, those cases in which the dose of poison absorbed seems too great to allow of the patient rallying from an operation; also, that if a patient can be tided over the acute attack the best time to operate is during an interval. But can we always tell when pus has formed, or shall we diagnose the formation of pus only when pyelo-phlebitis, or portal pyæmia, is setting in? For the signs by which we have been supposed to diagnose pus formation are more usually the signs of commencing

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toxæmia, and the case is probably then too far gone for operation to save it. Then, again, we are taking a very great risk—an unjustifiable risk—in the present state of our knowledge, in trying to tide a patient over the acute attack when we see it at the beginning of such an attack. How can anyone tell what is going to be the ending of even an apparently mild attack? Why wait for the interval when that waiting subjects the patient to absolutely unknown risks, of which the physician can judge no better than can the patient?

People are so educated to the dangers of appendicitis—each individual almost in the community knows of friends lost by waiting, though they may not realise that the waiting was more the cause of death than the disease—that when they know the name of the disease from which they are suffering they are prepared, in most cases, at once for operation, and only wait for their physician to suggest it for them to agree to it. The patient puts his trust in the judgment of his medical man more in these cases, perhaps, than in any other, and he is indeed a very foolish or very unscrupulous man who unaided will take upon his shoulders the unknown risk of "tiding a patient over" an acute attack to the interval, when he can obtain the services of a competent surgeon.

Now, what have we on the other side? If the appendix has perforated, then it is admitted an operation is urgently needed; if it has not perforated, and the trouble is still yet within the appendix, then, in the hands of a surgeon used to the cases, an operation is almost devoid of risk and should not be dreaded, and would in most cases—if both sides of the question were candidly put to the patient—be eagerly asked for, and free the medical man from an amount of responsibility that he has no right to assume.

It is not even as though a patient, having faced the unknown risks and come haply through with his life, is any better off than he was before; he still, in the great majority of cases, cannot avoid an ultimate operation, for no one can tell which individual case will be one of the lucky few which will get no further attacks; for there can be no argument against the fact that an appendix which has once been attacked—even mildly—is, by that attack, rendered more prone to future attacks; one attack does not give immunity, but the reverse. Why give a restive horse a chance of bolting? Besides which, an appendix at the commencement of an attack is in a better position for operation than one recovering from an attack, and the operation much more simple, for later there will be adhesions, &c., to complicate the case.

In support of these views I cannot do better than quote Barrett (13), of Chicago, who expresses himself most happily. He takes for his text an explanation a surgeon so often hears—or, I am glad to say, "used to hear"—"I was treating the case medically until I could detect pus," and says, "There is no greater fallacy than the idea that an unruptured appendicitis deserves medical treatment, but that the advent of pus outside the appendix is the indication for the insistence of an operation. A physician harboring this opinion—and there are not a few—should remember that so great an authority as Osler has said, 'There is no medical treatment of appendicitis.' Knowing the virulence of appendiceal infection, its tendency to break through the wall of the appendix, and occasion a fatal peritonitis, the man who deliberately sits waiting for pus and peritonitis can only be likened to a fireman who would stand, hose in hand, and contemplate a beginning conflagration in your house, and withold the stream for fear of soiling the rugs." He continues, "The much talked of *interval operation* is a wise thing to advocate for a patient *during the interval*, but it is sometimes a most dangerous thing to advocate and wait for *during an attack*; any treatment that goes sparring for a diagnosis during the first thirty-six hours, and manœuvring for the advantages of an

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interval operation during the next few days, and then closes for a life or death struggle on the fifth or sixth day, will largely increase the death rate. There *is* an interval—but not the one so often alluded to—that should interest the patient and the physician; this is the interval between the onset of infection in the appendix and the time when a dangerous amount of the infection would be found outside the appendix if it were not removed. The interval between pus in the appendix and pus everywhere. This is the interval which, had a surgeon's knife cut short the possibility of pus everywhere, would have saved the lives of most of these patients."

"A patient going through an attack (suppurative) without operation is saved by adhesions—adhesions are life-saving at the time, but death-dealing afterwards. Waiting treatment favors adhesions; early operation avoids them."

I think, therefore, that no one should argue against the early operation, *before* serious trouble has set in. The risk of operation at that time is so little, the wound need be so small, the convalescence so speedy, the chance of post-operative hernia so slight—in fact, the operation which saves is less painful, less irksome to the patient, than the attack which may kill. It all hinges on the diagnosis; if the case is diagnosed early, then operate at once. Of course if an error has been made, and a correct diagnosis not arrived at until the disease has ceased to be limited by the appendiceal walls, then no hard-and-fast rule can be laid down for guidance; but it must be considered a misfortune that the case has not come for operation sooner. This is where judgment and experience will tell, for it is on these that our decision as to operation must now rest. When an acute case comes first under notice, the attack having lasted a day or two, at the Sydney Hospital, the case is placed at once in a surgical ward, and if it is manifestly getting over the attack operation is not postponed, but delayed from hour to hour, blood counts taken, and convalescence from that attack hoped for, and then the operation done. On the other hand, operation is at once done if thought advisable; but one has to be very sure that the improvement is absolute, and not only "a lull before the storm."

Now come another class of cases—those that are no longer cases of appendicitis, or inflammation of the appendix, but have gone on to peritonitis or toxæmia. Mere removal of the cause will now do no good, and one has to judge of the amount of poison absorbed—that is, of the result of the poison on the system of that individual patient—and this depends on the virulence of the germ let loose, and on the power of that patient of withstanding the poison. In other words, will the patient stand the anæsthetic and operation on the top of his toxæmia, for in even the worst cases it is better to open and drain, if possible, the region where the poison is manufactured; but, in those cases where our judgment tells us that the patient will not stand the operation on top of his other troubles, then we must rely, not on medical treatment, but on "masterly inactivity"—the method of Ochsner being the best to follow—in the hope that the patient will overcome the toxæmia and allow of later operation.

Ochsner himself has not proposed his method as one of treatment of appendicitis—but, unfortunately, some medical men have allowed themselves to be misled into looking upon it as such—but only as aiding a desperate case in their fight with toxæmia, the result of neglected appendicitis. The method consists of keeping the patient at absolute rest, giving nothing by the mouth and little by the rectum, so as to check as much as possible the movements of the intestines; if necessary, washing out the stomach to check vomiting: in fact, the method consists of rest absolute, but to drain the part is the first essential in every case where it is practicable,

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Now, as to the removal of the appendix in pus cases. I think this should be done in every case, if possible, without adding too much to the risk or length of operation, and it is generally possible to a surgeon experienced in these cases. I know that such an authority as Treves has stated that after a severe suppuration most cases never have further attacks, but I think he goes rather far in that statement. Only the cases which result in sloughing of the entire appendix are likely to be free from further attacks. Those caused by a perforated appendix are more likely than ever to have further attacks. I have a specimen removed at a second operation. There was an acute abscess well walled off: I could not feel the appendix, so left it; subsequently I removed in one piece the old cicatrix with the appendix attached by its middle, the free end pointing to the enteronic area—distended, and looking as though it were again on the point of rupture. I was astonished to find that, after the lapse of nine months, all or most of the adhesions had become absorbed.

There is no class of case in which the experience and practice of an operator are more called upon than in this, so that in recommending early and frequent operation I only do so when it is possible to secure the services of a trained operator; for, treacherous as the disease is, I am not sure it would not be safer to trust to it rather than to the inexperienced judgment and surgical indiscretions of some self-appointed operator.

If we are to press early operation upon our patients, we must be able to tell them that in all probability the shock of the operation will be slight, that they will not be laid up in bed long (probably not as long as by waiting for the interval), that they will not be disfigured by a great and unsightly scar, with the tendency ever afterwards to a hernial protrusion and the necessity of wearing an irksome abdominal support. If we are, by our operation, to leave the belly wall so maimed that a hernia is a likely result, then it is questionable if we should ever advocate an operation except when our hand is forced; but it is quite an exception that a long incision is necessary, or even desirable. Of course the first requirement of an operation is to be life-saving, but if we can achieve that object and be artistic as well, then so much the better. I will go farther, and say that with a short incision we can do better work than with a larger one, when that incision is placed in the right position. I seldom fail to find and remove the appendix (I may say I have never failed except where I have thought it undesirable to try) with the least amount of shock and disturbance of the intestines, a speedy convalescence, and no tendency to hernia.

When I first began to use the one and a half inch incision I followed R. T. Morris, of New York, but since becoming more experienced I have modified somewhat his method. I have made the short gridiron incision in every case I have operated upon since 1900, whether acute or chronic, suppurating or not; and at times, to show that it was possible, I have made the incision considerably under one and a half inches. And my statistics are good; for though holding an appointment at a large hospital where we get all sorts of cases, I have not lost a case for three and a half years, and only one in private for a longer period than that: and I attribute this to the slight amount of traumatism necessary. With experience, the sense of touch becomes more accurate and more useful than the sense of sight, the appendix being more readily recognised, more easily freed, less damage done to surrounding parts, than when a full view is necessary to do the work. Howard Kelly quotes an operation of Professor Dandridge (14) of Cincinnati. He could feel a mass towards the middle line—cut over it, at the right border of the rectus, freed the mass, but before he could remove it had to tie a somewhat large vessel; the mass proved to consist of enlarged mesenteric glands (inflammatory), but

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he could not find the appendix; afterwards discovered it to the outer side of the colon. The wound healed, and then broke down, and about 15 inches of small intestine and cæcum sloughed away, leaving a fæcal fistula (which took four serious operations to close). He had tied off the blood supply of part of the ileum and cæcum by means of this sense of sight; whereas, had he depended more on his sense of touch, and started from the right place, he would at once have found and removed the appendix, and left the mass to the mercy of time and the peritoneum, and have avoided the disaster.

Of course this is an extreme case; but I quote it as showing the importance of *not* cutting over an inflammatory mass (for, never mind how large the incision, it is the way to find trouble), but beginning the search, not from where the appendix *probably ends*, but from where it *more than probably begins;* for the inflammatory mass is of little import, it is the cause of the mass which is our chief concern—remove that, and the peritoneum will attend to the inflammatory mass, whether it be composed of inflamed glands or thickened omentum. I have demonstrated this on many occasions. To cut down near the mid line, in the hopes of coming across the unknown position of the distal end of the appendix, even though there may be a mass, is a somewhat hazardous proceeding when one can keep to the colonic side of trouble and probably with ease find the proximal end, with a certainty of finding the cæcum, with its tæniæ waiting to act as a guide. It savors somewhat of setting a "black tracker" to pick up the tracks of an escaped criminal anywhere he can, rather than letting him trace them from the place where he was known to have been; besides, who would engage in a frontal attack when he can take the enemy from the rear?

The commencement of trouble is generally some little distance from the base of the appendix, and we should begin from there, where the parts are probably healthy. By using this method I have often dug an appendix out of the midst of an inflamed mass, put a drainage tube into the cavity I had left, and in a few days the mass had disappeared—and this with the smallest of incisions. This cannot be done by sight; it has to be done by touch. To use sight, a very large incision would have to be made, and probably the task given up; whilst, by keeping in touch with the appendix, it can be stripped out, or the mucous lining brought away, leaving the outer coats, but generally the whole organ can be brought away without disturbing the outer walls. If there is pus in the cavity, this is even more easy and quite safe, as one works only in the shut-off cavity, with the healthy intestines packed off with gauze. If the attack is fairly recent there is a natural cleavage line, which gives way easily. Experience has taught me that bleeding need not be feared, as we can tie the principal appendical vessel at the base, and if there is any oozing, can pack gauze around the drainage tube and so control it.

If the operator starts over the mass he will probably have to open up to the inner or enteronic side, with the added danger of contaminating this region, and when he has "come on" the mass he has then to open it up from above; he does not know what he is going through, in spite of his vision (perhaps the ileo-colic artery), and he has a large incision in the dangerous area crammed full of sponges, pads, and other paraphernalia. He says he wants lots of room—as it is safe—and at once proceeds to minimise his room by stuffing his wound chock full of pads, &c.; and, perhaps, after pulling his patient about for an hour, scrubbing the tender surfaces by ever-changing pads, finds that his appendix is not there, and is lucky if he has only to give up the search without having done any irretrievable damage. What I want to emphasize is, that it is not necessary to remove or open up the inflammatory mass, but the cause of that mass—leave the house untenanted, and nature will demolish it. Of

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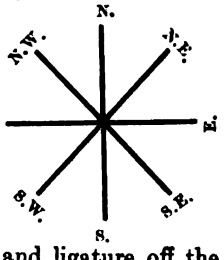
course if it were necessary, or even desirable, to remove all the inflamed surroundings of an appendix, then a large incision would become necessary, and no one could do the operation without it; but, to remove the appendix, it is not necessary, and it is astonishing how much can be seen through a small opening if desired. In ordinary cases, when the cæcum is drawn up, it acts as a plug to the wound, and there is seldom occasion to use even one small pad; the operation often is an extra-peritoneal one, with the exposure of a piece of the gut the size of half a crown.

I have seen some appendix operations done when it was hard to realise, except for the pads and blood, that it was not a *post-mortem* examination of the abdominal contents that was in progress; and have often wondered how much pulling about the human body *would* stand, and whether it was advisable to operate quickly, and whether the peritoneum did not improve by being scrubbed by the pushing in and pulling out of innumerable pads, and whether long continued æther administration did not improve the patient's vitality and kidney function. And yet people will argue in favor of big incisions. The operation I advocate is really the operation of R. T. Morris, of New York—a small incision, splitting the muscles, &c.; but I make the skin incision, not at McBurney's point, but further out—further out, I think, than any other surgeon. I start about half an inch above the level of the anterior superior spine of the ilium, and about 1 inch (in thin people, a little more) to the inner side, and directed *not* towards the rectus but almost parallel with its border, towards Poupart's ligament. This I have found the best place to make the incision, and, in ordinary cases, one will at once come on the appendix; if one does not, the cæcum at least will appear, and the tæniæ act as a guide; if we have to search for the appendix, the nature of the attack will aid us somewhat as to the location of it when the base is hidden, as it sometimes is. Guided by this information, I feel for it first in one direction, then in another. If the attack is an acute or sub-acute one, it is probably situated in the S.E. direction, less often N.E., and least often E. If the attack is a chronic one, it is probably S.W. or N.W., going by the compass on the descriptive plan suggested by G. R. Fowler some years ago: taking the points of the compass and fixing the N. upwards, S. downwards, E. towards the middle line of the body, and W. pointing outwards. But generally we can fix the base and feel for the tip, which can generally be felt and recognised by its thickened and bulbous extremity, and can be freed and hooked up to the surface with the finger; if too fixed for that procedure, it can usually be dug out from its bed; if surrounded by omentum, the whole mass can be often coaxed out of the wound: in these cases the appendix is generally pointing E., is a perforating or perforated one, and superficial. I wall off the enteronic area with gauze, shell out the appendix, and ligature off the omentum. If there is pus, I leave the gauze and put in a drainage tube. Sometimes the appendix is buried in the wall of the gut, or in the muscles to the outer side, or behind the cæcum, the cæcum having also become attached, hiding the base of the organ; in these cases sight or the size of the incision will not help one in the least (though the position of it will), and it can only be found by the sense of touch. In these cases I do not try to see. There are cases in which, though we locate the organ, it seems next to impossible to remove it; but, by perseverance and the aid of finger nails, we can generally get it either with or without its external coats; in fact, I have yet to come across a case where I located and tried to get an appendix that I have failed.



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In cases with acute abscess, if I feel and recognise the appendix, I remove it, as I think it better always to do this where one can; but in cases of acute abscess, where I cannot feel the organ, I do not search too long, and hope that it has been a case of gangrene, and that it may have become destroyed. I simply swab the cavity well out and drain, making a counter-opening, if I think it advisable; an abscess containing a sloughing appendix is generally walled off. If I could locate the base and could not remove the appendix, I should be inclined (though I have never had such a case) to tie the vessel at the cæcal angle, and ligate the appendix, and leave it in the hopes that it would slough.

In acute fulminating cases the appendix is usually easily found and removed; if there is general peritonitis I make free counter-openings, and put in drainage tubes in the loins and above the pubes, but seldom wash out. Practice has taught me, purely on empirical grounds, not to do so, except in those cases without any limiting membrane and no adhesions, with a lot of thin fluid, like tea, with flakes of cream in it.

Dudgeon and Sargeant say, in their Erasmus Wilson lecture on the "Bacteriology of Peritonitis," that the surgeon has friends as well as foes amongst the bacteria (and so give scientific reasons for facts arrived at by experience), that the first bacterium to make its appearance in irritation of the gut is the staphylococcus albus; it is a bacterium of but little virulence, and its *rôle* is to call to the part many phagocytic cells which are able to overpower the Bacillus Communis Coli, which appears a little later on the scene, when the gut has become more damaged. To wash out, besides washing out also the defenders, may mean washing the Bacillus Communis Coli to healthy parts not prepared by the staphylococcus albus for their reception; and, therefore, to wash out is a bad policy. But that the phagocytes have no power over the more virulent organisms, such as the streptococcus pyogenes, or the bacillus pyocyaneus, so that in infections where these organisms predominate the only chance lies in a thorough washing out in the hopes of getting rid of them.

They also describe as harmful the practice of peeling off lymph covering the bowels, so damaging the underlying endothælium and leading to the formation of adhesions.

Conclusions.

Foreign bodies are not often the cause of appendicitis.

Factors causing irritation of the mucous lining of the intestines are frequent causes.

Pain generally commences at or near the navel, but may start anywhere in the belly.

Any pain in the belly should have immediate and skilled attendance.

Pain continuing for six hours, and tending to settle in the right flank or groin, should call for immediate operation. Operation at this time is attended by little or no risk when compared with the risk of waiting.

Recovery from an attack does not mean recovery from the disease: it means only a respite; each succeeding attack increases the danger and difficulties of operation.

Any attack may end in death, or in chronic ill-health.

Chronic appendicitis is very often the unsuspected cause of ill-health.

There is no medical treatment for appendicitis, any more than there is for a broken leg.

"Waiting" on the doctor's part, neglect on the patient's part, or occasional unavoidable error in diagnosis is almost the sole cause of death.

No one individual can expect (on the ordinary laws of chance) to be one of the very few who will get no further attack.

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(14) Dandridge, "Anna's of Surgery."

DISCUSSION ON APPENDICITIS.

DR. STEER BOWKER: I was very surprised to hear the statements made by Dr. Hamilton Russell.

To set out with the intention of removing the appendix in *all* suppurating cases was good; but there are cases where the wisest course is not to try to remove it, but simply to drain, and wait for a more favorable time. I am sure that to always remove it would, in some cases, turn the balance against the patient.

In certain cases the mere pulling about, and length of time necessary to do this, would destroy the patient's chance of life. I seldom have had to leave an appendix, and always feel much happier when it is removed; still I recognise the fact that there will be an occasional case where it is wiser not to try. My own practice is, when I can feel the appendix, to remove it; but do not waste time in hunting for it, as I believe speed in operating in these cases to be the chief factor governing the result.

With regard to his statements concerning the length of the wound, I can hardly think he meant what he said, for a huge incision is most undesirable, and never mind how accurately the incision is sutured, the drainage pack is not the only place where a hernia may result, for in a large incision nerves and vessels supplying the muscles are divided, and atrophy of the muscle prone to result in consequent hernia; besides which, every inch added to the incision renders more liable the formation of adhesion to the wound surface.

If the pus is thick, I may use a little more endeavor to get the appendix; but where it is thin and watery, I am more careful. If the appendix comes readily to hand and I can remove it without wasting time, I always do so.

CRITICISM ON PAPERS BY H. L. MAITLAND.

(1) *Appendicitis.*—The chief debatable points that have arisen in these very excellent papers on appendicitis are—(1) when to operate; (2) should attempts be made to remove the appendix in abscess cases; (3) the length and location of the abdominal incision.

First of all, when to operate.—I am of opinion that every case of appendicitis should be operated upon preferably in the interval.

If a case be seen in the first ten or twelve hours, then the appendix should be removed immediately, because the condition of the appendix and surrounding structures is not far removed from the normal; but quite another condition exists if the case is seen during the full blast of an acute attack. Then

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the appendix and the surrounding structures are loaded with inflammatory products and bacteria, and operation is better postponed till after the acute attack is over.

Of course, if special indications arise pointing to the necessity of operation, then it should be done. If the pulse rate becomes high (120), or a rigor, or the patient look ill, or if there be any doubt as to whether the patient will recover from the attack or not, then it is better to operate. I object to a hard and fast rule of operating immediately in every case.

With regard to the removal of the appendix in abscess cases, I am firmly of opinion that if the appendix is easily found, without separating adhesions, then it *should* be removed, but not otherwise. It is better to wait till the abscess has healed, and then at a later date remove the appendix, if necessary.

The incision that Dr. Bowker uses is the one I have adopted. Dr. Bowker removes his appendices through a very small incision, and by the sense of touch; and I know his results are excellent. I prefer to see as well as feel when I am removing the appendix, and use a slightly larger incision, but in the same place and on the same principles as he does.

SOME POSSIBILITIES OF NERVE CROSSING.

By Dr. Kelvington.

Some three years ago I commenced a series of experiments on crossing the popliteal nerves in dogs, with the object of accurately determining the result. These experiments are not yet quite complete, but certain conclusions can be drawn which seem of sufficient interest to bring before members of the Congress. I think they warrant the trial of nerve-crossing in certain suitable—and I should like to emphasize this limitation—cases of infantile paralysis, and some rarer forms of paralysis due to destruction of portion of a nerve. This particular branch of surgery is not quite new, as it has been applied in certain cases of facial paralysis by Ballance and Purves, Stewart, and others, and, in one case of facial spasmodic tic, by Robert Kennedy. The facial was in most cases implanted in the spinal accessory, though the hypoglossal has also been used, and is possibly the more suitable.

It is needless to discuss the unsettled question as to whether regeneration takes place in the peripheral end of a divided nerve autogenetically, or only by the growing down of nerve fibres from the central end into the peripheral trunk. It may be mentioned that the latter is now regarded as the more probable method.

Any nerve we can experiment on contains several kinds of fibres, for even a so-called purely motor nerve like the facial carries sensory fibres, which allow the state of tension of the muscle to be known. After dividing the radial nerve, Head found the de-nervated parts of the hand, which had been rendered totally insensible to cutaneous stimuli, still remain sensitive to pressure that deformed the sub-cutaneous tissue. This, which he designated "deep sensibility," was due to fibres running with the motor nerves.

The various kinds of nerves, after being divided, regenerate at different rates. After section of the popliteals in the dog, motor power commences to return in from two to two and a half months: sensation commences to return probably somewhat earlier. I have found it completely absent six weeks after division and immediate suture of the sciatic in the dog; but the so-called sensory nerves contain separate fibre for touch, pain, and tempera-

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ture. Head, in the Marshall Hall address of this year, stated that regeneration occurs first in those fibres which convey the sensation of extremes of temperature and of pain, while the ability to appreciate light touch and accurate localisation of pressure appears later. The regeneration of vaso-motor fibres is a much slower process. Professor Osborne and myself have found that vaso-constriction commences about 200 days after division and immediate suture of the dog's sciatic, and gradually improves. In a dog, whose external popliteal was implanted into a gap in the internal popliteal, regeneration of the vaso-constrictor fibres was well marked after an interval of 293 days. About the regeneration of the vaso-dilators, which of course run separately from the vaso-constrictors, nothing is yet definitely known.

In performing nerve suture it is important to use some absorbable material. If silk be passed through the substance of the nerve it is never absorbed, and, from its mechanical irritation, leaves an area of scar tissue around it which interferes with the growth of fibres from the central to the peripheral end of the nerve. Silk can be used in the larger nerves if it merely be passed through the nerve sheath, and does not penetrate the nerve substance. Even in these large nerves I prefer to add another suture of absorbable material, which goes through the nerve substance, taking a good hold of both ends. With small nerves like the facial only absorbable sutures are permissible, and even in large ones, like the popliteals, are preferable. Chromicised catgut, prepared by Lister's method, is the material I have used. Ordinary catgut is absorbed before the two ends of the nerve are at all firmly united, and they are apt to pull apart on movement of the limb with very disastrous results. Strict asepsis is essential in nerve suture. Suppuration in the wound causes the stitches to give way, and the ends of the nerve to come apart. Even mild infection of a suture, without any suppuration, may cause an area of scar tissue about it which gradually strangles the nerve fibres, leaving a very unsatisfactory function. I have had this occur in a dog in which the skin wound healed by first intention. No suspicion of anything wrong was expected till the function of the limb gradually deteriorated. On cutting down, a fairly dense fibrous area was seen where the suture had joined the central and peripheral end of the nerve.

Before describing the details of the experiment, the anatomy of the nerves used may be briefly mentioned. The sciatic nerve divides, at a variable point on the back of the thigh, into the two popliteals; but even in the sciatic trunk, for some distance, the fibres of the two nerves are distinct: the two are bound together by an areolar tissue to form the main sciatic. The external popliteal, which is much the smaller, passes over the neck of the fibula, and supplies the peronei and all the muscles on the front of the leg which extend the toes and flex the ankle: it also supplies skin sensation to nearly the whole of the front of the leg and foot. The larger internal popliteal supplies the calf muscles and the muscles of the sole of the foot, which flex the toes and extend the ankle: it also gives skin sensation to the sole of the foot. The experiments were arranged so as to allow of one nerve being implanted into the other. The central end of the implanted nerve was cut off short, so as to permit it to affect no junction with its own peripheral trunk After some time it was found a very useful function was obtained after this operation. The following are the details of some of the actual facts:—

Dog 1.—Both the internal and external popliteal were cut completely across in the left hind limb. As much as possible of the central end ($\frac{3}{4}$in.) of EP was excised. The central end of IP was sutured to the peripheral end of both IP and EP. After recovery from the anæsthetic, the animal walked on the dorsum of the foot owing to the preponderating action of the muscles at the back of the leg. This, unless carefully guarded against, results in the

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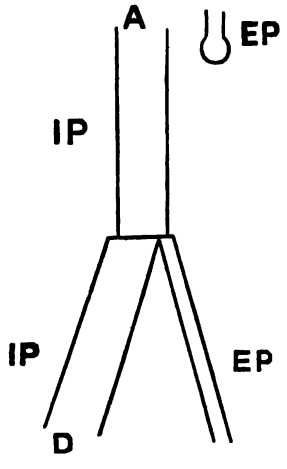
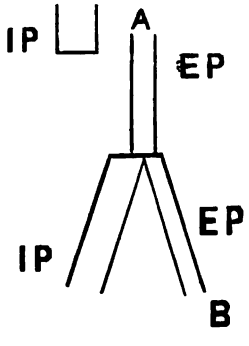
formation of an ulcer on the soft skin of the dorsum of the foot, which is easily abraded, and is now quite insensitive. The wound healed by first intention, function gradually improved, and at the time it was killed (139 days after the operation) the animal had a very serviceable limb. The finer co-ordinated movements such as scratching a particular spot, were the last to be recovered, and were not at all perfect at the end of four and a half months. On walking and running, however, it was not easy, even at this time, to tell the affected limb. On anæsthetising the animal, and cutting down on the popliteal space, the place of suture of the three nerves showed a slight swelling. The central free end of EP had a bulbous termination, but no strands of nerve fibres extended down to the place of suture. This was always carefully excluded, and, in some cases, the fibrous tissue below was examined microscopically to show the absence of nerve fibres. The stimulation of the nerves was performed with the Faradic current, IP was cut across at A, above the line of suture, and electrical stimulation here resulted in vigorous contraction of both flexor and extensor muscles. The peripheral part of IP was then cut across at D. Stimulation at A now resulted in movement of the extensor of the toes only. These are, of course, supplied by the EP. Thus the muscles normally supplied by the EP nerve are now innervated through IP fibres which have grown down the peripheral EP trunk.



In dog 2 a similar operation was performed, but the smaller EP was fixed to the peripheral ends of both nerves, about an inch of IP being excised. In this case it was impossible to prevent two small ulcers forming on the dorsum of the foot; but these subsequently healed. After nearly four months the use of the leg had been considerably restored, though the finer co-ordinated movements were not so good as where the larger nerve had been used. The animal walked fairly well, but occasionally the left foot would double over on walking. On opening up the popliteal space, and stimulating the nerve, it was found that EP innervated both flexors and extensors: thus stimulation at A produced vigorous contraction of all the muscles of the leg. After cutting through the EP at B, stimulation resulted in action of the flexors of the toes alone. In another animal, where the same operation was performed, stimulation after four and a half months of the EP at B produced remarkable results: contraction of the flexor muscles through the distal end of IP resulted. This is known as the axon reflex phenomenon, and its significance I intend to discuss later. In this last animal it was shown that no vaso-motor regeneration had taken place. The nerves of these two animals were imbedded in celloidin and stained by Pal's method, and the fibres counted. In one case the central end of EP contained 4,435 fibres, and the distal end 2,786: the distal end of IP had 7,196. In the second case there were 5,826 in the upper and 8,868 in the lower nerve. In other words, there were more fibres in the lower two than in the upper nerve. This leads to the remarkable conclusion that where one nerve is sutured to two distal ones the axis cylinders of the



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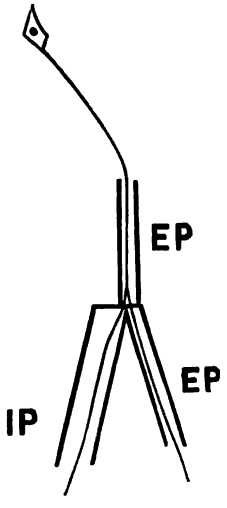
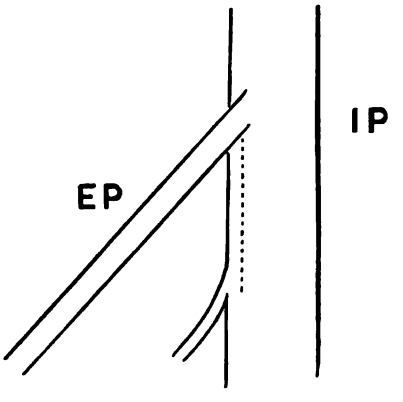
motor nerve divide, and a single fibre in the central end may join with two or more fibres in the distal nerve. The difference in number of the two is still more startling, if we remember that there are no vaso-motor fibres in the distal nerve while they are present in the central nerve. We may look on this as an attempt to compensate for the smaller number of nerve fibres which supply a muscle after this procedure. When a nerve is divided, and immediately sutured, the fibres of the central end are described as dividing and forming a kind of brushwork: these have a wavy course, and ultimately one branch from each axis cylinder joins with or grows down the fibre in the distal nerve. The rest of the branches from the central fibre gradually disappear. But if a more than corresponding number of fibres be present in the distal end, some of these branches, instead of disappearing, remain permanently, and have a definite use.



The axon reflex phenomenon shows that a single fibre from a motor horn cell in the spinal cord cannot only divide, but can divide and supply two different—and in this case antagonistic—muscles. (See Fig.) This can be the only explanation, as the electric stimulus can only be spread by continuity of a nerve fibre. I do not suppose that every dividing axis cylinder behaves in this way: probably is it exceptional. As any discharge from such a motor cell results in contraction of the two sets of muscles, it is evident that accurate co-ordination would be impossible with this method of suture. At the same time, some of the muscular contraction would be wasted, and dissociated movements impossible—or, at any rate, only possible by ceasing to use those muscular fibres which are thus innervated. Ballance and Purves Stewart's cases of implantation of the facial into the spinal accessory confirm this. The method of suturing the facial into a gap in the spinal accessory allows of this axis cylinder splitting. These authors say, from their experience of seven cases, "So far in our series of facial accessory cases we have not observed any independent movement of the face unassociated with that of the trapezius and sterno-mastoid (which are innervated by the spinal accessory nerve), though in several cases a minimal innervation of the trapesius is sufficient to cause facial contraction. Körte reported a case where the facial was implanted in a gap in the hypoglossal of the same side. He found that, after movement had been restored, any contraction of the facial muscles was accompanied by movement of the tongue. When the tongue was moved facial contraction also took place. This implantation of one nerve into a gap in another (as shown in the diagram) at first seems to offer a considerable advantage over the methods of completely dividing the nerve. It is obvious that the shock of the operation is less, and that the paralysis resulting from division of the nerve will not be complete, as some of the trunk has not been injured. But there are di-



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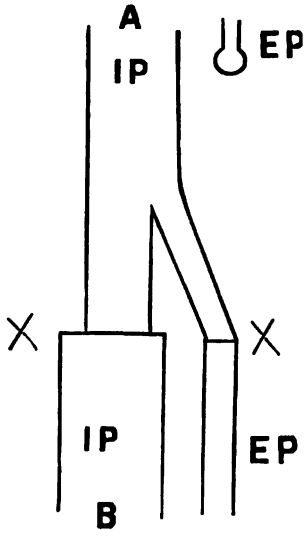
advantages which more than counterbalance the good effects of this procedure. In the first place, it does not do away with the associated movements owing to the peculiar form of splitting of the axis cylinder I have described. Secondly, it does not utilise to the utmost the capacity for division of the nerve fibres which occurs when one nerve is sutured to two distal trunks. An uninjured active cylinder cannot multiply in this way. I am unable to say into how many branches a single fibre can divide, except that it can establish connection with at least two fibres in the distal nerve. It seems also impossible to find this out even by the method of counting, as any nerve contains not only motor but sensory—and probably also vaso-motor—fibres: these may equally as well divide. There is no reason why the number should not be more than two. Possibly, too, the number of branches may be greater when there is a greater number of fibres in the distal nerve compared with those in the central nerve. From the less satisfactory result obtained when the small EP was used, it would appear that a limit for this proliferation is soon reached. Thirdly, with this method, some of the branches coming from the nerve on the side where the other is implanted in it may be mechanically or otherwise deprived of all fibres from the central end. This actually happened in one case I studied. A muscular twig (see Fig.), as shown by stained sections, contained no fibres whatever. This is a very serious objection, and I think of itself puts this particular operation out of the question.

My next attempt lay in the direction of abolishing or reducing to a minimum the associated movements due to the splitting of a nerve fibre, so that one branch goes to one set and the other to another group of muscles. The following experiment was actually proved to effect this object, and from a functional result was the best I have ever obtained. The left leg was operated on, and about 1 inch of EP excised. The IP was separated into two parts: the separation was made between the fascicular bundles, and as little damage as possible was done to the actual nerve tissue. These were then cut transversely across, and the smaller part was sutured to the distal end of EP. The larger one was then fixed by a single suture to the di tal end of the entire IP (see diagram). After about five months the function was almost perfect. Scratching, which I look on as the best test of accurate co-ordination in the dog, was done nearly perfectly. Professor Watson saw the dog, and was hardly able to say which leg had been interfered with: the animal could stand on and jump with the hind legs alone perfectly well. The dog was anæsthetised, and stimulation performed. The IP was cut across at A, and stimulation produced vigorous response in the muscles of the front and back of the leg. The distal end of IP was then divided across at B.



Stimulation at A now produced contraction of the extensors of the toes alone: stimulation of IP at B gave no contraction of the extensor muscles. This proves that no single fibre in the central nerve was supplying antagonistic muscles by the distal nerves. The muscles of the front and back of the leg were now dissected. They were well developed, and nearly as bulky as on the healthy side. I may mention, as a confirmatory proof of this device, that in another dog, where the same operation was performed, it was noticed after two and a half months that the co-ordination was getting rather worse. The wound was

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re-opened, and it was found that the nerve at XX had fused considerably in a common mass. There was also a good deal of scar tissue at this spot, as the catgut sutures had evidently been mildly infected. The two bulbs at XX were then separated, and the wound stitched up again. In a few days it was noticed that the function had greatly improved, notwithstanding that a good many fibres must have been cut in separating the fused nerves. The animal is alive at the present time; but the result is not a great success, for I believe the scar is contracting and strangling many of the nerve fibres. It will often be impossible to avoid injury of some few nerve fibres in this splitting operation, especially where small nerves such as the facial and spinal accessory are used; but even if we do this, it is only a few fibres at the angle of separation that will supply antagonistic muscles, and the associated movements will be reduced to a minimum. I believe it will be of great service in facial anastomosis, for here any associated movements are especially objectionable. It is also evident that, at first, associated movements will take place even with splitting until the co-ordinated mechanism has adapted itself to the new conditions. But this will probably disappear as the cortical or other centres become re-educated.

Having now decided on the best method in which the central end of one nerve is able to innervate the distal ends of two (and there is no reason why there should not be more than two), we may discuss its application in a very common form of paralysis. In infantile paralysis we have a loss of power in particular muscles as the result of destruction of a certain number of motor cells in the spinal cord. Skin sensation is quite unaffected. The foregoing experiments prove that a certain number of these motor cells can be dispensed with, and yet all the functions of the limb be efficiently retained. This suggests the idea that it is probable a good result could be obtained by suturing the nerve supplying the paralysed muscle on to the cut face of the proximal end of an adjacent healthy nerve. I think nerve-crossing would be especially applicable in the common form of infantile paralysis, where the extensors of the toes and peroneii are affected. The external popliteal being small compared with the internal, experience with the dog would lead us to expect a good functional result. Where the calf muscles were paralysed we should expect a useful though not so strong a limb as in the former case. There are three classes of cases where it is probable nerve-crossing would be inadvisable: First, in those where a large amount of recovery takes place naturally, with a fairly useful limb, it is very doubtful if we could produce a better result, even if as good a one, by nerve suture: a good many cases of infantile paralysis go on to almost complete recovery in the course of time. Secondly, in very severe cases, where only a few muscles remain intact (as, for example, a case where the peroneii are the only muscles of the leg to retain their function), it is very questionable if the fibres of the musculo-cutaneous nerve could innervate at all efficiently the whole of the muscles of the leg. In these cases arthrodesis, with anchylosis of joints and tendon transplantation, are our only resource. In a case with loss of power in the whole of the leg muscles it might be possible to bring the sciatic through the adductor muscles, and fix it on to the anterior crural; but, very probably, just as much good would result from anchylosis of the knee and ankle joints, rendering the limb a firm and rigid support in progression. Excellent results are obtained from the transplantation of tendons and muscles. It is to be noted that tendon transplantation merely distributes to advantage muscles remaining healthy. Nerve-crossing aims at *restoring* the affected muscle. Thirdly, nerve-crossing is useless in old cases of infantile paralysis. It is known that when a muscle has been cut off from its nerve supply for some time (about two years), little or no recovery follows suture of the separated ends of the

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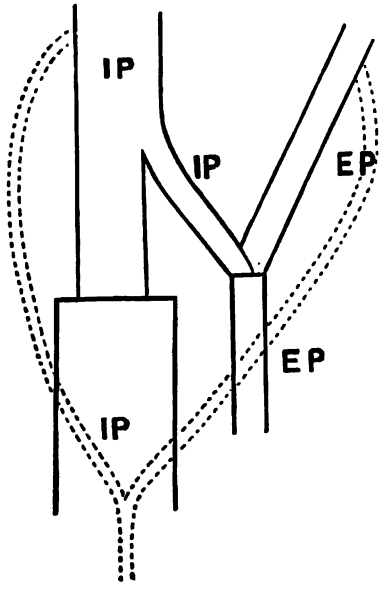
nerve—that is, as far as motor functions are concerned, for sensation can apparently be recovered after a much longer interval. This is usually put down to complete atrophy and degeneration of the muscle fibres, but it may be caused by changes in the delicate end plate of the nerve to the muscle. Somewhere about two years would appear to be the limit in which nerve implantation can be of use. It should be possible to come to an opinion as to whether a particular case merits trial of nerve-crossing in about six months at the outside limit, and often in a much shorter time. Meanwhile the nutrition of the muscles should be kept up by electricity and massage, so that if suture be performed they should be as healthy as possible. The electrical reaction of the muscles gives an idea as to how the case is progressing, and allows of an exact localisation of the injury to be made. The operation I suggest, in a suitable case, where the peroneii and extensors are affected, is as follows:—The internal popliteal nerve is first split into two parts, which are divided transversely across. The external popliteal is cut through at the same level, and the central end of the smaller separated part of IP sutured to the peripheral end of EP. At the same time the central end of EP should be fixed in the same place, as usually some motor fibres have escaped, and it is advisable to use them if possible. Even if these be all destroyed, the EP still contains sensory fibres, which will regenerate along their old paths. The other central part of IP should now be sutured to the whole of its own distal trunk. Theoretically, we wish to interfere with the motor fibres only; but for anatomical reasons, this is obviously impossible. If any purely sensory branches can be spared they should be kept out of the way during the operation. In the present case, two sensory branches—the nervous communicans tibialis and fibularis—come from the IP and EP, respectively, and join to form the external saphenous, which give sensation to a part of the foot. It is all the more important, as the insensitive limb is liable to ulceration from pressure on the insensitive part: this is probably the origin of the so-called "trophic" ulcers, though the term "anæsthetic ulcers" would be a better one. The exact form of procedure will, of course, vary with the distribution of the paralysis, and can be only worked out for each case by carefully noting the individual muscles affected. There is a great deal of shock after any operation involving nerve-cutting, and several of my dogs have died from this within the first few days. All precautions against shock must be taken; and I believe cocainisation of the nerves central to where they are to be divided would be of the utmost value. This is done by injecting a small amount of sterilised 2 per cent. solution of cocaine within the nerve sheath: this produces a block, preventing afferent impulses reaching the vaso-motor centres, and causing a great fall in blood pressure. For the first three months there will be absolute paralysis in the parts supplied by the divided nerve, and great care will have to be taken to avoid sores from pressure. After about a fortnight—or even



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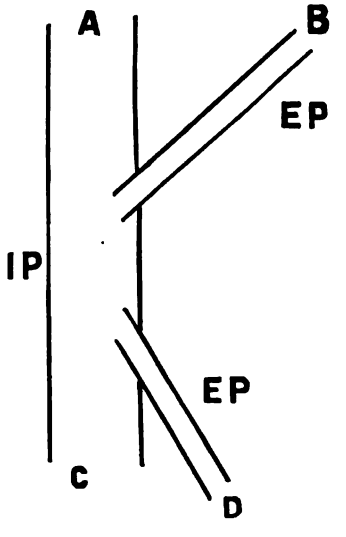
from the first—massage should be practised daily; but we should be careful not to put any strain on the sutured nerve for some time. In fact it would be safer to put the child's limb in a splint to avoid this. The amount of function returning will depend on the size of the healthy nerve available, and, probably, on the duration of the paralysis. In no case will it be quite as good as in the normal state. I have examined a good many nerves microscopically, before and after nerve-crossing, and find that the nerve is always smaller than in the healthy state. If a nerve be divided and immediately joined, the finest movements are never quite so accurately under control as before. It is probable that if a pianist had his median nerve severed, and shortly after sutured, he would never be able to command delicate effects on his instrument. But this would be of little consequence in the lower limbs, though in the hand and arm it may be a drawback. When a nerve is divided and sutured it is unlikely that exactly the same axis of the trunk is maintained, and many of the fibres in the central end will not grow down their old paths. Thus re-education will be needed to effect movement, and all such processes take place much easier in young people. In children the nervous tracts are not so fixed as in the adult, and alternative routes are comparatively easily developed. Though these experiments are not yet complete, enough has been done to warrant the trial of nerve-crossing in selected cases of infantile paralysis. If performed at an early date, it may prevent much of the deformity which results from this distressing condition.

The results of nerve implantation suggest that it will be a means of treating cases where a considerable length of nerve is destroyed by injury, involvment in callus, or tumor, &c.; provided there were a companion nerve available for re-inforcement. But it would be preferable to use the central end of the injured nerve in some way, as the motor cells in this case are intact. The following experiment was performed on several animals to test this. Very interesting results were obtained. About 1 inch of EP was excised. The IP was cut nearly half way through, in two places, and into these gaps the central and peripheral ends of EP were fixed by single sutures. After a time very good use of the leg was regained, and after about six months the function of the leg seemed almost perfect. The dogs were anæsthetised and the nerve stimulated. IP and EP were cut completely across at A and B. Stimulation at A with the Faradic current resulted in contraction of the muscles at the front and back of the leg: stimulation at B gave the same result, but less vigorously. After cutting IP at C, stimulation here, caused by the axon reflex contraction of the extensors and peroneii. In a second dog stimulation of EP at D resulted in movement of the calf muscles. It is to be noted, too, that the response in the extensor muscles from stimulation of IP at C is more vigorous than when the central end of EP is stimulated: in other words, the associated movements (which prevent co-ordination) are more active than the movements produced by EP alone. The condition of the nerves is evidently a very complicated one. After 300 days Professor Osborne and myself obtained regeneration in the vaso-constrictor fibres in the peripheral part of EP.



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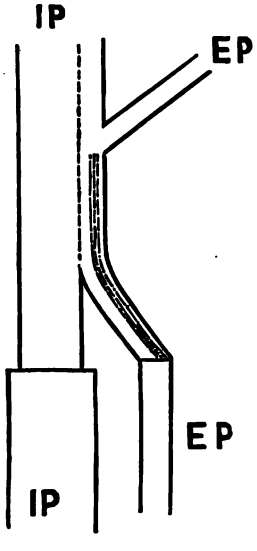
A very important point is that it is presumably a matter of indifference what length of EP be excised. After loss of a short part of a nerve trunk in a limb, the two ends can be brought together by stretching the nerves and relaxing the limb. An interval, of not more than 2 inches at the most, can be bridged over by procedures which offer an absorbable scaffolding down which nerve fibres can find their way. This is supposed to act as a guide to the fibres coming from the central end; but I am not aware that this has actually been proved. It seems immaterial what absorbable material is used for this purpose. A piece of nerve excised from some animal apparently does no better than several strands of catgut. But none of these methods can offer the inducement that a living nerve *in situ* does, for this exerts a definite chemiotactic attraction for nerve fibres to grow down it. When this interval of about 2 inches is exceeded, the resources of surgery are at present very inadequate. Probably even at this limit the functional result would be unsatisfactory. The very extensive operation of shortening the limb by re-secting the bone, to allow of the separated ends of the nerve being approximated, is advocated, but this results in mutilation and in a serious operation. Nerve implantation in these cases is almost ignored. Cheyne and Burghard (2) mention it, only to condemn it. The cause of failure seems to be due to the fact that only the sheath of the sound nerve is opened, and few, if any, of its fibres are divided. When the injured nerve is implanted in such a way it has no chance of getting any fibres to grow down its trunk. It is essential that a considerable gap dividing many fibres should be made in the sound nerve, and into this gap the central and peripheral ends of the injured one should be fixed. It is a very dubious point as to whether one ought to use the central end of the injured nerve, as in the above experiment. The hind legs of the dog do not allow one to judge accurately of the finer movements. As far as can be decided from these cases, it would be better not to use the central end in this way. Dog 7 showed that the contraction resultng from stimulation of the central end of the injured nerve was less vigorous than that resulting from the axon reflex; or, in other words, the fibres actually proceeding from the central to the peripheral part of the injured nerve produce less result than those which are coming from the sound nerve and splitting.



The alternative method allows of splitting of the healthy nerve, and does away with the axon reflex. It can be easily seen that certain strands in nerve occupy a particular position in the nerve trunk. For example, the external popliteal fasciculi occupy the outer part of the sciatic trunk, and can easily be dissected for a considerable distance up the thigh: in fact they seem to be anatomically separate up to the pelvis, and it is not uncommon to find the sciatic nerve coming out of the sciatic foramen in two trunks, the external and internal popliteal separated by some of the fibres of the pyriformis muscle. The same separation can be made for a shorter distance with the muscular twigs coming off the nerve trunk. To apply this to the condition in question, the piece of IP sutured to the distal end of EP can be separated up along the dotted line (see diagram). If a short cut be made in this for the central end of the EP to be inserted, some of the EP fibres would now reach their own distal trunk; at the same time none would

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run into the rest of the internal popliteal going to the flexor muscles, and no associated movements would result. Part of the split internal popliteal here plays somewhat the role of a graft (part shaded to right of diagram) *in situ*, while the rest of the split consists of IP fibres going now by means of the EP trunk to the extensors of the toes and peroneii. The actual condition of the nerves can be seen by a glance at the diagram. This form of nerve implantation would be chiefly applicable to the arm and forearm, or for anatomical reasons.

I have to express my thanks to Dr. Bird for taking charge of my paper in my unavoidable absence from the Congress.

THE TREATMENT OF CLEFT PALATE BY BROPHY'S METHOD.

By Dr. M. Morton.

Although descriptions of the methods originated by Brophy for the treatment of congenital defects of the palate have been published in America since the year 1893, its merits have very tardily been recognised in English surgical literature. In all probability this might be explained by the fact that Brophy addressed himself rather to dentists than to surgeons; and, in America, the dentist very commonly undertakes the treatment of oral as well as of dental diseases. At length, however, the merits of this excellent operation are beginning to receive the recognition at the hands of English surgeons which they deserve; and I can now refer you to a description of the operation in the *British Medical Journal* of June 24th, 1905, where Sir Thornley Stoker, of Dublin, records three cases successfully operated upon after Brophy's method, and to Mr. Edmund Owen, who recorded a successful case of his in the *Lancet* of December 19th, 1903, and recommends Brophy's operation in his recently published monograph on cleft palate.

Having recently delivered a patient of a child suffering from a complete cleft palate and hare-lip, I had much trouble in obtaining a good description of Brophy's operation. With the idea rather of assisting members of this Congress to a description of Brophy's methods than of recording my successful experience of them, I have thought that this paper might be useful. I shall not hesitate, therefore, to quote largely and freely from Brophy's published descriptions of his methods, because these lie, for the most part, in dental literature, not easy of access to surgeons.

The attitude of English authorities in the treatment of congenital palatal defects is, unfortunately, very conservative. A strange ignorance of Brophy's work still besets our standard authorities upon operative surgery, even in their latest editions. Probably, therefore, it is only fair to bear in mind that their opinions have been expressed in ignorance of Brophy's method, and with reference only to operation by the union of mucoperiosteal flaps.

Jacobson (*Operative Surgery*, 1902) says:—"With regard to the argument that cleft palates require operating upon in infancy, because of the difficulty in nourishing the patients, I would reply that this difficulty can be met by persevering care; and, when this is not the case, the little patient is not likely to be in a fit state to meet what is one of the severest operations in infancy."

With regard to the voice, while there is no doubt that the earlier a successful operation is performed the better will be the voice, it is possible that this has been too strongly put forward, to the exclusion of the other side of the

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question. Thus the possibility of an unsuccessful operation, with the inevitable loss of tissue and scarring of what remains, has been too much kept in the background, and children under 2 years of age cannot have contracted a habit of speech so bad as of itself to call for operation before this date.

Jacobson quotes the opinions of various English surgeons as to the most favorable period for operation, viz., Davies-Colley, who preferred the age of 14 months; E. Owen, for a soft palate, in the first six months, for a hard and soft palate together, in the second year; Clutton, before the second year; Arbuthnot Lane, during the fifth week; G. A. Wright, of Manchester, between the fourth and sixth years. Owen, for one at least, has probably been converted since to Brophy's opinions, as in the *Lancet* of December, 1903, he recorded a successful case operated upon by Brophy's method, and in his recent monograph expresses his complete accord with Brophy's principles.

Jacobson concludes: "Writing, as I do, for the guidance of many of my younger brethren, I consider that the *end of the first year* should be reached before a cleft of the soft palate should be operated upon, and then only under favorable conditions, and that the patient should be 2 *years old at least* before a *complete cleft* of the palate is operated upon. As experience is gained, operations may, no doubt, be performed successfully at an earlier date; but at any time during the first year of life the risk of failures is great, owing to the effects of hæmorrhage, the readiness with which convulsions are excited, the liabilities to bronchitis and diarrhœa, and, lastly, the delicacy of the soft parts and the readiness with which they tear."

Cheyne and Burghard (*Surgical Therapeutics*, 1901) say:—"The operation for a cleft palate demands more time than that for hare-lip, and entails greater loss of blood and more shock; and, for these reasons, it cannot be done with safety at so early a period of life." They advise, in slight simple cases, operation upon hare-lip within a few weeks—or even a few days—after birth; but in wide clefts, extending up into the nostril, that operation should be delayed until at least three months. They admit, however, that "it is essential that the cleft should be closed before the child learns to talk."

"Unless there are very strong reasons against it, the operation should not be delayed beyond the third year of life—the most favorable periods being from eighteen months to two and a half years. In some cases, where the cleft is not very bad, the operation may be performed before the eruption of the teeth—from the fifth to the eighth month. If both hard and soft palates be cleft, it is well, if possible, to unite both at the same operation."

Even the *International Textbook of Surgery*, 1901—although room for a brief and inadequate description of Brophy's operation is found—says:—"The operation for cleft palate and uvula is much more difficult and dangerous than that for hare-lip alone, *and should not be undertaken before the completion of the third year*."

As shown by reported debates of American medical societies, even American surgeons are not unanimous in their adoption of Brophy's principles, and Dr. Willy Meyer, at the New York Surgical Society (*Annals of Surgery*, February, 1904), said that he did not advocate operation before the end of the third month. Dr. Richard H. Harte, of Philadelphia (*Annals of Surgery*, June, 1904), says that the most desirable time to operate is about the time the child is beginning to talk, as the ability to make sounds will be more easily acquired. Operation upon children under 1 year of age is followed by a large mortality. Dr. James J. Young (*idem*) says that closure at the age mentioned is preferable to operation on the very young. In double hare-lip and cleft palate it is best to operate between the ages of 7 and 14, doing the plastic operation of the French.

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The first principle of Brophy's method is operation at as early an age as possible after birth. Brophy says (writing in 1901), "I am convinced—after making 570 operations for the cure of cleft palate, of which 211 were upon children younger than 6 months, the others being from 6 months to 52 years of age—that *the best age to select for operating is within three months after birth.* Of the children under 6 months, no deaths occurred. Two deaths followed operations upon children 3 years of age, one of which I am confident resulted from failure of proper care. Of the whole number of operations, two died, as above stated."

Brophy operates in early life for the following reasons :—

1. "The surgical shock is less, because the nervous system of the child is not well developed; that young children re-act better, and mental apprehension is eliminated."

All of us who have had any experience of midwifery work know in what an exceedingly "dilapidated" condition (if I may use the word) infants are very commonly ushered into the world, and how, even after prolonged, vigorous—and often violent—measures for resuscitation, the child in the course of an hour or two is in as good a condition as if born in an easy and rapid manner.

2. "Before the bones are fully calcified they may be bent or moved without fracture. Bone, at birth, is almost one-half organic matter; hence the injury is really less in closing a cleft than it would be if the calcification were more complete."

3. "The muscles are early brought into action, a good velum is secured, with consequent establishment of normal articulation when the child begins to talk."

4. "When the palatal processes of the maxillæ are united, it will be observed that the development of the bones of the alveolar processes of the upper jaw assumes a form nearly or quite normal, and when the teeth are erupted they will properly occlude with the lower ones, or nearly so."

Any resultant deformity of the teeth Brophy claims can be easily corrected by orthodontial methods; and he makes the important point, backed by very convincing evidence, that in nearly all cases there is sufficient tissue in both hard and soft palates, but the parts are not united.

5. "The existence of the hare-lip gives more room for work." Referring to the practice of early hare-lip operation and late cleft-palate operations, Brophy says :—"It is a great mistake to commence at the oral opening and partially close the only aperture through which a subsequent palate operation must be made. The surgeon needs all the space that can be secured."

For the following description of Brophy's method of closing clefts of the palate in the main I am indebted to my friend, Mr. R. J. B. Yule, D.D.S., of Melbourne, who was associated with Dr. Brophy in Chicago in the treatment of a great many of these cases. I have embodied a few points that my limited experience has suggested to me, and that I think will be found of advantage.

In addition to a skilled chloroformist, a good assistant is of great advantage. Two nurses will be required; and it is of immense advantage to operate in a well-appointed theatre of a hospital—public or private.

The child, having been anæsthetised, is wrapped up and pinned in a sterilised towel. If the weather be at all cold he is laid upon a hot water cushion throughout the operation. He is placed with the head hanging over the end of the table, with the face towards the light. A nurse undertakes the important duty of supporting the head with the palm of her hand. She sits, or stands, most conveniently to the right of the patient. The operator should be seated.

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After preliminary anæsthesia with the mask, the chloroform is continued with a Junker's inhaler throughout. As the tender skin of a young infant is easily excoriated, any strong antiseptic solution should be rinsed off the hands after sterilisation, in plain sterilised water or weak boric acid solution. The instruments should be taken out of sterilised water or weak boric acid lotion.

When operating upon the hard palate alone, a gag is unnecessary, and is more likely to be in the way: better exposure is obtained by a tongue depressure with a good curve. The tongue is thus kept out of the way, and sufficient force may be easily applied to keep down the lower jaw if the patient is well anæsthetised. I have not found Brophy's oral speculum necessary. For paring and suture of the soft palate, a Whitehead's gag is most useful.

After a preliminary cleansing and sponging of the face and mouth with boric acid lotion, raise the cheek, and—well towards the posterior extremity of the hard palate, just back of the Malar process, and high enough to ensure its being above the palate bone—insert, by means of a needle, a large braided silk suture, carrying it through the substance of the bone to the central fissure. When the needle appears in the cleft, pick up the thread which it carries with a tenaculum hook or forceps: withdraw the needle, leaving the thread *in situ*, and catch the ends of the thread in an artery forceps. One of the difficulties of the operation is the picking up of the thread from the eye of the needle in a small narrow space filled with blood. I think that the use of silk dyed black should render this part of the operation a little easier.

Through the corresponding part of the opposite bone pass a suture in same manner. Pass the one loop through the other and pull it out, carrying the other with it. Nearer the front portion of the maxilla, anterior to the Malar process, insert another silk suture in like manner. These can then be substituted by silver wires of No. 18 or 20 American gauge. The next step is to make lead plates, of No. 17 American gauge, and moulded to the convexity of the buccal surface of the bones. Provide these with eyeholes to correspond to the position and number of wire sutures. The protruded ends of wire are then passed through the lead plates upon each side: the plates are then placed gently in position. Trim up the edges of the fissure and also the edges of the palate bones. The wire sutures may then be drawn tight and twisted fogether, *i.e.*, the right end of the anterior to the right end of the posterior, and the same on the left side. With the fingers forcibly press the two maxillary bones together until the cleft is completely closed, and then the wires can be tightened to hold the parts together. If the edges of the cleft do not approximate, a further step may be taken. After the cheek has been well raised, divide the mucous membrane and bone through the Malar process. Carry the knife in a horizontal direction, and when well inserted sweep the handle backward and forward, thus dividing the maximum amount of bone and a minimum amount of mucous membrane. This done on both sides, the wire suture can then be tightened and the edges approximated.

Brophy adds —"The incision of the mucous membrane must be made as small as possible, as this membrane must serve to retain the bones in proximity, or to hold them nearly together . . . . . Separation of the bones is attended with very little hæmorrhage, which is easily controlled."

I would add myself that this division of the Malar process should be thorough—more free, probably, than Brophy's description would lead one to believe.

After approximation of the hard palate, the premaxilla will be dealt with as required to bring it into line with the alveolar border. The edges of the soft palate, which will have been refreshed at the same time as the hard

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palate, should now be closed by fine silver wire or horsehair sutures, and coaptation sutures of similar material inserted here and there along the hard palate, if required.

The after-treatment consists in absolute cleanliness, and careful spoon-feeding with sterilised milk. Frequent gentle syringings with weak boric acid lotion are required, and in the after-treatment a good nurse should always be obtained where possible.

Brophy says that the plates may be left in from two to four weeks. But, as they seem to cause little or no trouble, there is no reason why they should not be left for a longer period where much strain has been put upon the retention of wire sutures.

F. P., the subject of my operation, was operated upon June 11th, 1905, being then 14 days old. He suffered from a complete cleft of hard and soft palate and hare-lip on the left side. He was an otherwise healthy, well-nourished child, and weighed 9lbs. at birth. The child was anæsthetised with chloroform by Dr. H. M. Hewlett, was wrapped in a sterilised towel, and laid on a hot water bag. He was placed with the head hanging over the end of the table; the head being supported by the hand of a nurse throughout. A Whitehead gag was inserted, and the whole cleft was pared on each side. The silk sutures were then passed as above described, and stout silver wire substituted, No. 00 English gauge. The two lead plates, No. 17 American gauge, were then adjusted. The maxillæ were then forcibly pressed together, and the wire sutures tightened by twisting.

It was found that complete apposition was not possible, so Brophy's procedure of division of the Malar process was attempted. Better apposition was thus obtained, but not actual contact; a narrow cleft of barely one-eighth of an inch remained. I believe that a more thorough division of the Malar process would have given us complete apposition. The soft palate, having been freed at its junction with the hard palate, was then sutured with horsehair sutures; the smallest English semicircular surgical needle obtainable being used. The premaxillary bone was then forced into position, horizontal incision above the alveolus being necessary. This was retained in position by a silver wire suture. There was not much hæmorrhage, and the patient stood the operation, which occupied about three hours, very well.

I am obliged to Dr. Yule for giving me his skilled assistance, and the benefit of his valuable experience in the case.

The child had been spoon-fed from birth; partly with breast milk, which had been drawn with the breast-pump, partly with cow's milk. During the week following operation the child gained 7½ ounces, and during the subsequent week, 8½ ounces. A few days after operation the soft palate had completely broken down, but the anterior part of the hard palate united solidly.

On August 5th, a second operation was undertaken, the retention plates being still *in situ*. There being thus so little space available, it was impossible to use Brophy's method of approximation of mucoperiosteal flaps by means of lead plates. The whole of the cleft was pared, and mucoperiosteal flaps were raised from the hard palate, after longitudinal incision just within the alveolar border on each side. The whole of the hard and soft palate was united with fine horsehair sutures, threaded on Brophy's small needles. The hare-lip was then pared and closed. More blood was lost at this operation, but the child stood it well, and still continued to gain in weight during the succeeding week. The soft palate unfortunately broke down again, but the hard palate healed perfectly. The plates loosened, and were removed on August 19th.

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The child is now 3 months old. He has a good lip, a good alveolar border to his maxilla, and a solid hard palate. He is in perfect condition, and weighs 13½ pounds although still spoon-fed. The soft palate still remains to be closed on some future occasion.

I cannot imagine how such an excellent, although, as yet, partial, success could have been obtained otherwise than by Brophy's operation. Although the eruption of the teeth is still two or three months distant, the child's maxilla has been restored to a nearly normal condition, and there is still ample time to restore the soft palate long before articulation is required.

I think that there can be little doubt that Brophy's principles are sound, that the operation is a safe one, and that Brophy's operation is destined to be, in the future, the operation of selection in all cases coming under observation in early infancy.

For any who are anxious to find a full and particular description of Brophy's operation, inadequately sketched in this brief paper, I can refer them to the following, viz. :—

Binnie, J. F.—"Manual of Operative Surgery, 1905," where a short but clear description of the operation is given.

Brophy—*Dental Cosmos*, April, 1901, where an excellent description of Brophy's methods of treating clefts of both hard and soft palate is given.

Brophy—Reprint of paper by Brophy, "The Surgical Treatment of Palatal Defects," read at Columbian Dental Congress, Chicago, 1893.

Brophy—"Radical Cure of Congenital Cleft Palate," *Dental Cosmos*, September, 1899. *Boston Medical Journal.*

Owen, Edmund—*Lancet*, December, 19th 1903.

Owen, Edmund—"The Treatment of Cleft Palate," Monograph Series, 1904.

Stoker, Thornley—*B.M.J.*, June 24th, 1905.

EXTROVERSION OF THE BLADDER: ITS TREATMENT BY EXTRAPERITONEAL IMPLANTATION OF THE URETERS INTO THE RECTUM.

By H. Simpson Newland, M.B., M.S., Adel., F.R.C.S., Eng., Adelaide, South Australia.

At the last Intercolonial Medical Congress held in Hobart, Dr. Lendon, when introducing his original operation for the treatment of this distressing deformity to the notice of the profession, made the following prophecy: "I am inclined to think that it will prove to be the method of the future, subject, possibly, to some slight modifications." This prediction was made a comparatively short time ago, and it is too soon to say whether it will prove to be true. Maydl's intraperitoneal operation, or some modification of it, such as that of Borelius, is certainly more commonly performed. That very few cases have so far been treated by the extraperitoneal method is not surprising when the rarity of the deficiency is considered.

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With a view to calling the attention of surgeons to the advantages of extraperitoneal implantation of the ureters (uretero-proctostomy), this record of a successful case is published. Cases which have been operated upon by this method are collected in an appendix, which, however, lays no claim to completeness.

The following are the notes of the case which is the subject of this paper:—

H. G. W., a boy aged 7, fairly well developed for his age, was brought by his parents in March, 1904, for an opinion as to the advisability of an operation. The deformity of the bladder had been present from birth. At the age of 3 an operation had twice been unsuccessfully performed in the Adelaide Hospital. He had always enjoyed good health, apart from his deformity.

The family history revealed nothing of interest.

On examination, a pyriform area of bright red mucous membrane was to be seen in the pubic region. The stalk lay downwards and was formed by the penis, which was in a state of complete epispadias. Above and at the sides the junction of the skin and mucous membrane were well defined. The exposed surface of the bladder ended below in the cleft urethra, in the proximal portion of which the vera montanum could be recognised. A transverse sulcus, due to the folding of the urethral on the vesical mucous membrane, existed. When this was opened out by gently drawing the penis downwards, the vera montanum and ureteral orifices were more distinctly exposed. The latter lay at the summits of two papillæ, from which urine gushed forth intermittently, but seldom simultaneously. There was no ulceration of the vesical mucous membrane nor irritation of the surrounding skin. The cleft prepuce was very large. The scrotum was well developed. The testicles were incompletely descended; they lay below the external ring, and could be pushed into the scrotum. No hernia existed.

The pubic bones were widely separated. The gap could be felt distinctly, and was very obvious in a skiagraph. The pelvis was consequently flatter than normal, and the inner surface of the thighs, instead of meeting at a narrow angle at the perineum, were separated by a wide interval. The gait was therefore rather waddling in character.

The urine collected from the uncleansed surface of the bladder was slightly alkaline (phosphates); that collected from the ureters direct was acid, and free from albumen and pus. The thoracic and abdominal organs were healthy.

Dr. Lendon courteously granted me the use of one of his beds in the Children's Hospital, and kindly assisted me at the operation.

First Operation.—On March 29th, 1904, the following operation was performed after anæsthesia had been induced by the chloroform-ether sequence. The anus was dilated and the rectum washed out with saline solution. The large bowel had previously been emptied by a purge followed by an enema. To better expose the ureteral papillæ a silk retraction suture was passed through the glans penis and given to an assistant to hold. The vesical ends of the ureters were then freed according to the method employed by Peters. A No. 3 Jacques catheter was passed into each ureter for about 2 inches and fixed to the ureteral papilla—the one with a fine silk, the other with a fine catgut stitch. A circular incision of as great extent as possible was then made through the mucous membrane around each papilla. This was deepened until the bladder wall was completely cut through. The separation of the ureter from the loose cellular tissue, in which it lay along the wall of the pelvis, was easily effected. Each ureter was exposed for 1½ to 2 inches. With the left forefinger in the rectum and the right in the suprapubic wound, the

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anterior and lateral walls of the rectum were defined. A Lister sinus forceps was passed into the rectum, and its fine point thrust through the lateral wall about 1½ inch above the anus. The small wound was dilated by opening the blades of the forceps. The catheter in each ureter was grasped by the sinus forceps and drawn through the wound in the lateral wall of the rectum on the corresponding side. The ureter followed the course of the catheter, the rosette of vesical mucous membrane passing through the wound in the rectum, much as a button passes through a buttonhole. With a large rosette of mucous membrane and a small rectal wound there is no danger of the ureter slipping out of the rectum, and there is no necessity for a restraining suture. The catheter in the left ureter was forthwith removed. The rosette on the right side was not a large one, and it seemed better to leave the catheter in the ureter. The remainder of the vesical mucous membrane was not interfered with. The wound left by the transplantation of the ureters was gently packed with iodoform gauze. The free end of the catheter in the ureter was passed into a bottle containing boracic lotion. The operation, though tedious, was well borne, and the patient did not suffer from shock. A mixture containing salol and urotropine was ordered to be given every four hours. Urine commenced to flow from the right ureter immediately after the operation. Seven hours later the patient passed some blood-stained urine by the rectum, and during the night 1 ounce was drawn off by catheter passed into the rectum. The catheter in the right ureter was removed the day after the operation, and the rectum washed out with boracic lotion, and this was ordered to be repeated daily. Two days after the operation he could retain his water from two to three hours during the day, but was troubled with incontinence at night. He gradually acquired more and more control over the flow of urine, until at the end of the month he was able to remain dry all day, and only occasionally wet his bed at night.

On May 6th he developed a temperature of 102°, and for the next three weeks his evening temperature ranged between 101° and 104.8°. The child lost his appetite, he became apathetic, and his weight diminished rapidly. The tongue was red and dry, and the abdomen full. A swab from the throat and a "Widal" were negative. The temperature reached normal on May 27th, and from that day he rapidly gained flesh and strength, and was discharged from the hospital on August 31st. He was still troubled with occasional nocturnal incontinence. I believe the pyrexia to have been due to an attack of ascending nephritis.

Second Operation.—He was re-admitted in September, and on September 30th the remaining mucous membrane of the bladder was dissected away. The raw surface was diminished in size by the insertion of transverse Halstead sutures of catgut. The skin was then freed all round and extensively undermined. As the skin could still not be sutured across the raw surface without tension, a curved incision, with the convexity upwards and outwards, was made on each side from the upper limit of the raw surface left by the ablation of the vesical mucous membrane to the groin. The wound, when closed, presented a triradiate appearance. At the same time an attempt was made to close the proximal portion of the urethra. An incision was made along the junction of skin and mucous membrane. A flap of the latter was dissected up on each side and sutured the one to the other. The skin was undermined and sutured over the urethral mucous membrane. The whole wound healed well, with the exception of the small portion lying over the urethra.

Third Operation.—A month later another attempt was made to establish a urethral canal. On this occasion the penile urethra was treated as well.

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The mucous membrane was dissected up on each side and sutured. The skin was joined over this. There was considerable tension, and union again failed. I therefore determined to use the redundant prepuce to remedy the condition of epispadias. In the previous operations on the urethra the attempt had been made to form a canal lined by mucous membrane for the possible future escape of prostatic and seminal secretions. This idea was now regretfully abandoned. On December 21st the whole of the urethral mucous membrane was dissected away. An incision was made through the prepuce at its junction with the glans, and it was then stripped from the under surface of the penis. A large flap of skin was thus obtained. It was next buttonholed and the glans penis passed through the opening. The flap was then sutured along the dorsum of the penis. On this occasion the wound healed soundly.

I have collected eight cases which have been operated upon by the method of extraperitoneal implantation of the ureters: two died, giving a mortality of 25 per cent. Orloff has collected fifty-six cases of Maydl's operation with eleven deaths, or a death-rate of 20 per cent. These figures, however, only represent the published and not the real mortality of the operation. I know of several unpublished cases of both operations in which death has ensued. Consequently these statistics are of little use in gauging the relative merits of the two operations. We must, therefore, be guided by other considerations. In Maydl's operation and its modifications it must always be an objection that the peritoneal cavity is opened. The ureter is anatomically an extraperitoneal canal. In Orloff's statistics, of the eleven deaths four were due to peritonitis, and in the forty-five cases which recovered seven suffered from fæcal fistula. Peritoneal infection does not occur in the extraperitoneal operation, and fæcal fistula has not been recorded.

A risk common to both operations is that of ascending ureteritis and pyelonephritis. In the eight cases I record the two deaths were both due to this cause—one five days and the other two months after operation. Of the forty-five cases of Maydl's operation that survived more than three weeks, five died from pyelonephritis. It is argued that ascending infection in Maydl's operation is rendered less likely because the ureters are transplanted without disturbing their terminations in the bladder. As a matter of fact, in the extraperitoneal operation, if as large a rosette as possible of mucous membrane be isolated around each ureteral orifice, very little injury is done to them. Pyelonephritis is therefore no more likely to occur in the extraperitoneal than in Maydl's operation. Borelius has modified the latter operation by performing a lateral anastomosis of the limbs of the pelvic colon and then grafting the trigone of the bladder and the ureters into the end of the loop. It is considered that the lateral anastomosis will prevent the passage of the intestinal contents over the ureteral orifices, and diminish the liability to kidney infection. This passage, however, will occur unless, as has been suggested, the lumen of the afferent limb of the sigmoid loop be obliterated just distal to the anastomosis. Experience will show whether pyelonephritis will be prevented by this manœuvre, which certainly adds to the severity of the operation.

At best the intraperitoneal operation is a tedious and difficult one, and attended as a rule by shock. The extraperitoneal operation, though at first sight difficult, is in reality not so, and in my case at least was not attended by shock.

It is surprising how easily the ureters are separated from their surroundings when once the layers of the trigone have been divided around the ureteral papillæ.

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The ultimate results of the two operations are similar. The majority of cases can hold their water for several hours in the daytime. Some cases of nocturnal incontinence occur. The urine causes no irritation of the bowel. My patient, during his journey down to Adelaide to be shown at the Congress, held his water for six hours. He occasionally has nocturnal incontinence; the urine he passes then is clear and not discolored by fæces. The sigmoid flexure is said to form a better reservoir than the rectum. It must be remembered, however, that fæces are more constantly present in the sigmoid flexure than in the latter. When the development of the rectum is considered, it would seem to be a more natural reservoir for the urine than the sigmoid flexure. The weight of evidence, therefore, seems to favor the extraperitoneal operation. When either is successful, there can be no question that a comfortable existence replaces what has hitherto been a most distressing one.

From the patient's point of view the most potent arguments in favor of intestinal implantation of the ureters are:

1. Complete control over the evacuation of the urine is afforded.
2. An irritable and tender mucous surface is ablated.
3. The odor of ammoniacal decomposition of urine, so distressing to himself and to others, is abolished.

Intestinal implantation of the ureters also does away with the possibility of cancer supervening on the mucous membrane of an ectopic bladder. True, the gain in this direction may be balanced by a loss in another, for sufficient time has not elapsed to show whether intestinal implantation tends to cause cancer of the bowel. The following cases show that the supervention of cancer on ectopia vesicæ is no remote contingency:—

Case I.—Keitler* exhibited before a German medical society a woman, aged 55, with ectopia vesicæ and deficiency of the symphysis pubis; the right ureteral orifice could be distinguished, but not the left. A year and a half previously some tuberous masses were seen on the border of the bladder, and they had developed into a tumor the size of a fist, breaking down at several points. The case was quite inoperable.

Case II.—For permission to use the notes of this case I am indebted to Dr. Poulton, surgeon to the Adelaide Hospital. The patient, a man aged 60, it is interesting to note, had always earned his living by cracking stones. He first noticed a lump forming on the extroverted bladder nine weeks prior to admission. For a fortnight it had been very prone to bleed. He complained of considerable pain, especially down the legs. With the exception of the mucous membrane of the epispadiac penis, the whole of the extroverted surface was replaced by a prominent cauliflower-like mass the size of an egg, which bled freely when touched. The openings of the ejaculatory ducts could be distinctly seen, and exuded glutinous fluid. The ureteral orifices were lost in the growths. Both testicles were completely descended. The inguinal and external iliac glands were enlarged and hard. The condition was quite inoperable, and the patient died a month after admission, exhausted by pain and hæmorrhage. My microscopic sections of the growth and of one of the iliac lymphatic glands proved the tumor to be a carcinoma

In conclusion, I should like to say that abroad sufficient credit has not been given Dr. Lendon, of this city, for having devised this new method. It is usually known as Peters's operation. It is, however, only fair to state that Dr. Lendon performed his operation two months before Peters operated upon his first case in Toronto.

* Epitome, *British Medical Journal*, December 16th, 1905.

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Cases of Extraperitoneal Implantation of Ureters for Ectopia Vesicæ.

No.	Surgeon.	Date of Operation.	Age.	Sex.	Previous Operations.	Complications after Extraperitoneal Implantation.	Results.
1	Lendon, *Transactions Aust. Med. Congress*, 1901	May 12th, 1899	10	Male	Twenty-five previous operations	Right ureter slipped out of rectum, and was subsequently grafted into the sigmoid flexure	Recovery. In perfect health six years later. Perfect control over evacuation of urine.
2	Peters, *British Medical Journal*, June 22nd, 1901	July 15th, 1899	5½	Male	—	—	Recovery. Alive two and a half years later, and in perfect health.
3	Lendon, loc. cit.	July 21st, 1901	16	Male	Thirty plastic operations. Castration and radical cure of hernia on left side. Right nephrotomy, right nephrectomy, and later right ureterectomy	Anuria twenty-three hours. The ureter being kinked it was freed and reinserted into rectum, July 28th. Acute parotitis. Vomiting	Death from pyelonephritis, October 3rd, 1901, two months after operation.
4	Peters, *Canadian Journal of Med. and Surg.*, April, 1902	—	13	Male	—	—	Recovery.
5	Peters, loc. cit.	—	1	Female	—	—	Recovery.
6	Peters, loc. cit.	—	4½	Male	—	—	Death five days after operation from pyelonephritis.
7	Jacobson, *Journal of American Medical Association*, January 3rd, 1903	—	29	Male	Repeated operations	Urine contained blood and pus at operation	Recovery. Alive six months later.
8	Newland, *Australian Medical Gazette*, November, 1905	March 29th, 1904	7	Male	Two operations of uncertain nature	(?) Nephritis after operation	Recovery. In perfect health seventeen months later. Can hold his water for six hours.

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TREATMENT OF LARGE VASCULAR TUMORS BY THE INTRODUCTION OF ARROWS MADE OF MAGNESIUM-METAL, AFTER THE MANNER OF DR. PAYR OF GRAZ (AUSTRIA).

By Gavin McCallum, M.B., C.M., Glas.

Mr. President and Gentlemen—It gives me much pleasure to introduce to your notice a case which has been of considerable interest to myself, and I would fain hope to all of you, partly from the apparent hopelessness of the case and partly from a suggestion as to a treatment, of which, I must confess, until recently I was ignorant.

The case is one of a large vascular tumor (*Cavernous angioma*) occupying half the face of a young man.

There seems a general consensus of opinion that these are among the most difficult lesions with which the surgeon has to deal.

The suggested mode of treatment I refer to is that of Dr. Payr of Graz, *i.e.*, the introduction of little arrows made of magnesium-metal into the substance of the growth. The reason for using magnesium-metal depends upon its peculiar chemical properties.

I now show you a photograph of the case. The history is as follows :—

R.G., *aet.* 29 years, bank clerk. When about 18 years of age he first felt a tingling sensation at left half of upper lip, extending to the ear at that side. Later, the cheek swelled slightly, and the trouble was put down to a decayed tooth in the left upper jaw. This tooth was removed, but the swelling did not appear to be affected. The swelling continuing to extend, in about a year an attempt was made to remove it, but, owing to excessive bleeding, only a portion was removed. He was told by the doctor that it was an angioma. The growth continued to extend until within the last two years. During the last two years it has not extended, except for some puffiness at root of nose; and certainly there has been some shrinkage as regards the parts within the mouth. For some time after the operation severe bleedings took place from the nose, and, later, from the inside of the cheek. These bleedings used to take place with only a few days' interval, but have been very rare within the last three years. He has no notion of how it began, unless it was brought about from bad teeth in upper jaw. He did not receive any injury.

On examination there is seen a well-marked swelling of the whole left side of the face, from the lower jaw to the left temporal region, and from the left side of the nose to the left ear. Over the lower jaw there is seen the scar of the operation mentioned above. There is well-marked projection forward of the eyeball, with total blindness. The tumor itself is diffuse and smooth. A puffiness at the root of the nose is evidently due to enlarged veins; and there are seen large veins passing down to the neck from the tumor. In the left temporal region there is a well-marked pulsation, with puffiness. Blue veins can be seen over the anterior portion of tumor, but the pulsation is not so marked, and the skin is fairly natural in color. The tumor extends into left nostril, and blocks it up. It also extends into the roof of the mouth, nearly to middle line. The tumor formation would seem to have occupied all the region occupied by the superior maxillary bone, and to have bulged outward from it in all directions. The bones themselves have been displaced by the tumor in its growth, as can be readily detected on palpation. It would seem as if the growth under zygoma had pushed it outwards, making it occupy the superficial part of the tumor. At the roof of the mouth the left side of hard palate is replaced by the soft mass, except for a ridge along near the line of the teeth.

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Hearing is lost to air conduction, but not to bony. The drum membrane is tightly indrawn. The Eustachian tube would appear to be blocked.

The tumor formation would appear to occupy the position of a portion of the distribution of the external carotid artery, and more particularly the internal maxillary branch. There is a well-marked venous thrill to palpation just below the left ear, and a marked venous hum all over growth on auscultation. In the region of the zygoma and cheek the projecting bones prevent the tumor varying in size, but above and below that region great variations take place. Here the dilated blood vessels, which evidently constitute the mass of the tumor, appear at times to empty themselves, and at other times to become quite engorged.

In a reference notice to a German journal I observed that Dr. Payr of Graz, in Austria, reported a similar case, which has been very much reduced in size by his method of introducing magnesium darts or arrows. He inserted forty-two in six sittings. Pieces of this metal introduced into the living tissues produce certain definite chemical changes, which affect the absorption of the metal. Under its action the watery constituents of the tissues are reduced to the original elements, oxygen and hydrogen. The gases evolved accumulate around the metal, the oxygen combines with it, and forms a soluble magnesium oxide. The hydrogen is absorbed. The arrows are absorbed in from two to three weeks.

After injection the swelling gradually loses its softness to the touch, and in the course of a few days gaseous crepitation can be elicited around the foreign substance on pressure. This is apparently due to hydrogen, which is gradually evolved and then absorbed. Obstruction to the circulation takes place apparently over a considerable area.

Gradual cicatrisation follows in the area, with consequent diminution in size. The treatment is practically painless when done undel local anæsthesia, and seems to cause no ill effects, local or general. The patient can go about as usual.

By successive introductions large swellings may be thus reduced.

I noticed that the scar from the operation wound along the lower jaw in my case prevented the spread of the tumor downwards. On thinking over it, I determined to adopt this method of Dr. Payr's, and first of all introduced strips of magnesium ribbon half-inch long. I found the effects just as described by Dr. Payr. I placed myself in communication with him, and received the following reply :—

" Dear sir—I am glad to hear that you are using my method in the treatment of disease of blood-vessels. I am using myself only small needle-like arrows, pointed at one end and blunt at the other, which are inserted into the tissue by means of a tube and small rod. In case of need one can insert also arrows cut out of a magnesium ribbon, but they are far less efficacious, because the ribbon form of magnesium represents but rarely an absolutely pure form of the said metal. Arrows of magnesium, as I am in the habit of using, are to be got at Messrs. Rohrbeck Wien, 1 Kartnerstrasse 59. I enclose a copy of an article written by me for the *Centreblatt fur Chirurgie*, from which you will infer all the necessary further details of my operative *technic*. I have treated, from the time of those publications to this day, eleven cases of cavernous angioma, the results of all of which are excellent. In cases of a very large tumor there remains the spongious tissue, being shrivelled, sometimes a rather considerable nest of fibrous connective tissue ; cosmetic motives may at times lead to the excision of it. The largest angioma treated by me was about two fists in size, and occupied nearly half of the face of a young man. The recovery was nevertheless complete."

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I obtained from Dr. Payr his own apparatus, and have since been using it, injecting about half a dozen at a time. There is already well-marked improvement in the case, testified by appearances and by the patient's own feeling, both inside and outside the mouth. The patient himself is quite enthusiastic about it, and I injected half a dozen the day before I came away.

The length of treatment depends solely on the area involved. Any part of the tumor may be reached from the small opening made by trochar and canula.

The arguments in favor of this method would seem to be :—

1. It may be a gradual means of cure of large and otherwise inoperable tumors.

2. There is practically no scarring.

3. It seems to be a means at one's disposal of reversing the method of growth of those tumors, and causing a gradual cicatrisation.

4. By rendering the tissues less vascular, it may at a later date be operated on. It will thus be seen that this treatment neither excludes or forms a barrier to any other means of treatment. Excision becomes easier, less dangerous, and does not cause the same loss of blood. Galvano-cautery, thermo-cautery, electrolysis, and other methods of injection may all be employed without interfering with this treatment.

5. Only sub-cutaneous cavernous tumors are suitable for this treatment. I have referred to the skin of my patient having a fairly normal appearance, indicating its sub-cutaneous nature.

6. Dr. Payr considers the following vascular tumors unsuitable for this treatment :—Cutaneous angiomas and telangiectases, mixed vascular tumors, angiolipomas, angiofibromas, &c. These, as a rule, can be treated by other means.

7. Dr. Payr is of opinion that for cases such as my patient his method is the only one, and, at the same time, a very hopeful means of treatment in an otherwise hopeless case.

8. Dr. Payr has never had any trouble from embolism, or from the thrombotic process extending into the neighboring tissues. He has made a great many experiments on animals, and made a careful examination with regard to these possibilities. In the great number of patients whom he has now treated, he has never had any trouble. The bubbles of hydrogen gas in the tissues never caused the slightest anxiety.

CASE OF PAPILLOMA OF THE KIDNEY.

By A. A. Lendon, M.D., Lond.,

With Pathological Notes by W. R. Cavenagh-Mainwaring, M.B., Adel., F.R.C.S., Eng.

Mr. H., a pastoralist, was first discovered to have albuminuria in November, 1898 ; he was then 75 years of age, hearty, and well in most other respects. The urine had a specific gravity of 1020 ; it was acid and free from sugar ; in the slight sediment deposited, on standing, were seen, under the microscope, a few pus cells and some fine oxalate of lime crystals ; the quantity of albumen was found to vary from a faint trace to a marked ring by Heller's test. On February 24th, 1899, blood was found in the urine for the first time, but in small amount ; the smoky tint of the specimen suggesting a renal rather than vesical origin for it. The patient was noticed to be sallow-looking, but his

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weight was inclined to increase from its minimum of 9 stone 9 pounds, his usual weight being 10 stone 4 pounds. In the November of that year there was an attack of hæmaturia, spread over several days. It was attributed by the patient to a severe bout of influenza; no pain accompanied the bleeding, nor was there any frequency of micturition; rest in bed did not seem to appreciably diminish it, but it appeared to be best controlled by the internal administration of turpentine in capsules. From this date the bleeding became of a fairly frequent occurrence; a mild attack lasting a couple of days, and a severe one a couple of weeks perhaps. The intimate admixture of the blood with the urine, and the absence of clots, still supported the suggestion that it had a renal origin, and an endeavor was made, by sounding the bladder, to exclude the possibility of its being derived from that viscus. An interval between the attacks was chosen for that purpose, but nothing was detected, nor was any fresh hæmorrhage provoked. There was no renal enlargement nor tenderness. Sometimes in former years the patient had suffered from attacks of lumbago without hæmaturia, and now he often complained of a little pain with tenderness over the lower dorsal vertebral spines. In October, 1901, the correct diagnosis was thought to have been established, because the patient had a severe attack of left renal colic, which required hypodermic injections of morphine, and the continuous administration of chloroform for its relief. The bladder was again sounded after his recovery from the attack, but no stone found, so that it was assumed to have remained behind in the renal pelvis. This attack and its accompanying bleeding were supposed to have been precipitated by a ride on a rough horse on the previous day. The temperature was not raised, nor did the patient have a rigor.

When just 79 years of age, Mr. H. had an attack of suppurative appendicitis, and after he had recovered from his operation he began to put on weight, and finally reached 10 stone 12 pounds—a heavier weight than he had been for many a long year. In spite of his general and progressing weakness and the frequent hæmaturia, the weight had not materially decreased when he was last weighed in February, 1904.

In June, 1903, a correct pathological diagnosis became possible, because for the first time a piece of papillomatous growth was found amongst some blood-clots in the chamber. The renal colic two years previously further pointed to the passage of a piece of the growth down the ureter. Since that event, too, the bleeding had been observed to be much freer in character, and sometimes the passage of clots from the bladder gave rise to pain.

In May, 1904, an attack of bleeding, more profuse than ever, was followed by cystitis, with decomposition of the urine, and death ensued from exhaustion. A partial *post-mortem* examination was made, and the left kidney and the bladder were submitted to my colleague, Dr. Cavenagh-Mainwaring, for examination and report.

One remarkable feature about the case is this, that when in town the patient resided in a suburban street where the only other cases of papilloma of the urinary organs which I have met with during twenty-two years of practice in Adelaide also lived—one next door to him, and the other further up the street. Both cases were reported in the transactions of the South Australian Branch of the British Medical Association (*Australasian Medical Gazette*, 189_, pp. 171 and 174; also 1894, p. 300).

The question naturally arises as to whether an accurate diagnosis—anatomical and pathological—might not have been arrived at sooner. At the outset it might be said that the reported cases of this disease are but few, Dr. Drew in 1896 being only able to find records or specimens of eight instances. Again, neither Dr. Murchison nor Mr. Campbell de Morgan, in 1869, diagnosed their cases during life, nor was Mr. Barker's case in 1896 detected,

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even after a nephrotomy; Mr. Knowsley Thornton's was only found out after nephrectomy. On the other hand, Mr. Jones's case and Mr. Battle's were both diagnosed by means of an exploratory nephrotomy, followed in each case at a subsequent date by removal of the kidney.

But it may be said that with cystoscopes, urine segregators, and X-rays we have advantages over the men of the nineteenth century. This is true; and all these means should have been employed, had it been possible. But the patient was elderly, and objected to manipulations. He experienced no pain, except during the one attack of colic, and he argued that he was too old to be operated upon even if a stone had been found in the kidney. When the piece of growth was passed he was undoubtedly too weak for such a thing to be contemplated. In a younger man the cystoscope might, and probably would, have anticipated the discovery that the bladder was free from disease, and that the lesion was situated in the left kidney: the separator might, and probably would, have given us the same information. Private patients, however, and especially those who are elderly, cannot be treated quite in the same imperative manner as hospital inmates, greatly to their own disadvantage. An early diagnosis in such a case (had the patient been younger) would have been all-important: the kidney might have been removed, as was done successfully by Mr. Battle in a man 53 years of age.

PATHOLOGICAL NOTES BY DR. CAVENAGH-MAINWARING.

The left kidney was increased to about one and a half times its normal size, and consisted of two portions. The upper portion, forming about two-thirds, was dilated into a thin-walled sac, with thin walls somewhat resembling the capsule of a hydatid, and about $\frac{1}{4}$ inch in thickness. Its interior was filled with a bloody mass of mixed fibrin and débris, and scattered over the inner surface were numerous shaggy masses of papillary growth, infiltrating the wall of the sac at their base, the whole being divided by incomplete partitions into three saccules. The lower third comprised a single saccule, the walls of which were formed of healthy kidney substance enclosing a somewhat dilated pelvis, into which four flattened papillæ projected. The internal wall of this portion was smooth, except at one spot above where the cavity communicated with the sac of the upper portion, where was placed a single compound outgrowth similar in nature to those found above. The two portions were drained by separate ureters; the upper one was dilated, and with thicker walls than the lower one, whilst scattered all the way down were numerous implantation papillomata. The lower one was somewhat smaller than normal, with unthickened walls and a smooth lining. Three ureters had been torn away from below from their entrance into the bladder, so that it was impossible to tell whether they had a double entrance there or not; but it appeared more probable that they coalesced close to that viscus.

The bladder itself was somewhat thickened and contracted; its mucous membrane presented some general thickening and roughness, most marked in the neighborhood of the opening for the left ureter, but the walls beneath presented no evidence of new growth. Posteriorly the bladder was adherent to the rectum by a thickened mass, apparently inflammatory in origin.

Microscopical sections through the wall of the upper dilated portion of the kidney showed that it was composed almost entirely of new growth, the proper kidney substance being represented only by a narrow fibrous stratum outside this. The growth itself showed externally an abundant fibrous stroma, arranged in the form of alveoli, containing solid columns of cubical cells, resembling the glandular epithelium of the tubules, but more internally the alveolar structure became less marked, and the character of the cells

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became changed, in places becoming elongated and flattened, and somewhat resembling endothelium, and in others round and oval, with a general resemblance to sarcoma elements. When the actual relation of the stroma to the cells was carefully observed it was found that though the general appearance was an alveolar one, similar to that met with in carcinomata, still in nearly every instance the cells were not entirely separate and lying loose in the alveoli, but were at some point in intimate relation with its walls, at which point the stroma appeared to run into the mass of cells and to become developed directly into them.

From a pathological standpoint the most interesting question in this case is the nature of the growth; that is to say, whether it should be placed amongst the carcinomata or epithelial malignant neoplasms, or amongst the sarcomata or mesoblastic malignant tumors. The kidney is apparently partly developed by an outgrowth from the Wolffian duct, which is sometimes double, and partly by the development of glomeruli and tubules in a portion of mesoblast known as the intermediate cell mass, the two portions subsequently becoming connected with one another.

The ureters, pelvis, and collecting tubules are probably developed from the former (and when that is double there will be two ureters); the glomeruli, convoluted tubules, and rest of the kidney from the latter. Embryologists differ as to the exact origin of the Wolffian duct. It appears at a very early period of development as a thickening growing on the one hand in connection with the intermediate cell mass and with the epiblast on the other. This thickening soon obtains a lumen, and grows down along and at the expense of the epiblast to open posteriorly into the cloaca.

According to some this tube is derived primarily from mesoblast, whilst others think it is derived from epiblast, and becomes connected with the intermediate cell mass secondarily. Under the first theory the epithelium of the pelvis, ureters, and collecting tubules of the kidney would have a mesoblastic origin; under the second, an epiblastic one. This difference of opinion gives a special interest to the morphological study of the epithelioid tumors of the kidney, as possibly throwing some light upon the disputed question.

A carcinoma may be roughly described as an abnormal proliferation of epithelium in some part of the body, which is atypical in nature and situation and of unknown causation; and a sarcoma as a similar overgrowth of the connective tissue, the tendency of the new-formed tissue being to revert to the embryonic form and to become cellular rather than fully formed. In the carcinomata the stroma is not affected primarily, though a secondary irritative overgrowth may occur. The epithelial cells are the intrinsic and distinguishing features of the growth, and groups of these cells are found in close approximation with the intervening connective tissue, the groups lying in, but separate from, spaces in the connective tissue known as alveoli, the walls of which may, however, become infiltrated with the epithelial elements at a later period. The blood vessels formed in connection with such tumors have fully-formed connective tissue walls, and are only infiltrated secondarily.

In the sarcomata, on the other hand, the cells are directly derived from the connective tissue that forms the stroma of the neoplasm, and, consequently, the individual cells have a much closer relation with that stroma, and even when the alveolar structure of a carcinoma is closely imitated do not lie free in the alveoli, but are in places in intimate relation with their walls. The same tendency to reversion to the embryonic form is seen in the walls of the blood vessels, and these are, consequently, frequently actually composed of tumor substance, and the cells composing it gain a ready and early access to the blood stream, more especially the thin-walled veins. Consequently metastasis and secondary growths in sarcomata are frequently through the

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blood stream, especially to the lungs; whereas the carcinomata have to wait till they have penetrated into the vascular system before becoming disseminated; and as the lymph vascular system has the closest relations to the elements of the tissues, the path of dissemination is more frequently through those than through the blood vessels, though the latter may occur. The general opinion of pathologists as to the nature of these growths has varied from time to time. Previous to the work of Waldeyer the accepted opinion was that they arose by the proliferation of the connective tissue elements of the part; but since his researches the opinion has been widely accepted that they originated primarily from an abnormal proliferation of the epithelium. In a very able and interesting paper on the subject, delivered at the Brisbane Congress by Professor Allen and Dr. Cherry, the earlier view was upheld, and the histological features of this case are, I think, distinctly in support of the views expressed by them.

The first impression received on examining sections of the growth under the microscope is that it corresponds to the carcinomatous type of tumor; but, on closer examination, it is seen that though the cells be in definite alveoli, and free from the stroma wall in a large part of their circumference, still, generally, they are in contact with it in some part, and at that spot fibrous prolongations extend from the stroma wall into the midst of the cell masses, and are so intimately related to them that in many instances the cells seem to be developed directly out of the stroma. The type of cell varies considerably in different situations, and all gradations can be observed between those typically epithelioid down to those resembling ordinary sarcoma elements. The structure of the blood vessels inclines towards that met with in sarcomata, and in places the walls are composed of cells similar to those met with in the tumor proper.

The question of secondary growths was not settled at the autopsy, but it is an established fact that in similar tumors these frequently occur by the medium of the blood stream rather than the lymphatics, thus conforming to the sarcomatous rather than to the carcinomatous type of tumor; and, on the whole, it is my opinion it is in the former class rather than in the latter that such growths should be included. The condition of double and two separate portions of the kidney is an interesting one; and the extensive involvement of the upper portion and its ureter, and practical freedom of the lower portion, points strongly to the probability that, whatever be the causation of malignant growths—whether parasitic or a reversion to a germinal condition of the tissue—local conditions play an important part in the actual site of the growth.

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SECTION OF GYNÆCOLOGY AND OBSTETRICS.

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PRESIDENTIAL ADDRESS IN THE SECTION OF OBSTETRICS AND GYNÆCOLOGY.

By William S. Byrne, M.D., M.R.C.P., London, Brisbane.

Gentlemen—First of all, on behalf of the Executive of this Congress, allow me to welcome you to this the section of Gynæcology and Obstetrics; branches of our science and art which, during the past ten years, have made advances unexcelled by any part of the glorious profession of Medicine and Surgery.

The honor which has been conferred upon me by electing me President has been accepted with a full sense of my own shortcomings; but, at the same time, as a compliment paid not alone to myself individually but to the medical profession of the State to which I belong. When I look back upon the names of past presidents of this section I confess to a feeling of pride that I am considered worthy to follow a train of such eminent men.

Gentlemen, the history of Gynæcology is the history of Surgery, and the history of to-day depends upon the alphabet and language of yesterday. The practice of medicine is giving way largely to the art of surgery, and judging from the trend of comparatively recent events an elderly practitioner of the present day would be tempted to remark that the modern practice of medicine is surgery. And how true it is. I suppose most of us remember, in the old days when we were boys, being informed that Mrs. or Miss So-and-so was ill, or, in the words of that day, "delicate"; and we noticed that the lady in question was continually on the sofa, or in bed—being taken out for a drive on fine days, possibly—and that this sort of thing went on for years, and then our recollection of what happened grows faint and the case of Mrs. or Miss X has quite faded from our memory. What happened in those days to all the cases of pelvic adhesions, diseased tubes, pus cases, intractable retroversions and tumors? How often nowadays do we hear the remark from elderly ladies that in their time there were no operations, inferentially meaning that as none were performed in their day none ought to be done now? But our critics quite forget the years of chronic invalidism suffered by women in former times, and measure the ills of others by their own good health. The gynæcologist has been the pioneer in the forest of abdominal surgery since the time when Ephraim McDowell performed the first successful ovariotomy; but progress was slow, until the discoveries of Lister and Pasteur enabled surgeons to perform their work without fear of septic peritonitis. All our sympathies go out to Baker Brown in the early days of the Samaritan Hospital, when he abandoned sections on account of septic poisoning; and it is difficult for us now to appraise, at its true value, the work done, the courage displayed, the indomitable determination and the enduring perseverance shown, in spite of all obstacles thrown in his path, by that surgeon of surgeons, Spencer Wells. When we consider that not alone the public but even our own profession were violently opposed to the new procedure in the method of saving life by the removal of ovarian tumors, and that he was even threatened with a court of inquiry if a patient died after operation,

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his marvellous determination to succeed and his disregard of the bitterest attacks made upon him point him out to be not alone a surgeon amongst surgeons but a man amongst men.

Whilst acknowledging the brilliant gifts of Spencer Wells, let us not for one moment forget the still greater achievements of Pasteur, and our own countryman, Lister. Had it not been for Simpson and chloroform there would never have been a Wells; so, without a Lister and a Pasteur surgery would have remained simply the handmaiden of medicine, instead of becoming the virile and aggressive—but withal the kind—master.

Medicine and surgery are wedded, and the greatest triumphs of recent years have been won in the field of pelvic surgery.

Many of those amongst us will remember an article published not so many years ago entitled "A plea for the early removal of ovarian tumors"; and although we may look, with our present day experience, with some amusement at the title of that paper, yet it is an index to the feeling of the profession at that period. At the present time one is almost tempted to write "A plea for the early removal of uterine fibroids." It is still the tradition amongst physicians to view myomata as innocuous, harmless, and productive of but little discomfort; but the sooner this feeling is dispelled the better for our patients. Tradition is a good thing to have if we can only afford it, but in the treatment of fibroids it is apt to prove expensive. About five years ago a London specialist, in charge of the women's department at one of the large hospitals, published a paper emphasizing this tradition, and showing statistically that deaths from fibroids were most exceptional, and that removal except in urgent cases was not justifiable. I am glad to say that professional opinion is slowly veering round to surgical interference, and personally I strongly advise early removal. That uncomplicated myomæ can be removed safely at an early stage is beyond all question: the mortality is little more than that of an ordinary ophorectomy. The longer a tumor is allowed to develop, to contract adhesions, to block up the pelvis, to lessen resistance by continuous hæmorrhages, the more difficult and dangerous the surgical procedure becomes.

Who amongst us has not seen a wan, pallid woman—emaciated, unable to move unless it is to a couch, exhausted by continuous hæmorrhage, suffering from all the concomitants of the disease, a face drawn and disfigured by long and abiding discomfort, who has had drugs *ad nauseam* likely to alleviate the symptoms, and who, as a last resort, is handed over to the surgeon for operation? What are the probabilities of recovery from a long and difficult operation on such? We may view such a case with apprehension; but our duty is clear—we must afford the possible chance. Had the case been given to us in the early stage how different would our feelings have been on approaching operative measures. This is no fancy picture, but is fairly familiar to us all. When we know the primary dangers of fibroids—namely, malignant degeneration, decomposition and gangrene, chronic nephritis, phlebitis, embolism, the dangers of pregnancy and labor, and the secondary dangers produced by hæmorrhages, debility, and all the discomforts of an invalid life, rendering the subject an easy prey for intercurrent disease—we are warranted in looking on a myoma with the gravest apprehension and advising removal at a time when such procedure is not especially dangerous. The statement that fibroids do not kill may be true enough, but one might with the same confidence say that typhoid fever never kills: of course it does not, but the unfortunate patient dies all the same—of something else—either of pneumonia, exhaustion, heart failure, peritonitis, perforation, and a host of other complications, according to the fancy of the physician; but never of typhoid.

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CURETTAGE.

Amidst all this comparative safety in abdominal work, owing to the perfection of aseptic surgery, let us for a moment consider a minor operation which is performed daily, namely, curettage. I must confess I am always more uneasy and anxious after this so-called trifling operation than I am after a clean abdominal section. Salpingitis, pelvic abscess, chronic pelvic peritonitis, and a host of other ills follow in the wake of this procedure; and we are all cognisant of cases in which the cause of severe pelvic disease is the simple operation of curettage. The reason is not that the operation was improperly performed, nor that asepsis was not thoroughly carried out on the operator's part, but that in the majority of cases where it is necessary the interior of the uterus is already intensely septic; and as there are no means by which it can be made clean, the curettage opens up new avenues for infection, and a septic inflammation which was at first limited to the uterine interior extends to all parts of the pelvis. In another class of case there has been an old inflammation about the appendages, and the operation lights up the smouldering fire afresh, and much damage is done before the flames are extinguished. Curettage is not an operation to be lightly thought of or lightly undertaken—particularly in cases of old pelvic trouble—but rather to be considered with a full apprehension of its after effects; and, while not restricting its use to the specialist, I would warn the general practitioner that he may be called upon to perform a difficult vaginal or abdominal operation as a direct result of the primary interference.

RETRO-DEVIATIONS OF THE UTERUS.

The operations for the cure of retro-deviations of the uterus are becoming narrowed down to Kelly's ventro-suspensions, or fixation, and the Alexander Adams. Each procedure has its own especial advocates; but I cannot help thinking that, for the great majority of cases, a section is the better course to pursue. It is a great responsibility to condemn a woman to pessary-life; and I do not think I have inserted one of those awful implements six times during the past two years. The Alexander Adams operation is suitable enough for simple uncomplicated versions where there is no pelvic disease; but it is wonderful what a lot of unconsidered trifles there are in a pelvis when one has a good look at it. It does not quite lie with the operator whether a suspension or a fixation is to be the result; for I have meant to suspend on certainly three occasions, and years later, on having to open the abdomen for some fresh condition, I have found the uterus firmly fixed to the abdominal wall. In retro-deviation during the child-bearing period, when a fixation is a somewhat risky proceeding to adopt, I have been in the habit for some years of doing the following operation:—After the parts have been well exposed and any extra uterine trouble attended to, I free the round ligaments from the uterine attachment for about 2½ to 3 inches on each side, bore a hole with a pair of forceps through each broad ligament, pull the loops of round ligaments through, tie them together with linen thread at the back of the fundus, and insert a few catgut stitches through the now doubled ligaments and uterine peritoneum. This draws the uterus well forward, the ligaments are shortened at their strongest parts, and the risks of a future pregnancy diminished to a minimum. The operation is a little more difficult than a suspension or a fixation, but it seems to be more rational during the child-bearing period. I do not wish to claim priority for this little addition to the already great number of procedures in the treatment of versions, as a paper appeared in the *Australian Medical Gazette*, from the pen of a gentleman in West Australia, some months ago, in which much the same operation was described.

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LINEN THREAD.

As I have mentioned linen thread as a ligature and suture material, let me say that I regard it as superior to silk. It is finer, stronger, easily sterilised, can be boiled over and over again without diminishing its strength, it is inelastic, and does not break easily. In the Lady Lamington Hospital it has quite taken the place of silk for ligature material; in fact we have not used silk for a very long time. Of course catgut is the ideal material for buried suture, but where silk used to be always in evidence I now prefer linen.

ESERINE AND ATROPINE.

I have found the hypodermic injection of one two-hundredths of a grain of atropine just before an abdominal section, and one-fortieth of a grain of salicylate of eserine immediately after, to be of great comfort to patient and surgeon. It starts the peristaltic action of the intestines, subdues vomiting, and lessens the difficulty of procuring an evacuation on the following day—a great desideratum in abdominal work. Some months ago I wrote a short paper on the subject, which appeared in the *Australian Medical Gazette*, and further experience in the use of those two drugs has confirmed me in my opinion of their efficacy.

PUERPERAL AND ECLAMPSIA.

I was about to direct your attention to the treatment of puerperal eclampsia, but as I see there is a paper promised on this subject we shall be able to discuss it later. It has been aptly termed the disease of theories, and we are unfortunately no nearer the solution of the actual cause than we were when the connection between albuminuria and puerperal convulsions was first observed. Pathology does not lighten our darkness very much. In all cases we find parenchymatous nephritis with hæmorrhagic necrosis in the liver, thrombosis being primary and necrosis secondary; thrombi in the kidneys and lungs, pneumonia being common; hæmorrhagic extravasation between the muscle fibres of the heart, hæmorrhages in the stomach and intestinal walls and central ganglia. How much these extravasations depend upon the violence of the convulsions, and how much upon disease of the vessels arising from the circulation of highly toxic blood, it is impossible to say; probably both conditions favor their occurrence; but the main point is that the *post-mortem* appearances do not help us toward the solution of the *fons et origo* of the disease—they are only the effects. Can a chronic nephritis in a pregnant woman set up a puerperal nephritis, and can a puerperal nephritis cause a chronic nephritis? The general opinion answers these two questions in the negative, and I think that for our purpose we can consider puerperal nephritis a disease, apart altogether from the ordinary kidney inflammation. If recovery ensues after eclampsia the albuminuria disappears in the course of two or three weeks; and clinical experience shows that if the fœtus dies, as a rule the convulsions cease—that is, if the dose of the toxin has not been too large to be readily eliminated. Again, as in the acute nephritis of men and non-pregnant women, the so-called uræmic convulsion is not very common, whilst in the acute nephritis of pregnancy it is the rule; it is strongly suggested (although the kidneys are always seriously and acutely affected) that pregnancy is necessary for those eclamptic seizures, and, therefore, the real cause of the disease must be sought for in the fœtal or material structures *in utero*, and not in the kidneys, though both conditions are necessary for the production of the eclampsia. Intoxication, with the products of fœtal metabolism, seems to be the most feasible explanation of the entrance of the toxins into the blood; though one may be met with the objection that, if so, why should all eclamptics consist of 80 per cent. of

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primiparæ? I cannot help thinking that, somehow, nature after the first effort accommodates itself to conditions which have previously existed, and enables the excretory organs to eliminate the poisons.

In the treatment prophylaxis is the first consideration; and if we can prevent the onset of the convulsions our chief end is secured. The occurrence of a small quantity of albumen is by no means uncommon in pregnancy, so that although this sign by itself is not of necessity a danger signal, still it is enough to cause us a certain amount of anxiety as to future events. Purgation, a milk diet, and rest will probably tide us over the difficulty; but should a large amount of albumen be present or albuminuria, accompanied by œdema, or headache, or fainting, or visual disturbances, hyperemesis, or persistent hiccough, we may be fairly sure that eclampsia is not far off; and our duty is to terminate the pregnancy at once. It is not good practice to temporise longer; the sooner the feeding of the blood with toxins ceases the better. We should look upon the fœtus and maternal structures in this case in the same way that we look upon a decomposing placenta *in utero*—an offending substance, to be got rid of as soon as possible. Temporising where eclampsia is probable is a fatal policy; purgation, sweatings, diuretics and drugs of all kinds are useless as long as the cause is allowed to remain. It reminds one of the parson's remark when asked to pray for rain: he replied, "It's not a bit of good while the wind is in the west."

Eclampsia may occur *ante*, *intra*, or *post partum*. *Ante partum* eclampsia demands evacuation of the uterine contents as soon as possible, and the character of the convulsions influences the methods to be adopted for this purpose. If slight and not recurrent, one may be satisfied with passing two or three bougies into the uterus; or, if some more certainty and haste is requisite, dilatation and the introduction of de Ribes' bag. If the convulsions are very pronounced, and immediate delivery imperative, rapid dilatation by means of Bossi's instrument is indicated. Should it be impossible to dilate the canal to the required extent with a reasonable degree of safety, then Cæsarian section, either abdominal or vaginal, must be considered; and I am convinced that, in many cases of rigidity of the canal, section is safest and best for the patient.

Eclampsia *intra partum* must be treated on the same lines—extraction of the child and placenta at the earliest moment practicable, having due regard to the safety of the maternal structures.

In eclampsia *post partum* we have to deal with the results of toxæmia, and our efforts must be aimed at controlling the convulsions and eliminating the toxins. To fulfil the first condition morphine, in fairly large doses, commencing with half a grain, is to be recommended; in acute nephritis morphia can be given freely. It is in the chronic granular kidney that its use is contra-indicated. As well forty grains of chloral per rectum, repeated in an hour, is useful. Chloroform is of doubtful utility, though by its administration the eclampsia for a time is lessened; but its continuance for any lengthy period is harmful.

To eliminate the toxins free purgation is requisite, and I know of nothing better than croton oil dispensed in butter.

Where there is a bounding high-tension pulse venesection is indicated; but infusion of salt solution into a vein must be cautiously approached, as it increases blood pressure and may defeat the object we have in view. Pilocarpine has been to me a disappointing drug. I never could satisfy myself that it had any effect on the convulsions, though, theoretically, profuse perspiration ought to help in relieving the kidneys. If it is given, nothing much less than a third of a grain is of service.

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In conclusion, let me say how much I deprecate the opinions sometimes expressed, that nothing is any good for established eclampsia, *intra* or *post partum;* that we know nothing about it; and that our treatment, being empirical, is not likely to be of any use; that we should not deliver, but give baths and drugs, and pursue an expectant policy.

Gentlemen, pessimism in the treatment of puerperal eclampsia is to be condemned: want of faith in ourselves is the handmaiden of failure.

TREATMENT OF PELVIC SUPPURATION.

By Ralph Worrall, M.D., M.Ch.

Mr. President and Gentlemen—The opinions which I have ventured to express in this short paper are based upon my experience for many years past in the Sydney Hospital, and in private practice; but, for the purpose of illustrating my remarks, I have analysed and brought under your notice only the cases in which the abdominal cavity has been opened for suppurative conditions in the Sydney Hospital during the year 1904. I have done this because, in the Sydney Hospital as in all other well-regulated hospitals, the hon. medical staff collectively and individually have nothing whatever to do with the compilation of the annual report, a paid registrar being kept for this purpose. In the gynæcological report this, I fear, is but too evident from the out-of-date and confused nomenclature adopted. The report is sent each year to every registered practitioner in Sydney, and the work thus subjected to enlightened criticism. Anyone interested can verify the statistics with little trouble.

2. Of all the pathological conditions for which the abdomen is opened pelvic suppuration is the most frequent, and probably also the most dangerous and difficult.

In 110 cœliotomies performed by me in the Sydney Hospital last year forty-five were for pelvic suppuration in one form or another. I include in the term "pelvic suppuration" acute purulent salpingitis, abscess of the ovary, tubo-ovarian abscess, suppurating ovarian cysts, abscess in the uterine wall, in the cellular tissue of the pelvis and the pouch of Douglass (local circumscribed purulent peritonitis). In these forty-five cases of pelvic suppuration there were—Pyosalpinx 17, tubo-ovarian abscess 8, pelvic abscess 7, pelvic suppuration 4, acute purulent salpingitis 3, suppurating ovarian cyst 2, suppurating intra-ligamentary cyst 1, ovarian abscess 1, suppurating cellulitis 1, abscess of uterine wall 1.

3. In the forty-five cases there were two deaths; and as from fatal cases we usually learn most, I shall briefly touch upon them.

(1) Virulent streptococcus infection, with high pyrexia at time of operation. Vaginal cœliotomy was done first and several ounces of pus evacuated. As there was no improvement abdominal section was done a week afterwards, and bilateral tubo-ovarian abscesses removed. There was intense pelvic peritonitis and much exudation of lymph. The bowels were well moved afterwards, and there was no vomiting, but distension continued; insomnia was marked, and the patient became delirious, and died thirty-six hours after the second operation. Her history was that she had been curetted a week before admission for pelvic pains, which had troubled her for several months. There had been an abortion twelve months previously. A *post mortem* was made without throwing more light upon the case. It is evident either that

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the patient was poisoned at the time of the curettage, or that, more probably, this caused a leakage from pus sacs present since the abortion. [N.B.—By pelvic suppuration I mean a distribution of pus more extensive than in pelvic abscess.]

(2) The second death was a case in which criminal abortion had been produced ten days prior to admission. On the same day she had been curetted by one of the ostracised members of the profession. On admission to the Sydney Hospital, as an urgent case, an extensive sloughing wound of the anterior lip of the cervix was discovered. The temperature was 103, pulse 130, and resp. 36. Vaginal cœliotomy was immediately done, and about 2 ounces of pus evacuated; at the same time a considerable quantity of decidua was removed by curettage. Six days afterwards, but little improvement having taken place, the abdomen was opened in the middle line. The appendages were found to be acutely inflamed, but not cystic; the uterus was soft, friable, intensely congested; the left broad ligament contained an abscess the size of a large pigeon's egg; all the structures of the lower abdomen were congested and roughened. The uterus, appendages, and left broad ligament were removed. Death occurred in forty-eight hours. Numerous streptococci were found in the pus of the abscess cavity. A *post-mortem* examination disclosed nothing further. This was evidently a lymphatic infection, possibly from the wound in the anterior lip of the cervix. Both these cases are examples of the dangers of curettage undertaken without adequate care and knowledge.

Another case of special interest was one in which both streptococci and pneumococci were found in the pus of a bilateral pyosalpinx, and also in fluid which was twice drawn off from the right pleural cavity. The patient recovered completely, after extensive suppuration of the parieties. The origin of the trouble was undiscoverable.

With regard to the bacteriology, no note was made in twenty-one cases, which I much regret. In the other twenty-four there were no organisms found in the pus in nine, streptococci were found in five, B.C.C. in five, gonococci in four, and streptococci with pneumococci in one.

All the cases in which streptococci were discovered in the pus were clinically most serious; but, on the other hand, one or two cases in which the bacteriologist reported there were no organisms were equally critical, and at the operation showed as much congestion, softening, and friability of structures. The discovery of gonococci in only four cases inclines one to think that gonococci pus soon becomes sterile.

An attempt was made in each case to ascertain the origin of the trouble. In fourteen there was a history of gonorrhœa; in five of puerperal trouble; in three there had been recent delivery (all these proved to be serious cases): in six there had been recent abortion, and in three recent criminal abortion (these were also all serious cases): two were traumatic infections from curettage outside hospital; two were ovarian cysts with bowel infection; two were tubercular; two were uncertain origin, although streptococci were found in the pus. In the remainder the cause was undiscoverable. Pyrexia was present at time of operation in twenty-four cases, and within the week prior to operation in six others. The leucocytic count was increased in all the acute cases. One or both ovaries were not removed in fifteen cases. One tube appeared healthy, and was not removed in six cases. Two years ago I removed a gonorrhœal pyosalpinx, leaving the other tube, which appeared quite healthy; within a fortnight there was exacerbation of symptoms, for which the abdomen was again opened and this tube found distended with pus. Notwithstanding the risk of such an occurrence, I think it justifiable to leave a tube which is apparently healthy.

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In three cases only of the forty-five was there cellulitis with pus tubes. It was in the form of expansion of the broad ligaments from exudation into the cellular tissue.

There was one case of suppurative cellulitis without pus tubes, and it was cured by evacuating the pus through an incision, as for trying the external iliac artery. In every case curettage was done at the same time as the section, and in one instance the uterus was perforated by the curette; this was at once recognised, and no intra-uterine injections made. The case was one in which all the pelvic structures were much softened by septic processes, and would in any circumstances have required the removal of the uterus, which was done. The patient recovered. The uterus was thus removed with the appendages ten times: on eight occasions because it was so greatly involved in the infection as to make it appear dangerous to leave it, and twice because it was riddled with myomata.

The appendix was adherent to or incorporated with the pus sac in seven cases, and was removed.

The cæcum was once torn into where a pus collection was evidently just about to perforate. The opening was sutured with a continuous silk Lembert suture, in two rows, and no ill result followed. The parietal wound suppurated slightly once and extensively three times; in two of the latter there was a streptococcus infection.

In forty-four of the forty-five cases vaginal cœliotomy was done as a preliminary measure, either at the same sitting (thirty-three cases) or from six to fourteen days prior to the abdominal cœliotomy (six cases). In four cases only vaginal cœliotomy was done: once because it appeared to have effected a cure, both as regards symptoms and physical signs (puerperal case), and three times because the patients felt so well that they refused further operation, although at the time of their discharge from the hospital bi-manual examination disclosed the presence of the original pus foci. In my experience there is, sooner or later, a relapse in the vast majority of such cases.

On referring to my address as President of this Section at the Hobart Congress, nearly four years ago, it will be seen that the technique which I then advocated remains unaltered. Much has been written on the subject of "The treatment of pelvic suppuration"; there have been earnest advocates of the vaginal route and equally earnest advocates of the abdominal route, but I believe I have been the first to advocate and practise the systematic employment of both routes along certain well-defined lines. For the purposes of discussion I shall recapitulate these.

(1) In all cases in which it is evident from the symptoms and physical signs that pus exists in the pelvis, the treatment should be operative.

(2) When the patient is desperately ill, operation is done without a moment's delay.

(3) When it appears probable, after a careful examination and weighing of all the factors in the case, that the patient although seriously ill is likely to improve with rest and supporting measures, operation is postponed until the symptoms have subsided and the temperature become normal.

(4) The opening of the abdominal cavity is always preceded by curettage at the same sitting.

(5) The abdominal cavity is always opened first through the posterior vaginal fornix. If serious constitutional symptoms are present at the time of operation, and if pus can be evacuated from the pouch of Douglass, or from pus sacs within reach of the finger, and if bi-manual examination discloses no other collection which might be responsible for such constitutional symptoms, the pelvis is thoroughly cleansed, powdered with iodoform, and

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the pus sacs lightly packed with gauze. In a week the patient's condition has usually so much improved as to allow of abdominal section in the middle line and the removal of the pus sacs.

(6) If, on the other hand, there are no constitutional symptoms, or these are but slight, or if again it be impossible to reach and evacuate the pus from the presence of which constitutional symptoms have arisen, then the final step of medium abdominal section is undertaken at the same sitting, and the pus sacs completely removed.

(7) If the uterus appears to be involved in the septic process it also is removed.

(8) The same rule applies to the appendix.

(9) Flushing is not practised unless there is general purulent peritonitis.

(10) The pelvis is carefully cleansed by gauze swabs, and lightly powdered with iodoform.

(11) Every case is drained by a *single* strip of gauze, 2 inches wide, carried from the pelvis into the vagina.

This technique is the outcome of a large experience and much anxious thought; it gives good results, and I have therefore thought myself warranted in again bringing it under your notice.

SOME POINTS IN THE CAUSATION OF PELVIC SUPPURATION.

By G. Rothwell Adam, M.D., C.M.

Mr. President—In bringing before this Section so important a subject as Pelvic Suppuration, I must crave premission at the outset to extend my title so as to include all forms of pelvic inflammation; for I need not remind an audience such as this that it depends on the dosage and virulence of an infection, together with the resistance of the patient, whether an inflammation will pass through all its phases to suppuration. Nevertheless, this trinity of problems is presented to each one of us when face to face with a pelvic inflammation, and on a correct solution depends the forecast of the progress of the case. These remarks have been suggested, in some measure, by the careless use of the term "pyosalpinx," as signifying a pelvic suppuration, and not infrequently this loose diction is used to cover all forms of tubal inflammation. But, while it may be freely admitted that tubal inflammation is the most frequent cause of pelvic suppuration, there are other situations in which pus may be found in the pelvis.

Abscess of the broad ligament is not so uncommon; and, under certain conditions, to be discussed more fully hereafter, I believe purulent fluid will collect in the pelvic basin apart from a primary tubal origin. An unusual situation for pus to collect is in the uterine wall. Such an instance occurred in a lady under my care, who, when pregnant, contracted gonorrhœa. The labor was normal, and her medical attendant stated that the puerperium presented nothing unusual. Within six or seven weeks after labor she began to complain of pelvic pain, and had a rise of temperature: the uterus was found retro-deviated, and fixed by an extensive exudate. Under rest and the usual treatment the acute symptoms subsided: the abdomen was opened, with the intention of removing the infected adnexa and restoring the uterus to its normal position. In taking a V-shaped piece out of one uterine cornu, in order to thoroughly remove the affected tube, an abscess cavity was opened, and about three drams of pus evacuated. This cavity did not communicate with the fallopian tube in any way, nor was there any connection with the

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uterine cavity. The pus from the uterine abscess was sterile; at all events no cultivations could be obtained from it. An extended experience in operative work for purulent salpingitis has led me to note how frequently the isthmic portion of the tube is free from disease, while the ampulla may be distended with several drams of pus. This is more surprising when we consider that the infection in most cases, if not in all, has travelled by direct extension from the endometrium, through the tube, to its abdominal ostium. More than this, in many instances the remains of an infected endometritis still in existence can not infrequently be demonstrated; so that we have the two extremities of the tract bearing evidence of disease, while the middle portion, although necessarily traversed by the infection, is apparently free. It may be that the anatomical relation of the isthmus of the tube to the broad ligament, with its enormous lymphatic and vascular supply, is in a better position for recovery than either the uterus or the ampullary end of the tube. When these conditions exist, a further argument is advanced for the conservative treatment of suppurative salpingitis.

It is now universally recognised that the causes of pelvic suppuration fall under three heads, and the order in which they occur, as regards frequency, is as follows:—1. Gonorrhœa; 2. Sepsis (parturition and abortion); 3. Tuberculosis.

Since Noggerath years ago drew attention to the ravages wrought by the gonoccocus when spread to the endometrium, increasing experience has amply confirmed his views, so that it may be admitted that pelvic suppuration is chiefly due to this variety of infection. Nevertheless, a not inconsiderable number of cases owe their origin to septic infection, either after full-time parturition or after abortion. A much smaller proportion have tuberculosis as a cause, and, owing to peculiarities in pathological and clinical features, form a class apart from the two preceding groups. The object of this paper is to draw attention to the part played by sepsis in bringing about pelvic inflammation, which not infrequently proceeds to the stage of suppuration.

At the outset it is important to recognise that once the genital area is infected, by either the gonoccocus or septic organisms, the clinical phenomena are, to all intents and purposes, identical: that is to say, when confronted by a case of pelvic inflammation the only information that will enable us to differentiate between the two groups of causes will be found in the antecedent history. At first sight this might be thought to be easily obtainable, especially when the infection manifests itself shortly after parturition; but the case just recorded will serve to point out the difficulties in arriving at a correct judgment. How much greater, therefore, must the difficulties of a correct diagnosis be increased when the patient presents herself months, or it may be years, after a confinement?

Again, it is within the experience of most of us who deal with this class of case that pelvic suppuration by no means invariably, or even frequently, follows severe puerperal sepsis. In fact it has become well recognised that it is in those instances when the infection is of a mild degree that subsequent pelvic mischief is to be dreaded. I have seen a woman brought to death's door by puerperal sepsis, and yet within eighteen months delivered of a fine healthy child; and, what is more important for the purpose under discussion, convalesced with a normal puerperium.

In order to make my meaning more explicit it will perhaps be better to describe a typical case, as it presents itself to the examiner.

A married woman, the mother of several children, gives the history that all her labors have been normal, and that the convalescences, with the exception of the last (now some months ago), were also satisfactory. During the last puerperium, although not confined to bed for more than the usual time, she had for a few days some elevation of temperature, and offensive lochia.

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She also says she has never been quite well or strong, as she puts it, since this last confinement. Latterly the pelvic pain, which hitherto only annoyed her at varying intervals, has become persistent. Menstruation, when it occurred (often during lactation), was profuse, unhealthy, and accompained by pain. Each recurring menstrual period is now marked by a steady decline in her general health. On pelvic examination, the indisputable signs of tubal inflammation can be discovered, but without any extensive exudate. Not infrequently the uterus will be retro-posed, and may deceive the unwary by its apparent mobility.

Thus far the case is common enough; but a further stage, in not a few instances, is reached. Under favorable circumstances the mild, slowly developing infection will wear itself out, and the inflammatory process come to an end, leaving the tube occluded at the abdominal ostium, and perhaps adherent—or at all events fixed—in close proximity to intestine. Under such circumstances it is conceivable that it only requires something to lower the resistance of the tissues, *e.g.*, influenza, exhausting fevers, &c., in order that the colon bacillus may pass from the intestine along the line of adhesion and re-infect the damaged tube. That this does take place, when the appendix vermiformis becomes attached to the fallopian tube under such conditions, is almost unanimously admitted. Mr. Bland Sutton recently reported a case where the appendix vermiformis opened into an occluded fallopian tube, and discharged pus into the ampulla. The essential point, in the majority of instances, being the injury done to the tube by a prior infection from the uterus.

In looking over the histories of a number of chronic salpingitis cases, it was noticeable how many dated their ill-health to an antecedent abortion. Nor was this altogether confined to those patients who, either from lack of opportunity or careless ignorance, failed to obtain medical assistance; and in not a few the information was vouchsafed that "the doctor 'curetted.'" In these it might be permissible to doubt if the cure was not at all events as bad as the disease, seeing that the tubes became affected all the same.

When it is recollected that the pathological events which accompany abortion—the hæmorrhage, tearing of fœtal envelopes, &c.—all invite sepsis, it is not to be wondered at that a mild infection ensues with its attendant sequelæ. And it is quite conceivable that a failure to maintain an aseptic technique in the treatment of an incomplete abortion will result in an infection of the upper generative tract, notwithstanding an apparently immediate good recovery. It may be asserted as a fairly general rule that the morbidity resulting from an abortion works its greatest mischief at a late date. Instances such as are here mentioned must be familiar to everyone in family practice who cares to follow up obstetric histories by a careful gynæcological inquiry. Of course it is not intended to imply that every abortion is necessarily followed by a salpingitis, but merely to point out how favorable the phenomena of abortion are to the inception of a mild sepsis. One might as well say that, in the pre-antiseptic midwifery days, every parturient was doomed to sepsis. Those of us who experienced those times will remember the frequent outbursts of "puerperal fever," and also its mortality.

While directing your attention to some of the less obvious causes of pelvic inflammation, it may be of some profit to see what lessons can be drawn from them.

To take the cases following on a mild sepsis occurring during the puerperium first. If it once be recognised that such serious lesions as salpingitis and, more or less, pelvic peritonitis do sometimes owe their origin to a mild degree of infection arising during the recovery from parturition, it is obvious that more importance will be attached to abnormalities of the puerperium, even though they be slight and temporary.

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If practitioners would undertake to mould maternal opinion in the direction of submitting to systematic pelvic examination, instituted after the period of normal involution had passed, a step forward would be made in fighting so insidious a foe. Even if only those cases in which slight irregularities occurred during the puerperium were subsequently examined, much future trouble would be averted. A careful aseptic curettage, with the establishment of good drainage from the uterine cavity, at this stage would undoubtedly be efficacious in preventing many a subsequent dangerous and mutilating operation.

The second lesson, if short, is emphatic. It applies to the emptying of the uterus in abortion at the very earliest opportunity. If the principles which govern modern gynæcological practice are correct, no sound argument can be adduced in favor of waiting on nature to complete the abortion. No doubt nature is quite capable of so simple a matter as evacuating the uterus, but, as our knowledge of modern pathology tells us, she calls to her aid certain agents whose actions she cannot always control.

The third lesson to be drawn from the consideration of these few points is that an apparently quiescent salpingitis may become re-infected through adhesions to intestine. The woman with such a pelvic condition is never safe while she retains her damaged tubes.

Reflection on these cases of pelvic inflammation brings the conviction that many are in a sense preventable, and, moreover, that we possess two lines of defence. The first entails greater care during parturition of aseptic technique and closer observation of the puerperium, whereby the incidence of a mild sepsis may be prevented. The second line may be regarded as establishing the custom of *post* puerperal examination, especially where any irregularity in temperature, pulse rate, and condition of the lochia has been noted.

By these means I believe we shall help to bring gynæcology into line with other departments of medical practice, where the prevention of gross and other irreparable disease is the ideal aim.

Brilliant as the achievements of gynæcological surgery have been during the past twenty years in saving life and restoring the invalid to health, it must not be forgotten that these results are attained by methods crude in themselves and destructive of physiological function. That this is recognised in some measure by a number of pelvic surgeons the various plastic procedures on damaged tubes and re-sections of ovaries bear witness.

These so-called conservative operations must, at best, be classified as experimental; and we have to confess that at present we lack any systematised scheme calculated to restore the physiological integrity of these important organs.

Is not the time approaching when it is incumbent on us to seek out the prime causes of the gross lesion, and thereby prevent destruction of physiological function?

THE EVOLUTION OF THE ABDOMINAL OPERATION AND INCISION.

By J. Foreman, M.R.C.S., Eng., L.R.C.P., Sydney.

The evolution that has taken place in the abdominal operation since the first meeting of the Medical Congress at Adelaide, in 1886, has been so great that I thought the different stages of it might present some points of interest and mark a time from which to gauge further developments. The period, being almost twenty years, I may divide it into four, for purposes of convenience.

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The first five years: Those who were then practising abdominal surgery will only too well remember the mortality was high, and patients who recovered had, as a rule, a desperate struggle—distended abdomen, temperature and pulse raised, and suppuration of the incision being too common in a large proportion of the cases.

Twenty years ago, during an operation the patient lay on her back, with all the drawbacks and inconvenience of that posture. Little attention was paid to the disinfection of the skin, which was merely washed with soap and water, and the incision was made in the linea alba. The ovarian pedicle was transfixed and tied with the stoutest silk, without any attempt being made to cover it with peritoneum. The pedicle, in cases of fibroid tumor, was still brought to the surface of the wound. The abdomen was closed by interrupted sutures of silkworm gut, leaving on the peritoneal surface a succession of spaces, which were a fertile source of adhesions. In the toilet of the peritoneum sea sponges were invariably used. The process of cleansing the sponges was crude and imperfect; but, fortunately for the patients, Keith's tubes were in general use.

I now come to the second quinquennium of the period under review. By this time our knowledge of the germ theory had become more complete and accurate, and consequently our means of combating the great evil of asepsis more thoroughly adapted towards that end. Sponges were more carefully cleansed, and, so far as it was possible, rendered aseptic; while large tampons, which could more readily be kept clean, were occasionally substituted for them. The raw surface of the pedicle was completely covered by sewing the peritoneum over it, but the method of transfixing the pedicle was still adhered to. The peritoneal cavity was more efficiently cleansed and more care was taken to arrest hæmorrhage, as it was now recognised that the presence of blood in the cavity of the abdomen offered the most fertile medium for the development of micro-organisms. In other respects the operation resembled that of the previous period; but, as a result of the changes I have mentioned, the consequent mortality was much less.

In the third quinquennium there was a marked improvement in everything connected with abdominal operations, for at this time asepsis became the watchword of our art, instead of the feebler antisepsis, the insufficiency of which was generally recognised. Now we found the real reason why cases were lost in the failure to attain complete asepsis, and the various discussions on this subject were of the highest value, as they led to constant attempts after perfection in our methods: a perfection which we have not yet attained, but which we must never cease to strive after. Moreover, the operation was greatly facilitated by the introduction of the Trendelenberg position, while the pedicle was secured by sutures instead of by transfixion and ligature. The abdomen was still more thoroughly cleansed and dried, and drainage was seldom used except occasionally P.V. In using the drain in this manner it is done by means of gauze through a rubber tube. Wick makes the best drain, then plain gauze: gauze, with any powder in it, can only interfere with the flow of fluid. I think cases have been lost through putting in gauze and leaving it alone for two or three days. When it gets saturated drainage ceases, unless there is a dry pad in contact with it; even then, when lymph forms round it, it simply acts like a cork backing up the fluid in the pelvis. My own practice is to ease the drain the next day, and only in exceptional cases does it remain in more than one to two days.

The incision was made through the belly of the rectus instead of through the linea alba, and the abdominal wall was secured in separate layers with catgut sutures. Suppuration sometimes occurred in the incision; but the mortality rate was still further reduced.

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I now come to the main object of this paper, namely, to show our present position and analyse the reasons for our great progress; for we can fairly call it great when we are able to say that no surgeon in an extensive practice should have a mortality, allowing for peculiarities in patients, of more than 1 per cent. in clean cases. We must take clean cases only as a standard, for in other cases patients are so often poisoned and so near death that the question of operation becomes very doubtful. Some surgeons will operate so as to give patients a chance. Naturally many of them die; but some are saved, and, of course, cases of this kind cannot be brought under the general rule. Moreover, it is from clean cases that we learn whether there are any flaws in our mode of operating: that is to say, whether asepsis has been properly carried out, for it must never be forgotten that asepsis is by far the most important element of success in abdominal surgery.

The lessening in mortality is due to several causes, the first of which is greater strictness in aseptic methods and attention to minutiæ. Next to this I should place greater knowledge and skill in operating; then the Trendelenberg position; after that, the preparation of the patient; and, last, the closure of the abdominal wound and after care of the patient. We will consider these *seriatim*.

First, *Asepsis*.—Nowadays everyone understands the principles of asepsis, and so I shall say nothing about them; but I would like to point out that the translation of these principles into practice is not always a matter of course. You will see a man scrub his hands very thoroughly, using a nail brush with the greatest zeal, and straightway dry his hands with a towel without rinsing them either in clean water or under a tap. Manifestly in this process there is no security that the operator's hands have become germ-free. You will see another, after cleansing his hands *secundum artem* with soap and water, condy, and oxalic acid, quite unconsciously scratch his head, pull his moustache, or even put his hands in his pockets. Here, again, we must recognise a sad failure in translating aseptic principles into practice. The preparation of the skin is now very well carried out, but still is not as perfect as we hope to make it All persons present at the operation now wear white washing garments, which can be readily sterilised; and even the caps, which some have scoffed at, are valuable in their way, if only as indicating a striving after that perfection of method which it should be our constant endeavor to attain. The use of silk for ligatures has been abandoned, and in its place we use catgut, which can be rendered absolutely aseptic by one of several well-known methods. The chemical solutions employed to further the object of asepsis are very numerous, and each surgeon uses the one he prefers. Each has its particular value, and I need not dwell on them.

Second—The greater our practice in any art or occupation the greater will be the skill which we attain, and in the art of surgery that enhanced skill will be attended by a corresponding increased safety to the patient. Twenty years ago, even in the hands of the best operators, it was by no means uncommon to see the abdomen closed up without removing a growth on account of the supposed impossibility of completing the operation. Nowadays it may be said this never happens to a skilful operator. Formerly it was unavoidable, for the difficulties were immense and the knowledge but limited, and in this respect we can certainly claim great increase of skill in operating.

It would be difficult to over-estimate the advantages of the Trendelenberg position, which is now universally adopted. By it we are able to see the whole of the pelvic basin, and so to bring into close apposition the cut or torn margins of serous surfaces, and to remove blood clot, which, as I have already stated, is recognised as the most fertile source of septic infection;

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while, from the greater facilities it affords the operator, it has materially shortened the duration of the operation. It must be admitted that it is not applicable in every case, for where the pelvis contains free fluid, whether serous, sanguineous, or purulent, it would be manifestly unwise to adopt a position in which the fluid might find its way into the upper part of the abdomen. In such cases as these the supine position is used, as formerly.

The principles which underlie the preparation of the patient are now fairly well understood. Rest in bed, thorough unloading of the bowels, light diet, free action of the skin, and thorough cleansing of the skin of the abdomen by some approved chemical solution, are some of the principal measures on which we depend.

I now proceed to consider the modern operation, but I must again insist upon the extreme importance of absolute asepsis and careful cleansing of the cavity, for it is chiefly to these two factors that we owe our freedom from those distressing cases of abdominal distension which formerly were so common. The evolution of the abdominal incision has been to me an object of great interest. Formerly, following the example of Spencer Wells, it was the universal custom to make the incision in the linea alba; and this, especially with the system of drainage which was then in force, was a frequent source of hernia. Nowadays we never think of cutting in the linea alba: we open the sheath of the rectus, turn aside the belly, and open the peritoneum beneath it. This is a great improvement on the method of some years ago, which consisted in cutting through the rectus muscle, for, as will be at once seen, by the present method we avoid that interference with the nervous and vascular supply of the muscle which must necessarily result from an incision through its belly. The advantage of the new incision, which came into use about three years ago, will be seen when we consider the method of closing the abdomen. The cut edges of the peritoneum are brought into careful apposition by a continuous suture with chromic gut, and the cut edges of the rectal aponeurosis are similarly joined with the same material. The skin incision is closed with the continuous suture of white gut. It will be seen at once that an incision so closed up leaves practically no risk of hernia, while the risk of adhesions is very much reduced.

The treatment of the pedicle has been greatly improved. Instead of transfixing and ligaturing it with silk it is now compressed with the angiotribe—an instrument which I now show you—with the result that the parts compressed are quite bloodless when it is removed. The margins of the serous membranes are left in accurate apposition, while the broad ligament remains free from any traction upon it. Through the compressed part a white catgut continuous suture is run so as effectually to prevent any risk of secondary hæmorrhage. I need hardly say that gentleness of manipulation is important, so as to avoid as much as possible the bruising of the abdominal organs. Gauze pads are used instead of sea sponges.

The After Treatment.—Since Lawson Tait taught us that the proper way to treat peritonitis is not to lock up the bowels, but to get them to act freely, our principal care in cases of abdominal operation has been to ensure a brisk action of the bowels within, at most, twenty-four hours. If the bowels do not act the day following the operation care is taken to bring about this result. This may be said to be the main point in after treatment in abdominal cases, and the system of free purging has saved many lives and greatly increased the patient's comfort. A little consideration will show that, for the comfort of the patient, it is very important that the bandaging of the abdomen should not be too tight. Even in perfect health a very tight bandage round the abdomen is extremely uncomfortable, and for a woman who has

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just undergone a grave operation tight compression of the abdomen is very painful, and tends to restrict the action of the bowels. We must allow the patient to rest in the position most comfortable to her.

The record of abdominal surgery during the last twenty years has been one of almost uninterrupted improvement and success; but there is one point, I mean the prevention of adhesions, in respect to which our results are by no means so satisfactory as we could wish them to be. From time to time cases present themselves in which the presence of adhesions goes a long way to neutralise the success of a well thought-out and skilfully performed operation, and it is very distressing after a patient has been relieved of a dreadful disease to find her life made a burden by apparently unimportant adhesions. To prevent their occurrence is one of the principal desiderata of our art, and a conscientious surgeon must keep it constantly before him as an aim of the highest importance. Personally, I have tried aristol in large and small quantities without the success I expected, for on two occasions I have had to re-open the abdomen for adhesions and found the worst ones about the site of the powder. I have also left the abdominal cavity full of saline fluid, but, like many others, cannot trust much to it. At present I am trying the method of washing the pelvis with a sea sponge and saline, just as we wash the abdominal wound, and it is surprising what a different appearance the serous surface presents after it is done. It is too early yet to say if I shall be content with this, but in my opinion the problem to be worked out is this prevention of adhesions, and the one who is fortunate enough to give a fairly safe method will merit the gratitude not only of the patient but of the surgeon also.

CHORION EPITHELIOMA.

By F. C. Batchelor, M.D., M.R.C.S., Eng., Dunedin.

It is not my intention to read a paper dealing with the subject of Chorion Epithelioma: it has been so ably and fully discussed in recent literature that it would be absurd for me to attempt to generalise from one special case. So far as I know, very few cases have been recognised in Australia, and the one I propose to report to-day is the first that has been published from New Zealand.

M.C., aged 45, married twenty-three years; six children, the last six years ago. Two miscarriages, one twelve years ago at the third month, the last six months ago, about six weeks pregnant.

Admitted into the Dunedin Hospital on December 30th, 1904.

Patient weak and anæmic, has lost much flesh, complains of loss of appetite and thirst, with feverish attacks, stomach irritable, and occasional bouts of vomiting; some cough, with occasional slight hæmoptysis; almost constant metrorrhagia.

History.—Dates her illness from miscarriage six months ago; has never been well since; was attended by her doctor, who removed a mass from the womb, under an anæsthetic, and considered her six weeks pregnant. There was much hæmorrhage at the time.

Following the abortion had almost constant slight bloody discharge. At the end of a month a flooding came on; she was curetted, and took medicine; the flooding abated, but a blood-stained discharge continued from the vagina. She continued to get weaker, lost her appetite, complained of headache, dyspnœa, and general malaise, and occasionally spat up small quantities

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of dark blood; she continued under medical treatment, but did not improve; there was persistent menorrhagia, with occasional floodings. A few days before admission to the hospital there was a heavy hæmorrhage. Pulse 104, temperature 103°.8, with remissions, a rigor preceding the rise in temperature.

On examination, at the lower part of the vagina two irregular excavating ulcerations were noticed, one on the lower lateral wall, and one on the anterior wall, just above the meatus; the ulcerations were about half an inch in length and a quarter of an inch broad; they extended through the mucous membrane, the margins were dark-bluish in color, somewhat resembling a tubercular ulcer. On the posterior vaginal wall, lying just within the caruncle, was a small dark-blue sub-mucous tumor, about the size of a bean; the cervix was thickened and irregular, blood and fœtid discharge was exuding from the os, the uterus was retro-flexed; when drawn forward it was found to be enlarged, soft, and flabby, with a nodular thickening about the right cornu posteriorly.

The nodule from the vaginal wall was excised; it was entirely beneath the mucous membrane, and enucleated readily. It looked like a blood clot; there was no active bleeding from the capsule; it was set aside for microscopic examination. The uterus was dilated. On curetting soft friable tissue came away in large quantities, accompanied by severe hæmorrhage; it was evident that it would be dangerous to continue curetting, as the uterine wall would have been readily perforated. The uterine cavity was freely douched with hot formalin solution, and plugged with iodoform gauze; further measures to be determined by examination of the vaginal growth. Chorion epithelioma was suspected.

The tumor from the vagina was found to be composed chiefly of blood-clot, with decidual-like groups of cells dispersed freely through its substance. [Section shown.]

Diagnosis.—Chorion epithelioma plus sepsis. Vaginal hysterectomy decided on.

January 7th, 1905.—Uterine cavity washed out and filled with iodoform gauze, external os closed by strong silk sutures, and uterus, ovaries, and tubes removed by the vagina. Patient was in an extremely bad condition, but stood the operation well, and for some days there was a decided improvement. On the 13th a rise of temperature occurred, with a return of septic symptoms.

On February 8th a large abscess under the deep fascia of the right side was opened, and a quantity of fœtid pus evacuated. A portion of the sac wall was excised for microscopic examination and pronounced to be granulation tissue. The septic symptoms, however, still persisted; for a day or two she would keep comparatively well, then a rigor and rise of temperature would occur.

About the middle of March, for some ten days, she improved remarkably; so much so that she was able to be removed to the sun-room for some hours daily, and had determined to return home.

On the 26th of March, however, the rigors and rise of temperature recurred, and from thence she gradually went to the bad.

A fortnight before her death it was noticed that there was paresis of the left arm and leg; this gradually deepened, and for some days before her death she was hemiplegic and comatose. She died on April 18th.

The two ulcerations in the vaginal wall healed spontaneously, and the incision for removal of vaginal growth closed by primary union; the abscess in the right thigh had also healed before her death.

No thorough *post mortem* could be obtained; but the abdomen was opened and examined before removal of the body by the friends. The pelvic

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floor had united perfectly, and there was no growth in the pelvis. During her illness it had been noted that the liver was enlarged and tender; a portion was torn away; it had a nutmeg appearance, and, in the portion removed, two small abscesses, containing about half a drachm of thick pus, were found.

There were also a number of small, stellate, fibrous-like patches in the liver substance removed. Examined microscopically these show broken-down liver cells and fibrous tissue, which contain small deeply-stained brown cells, with marked nuclei. [Section shown.]

The spleen was soft and diffluent, and there was a small abscess in the cortical portion of the left kidney.

The uterus [specimen shown] has a large fungating mass sprouting into the cavity and excavating deeply into the muscular wall of the uterus on the right posterior wall, near the fundus; at a portion just below the level of the right tube there was little but the peritoneal coat of the uterus left; bluish masses of the growth could be seen extending into the uterine wall, off-shoots of the main growth. Microscopically the characteristic cubical and syncthythial cells are seen invading the muscular walls of the uterus.

I was unable to obtain clear data from the gentleman who attended her as to the character of the miscarriage. He was, however, impressed by the fact that the uterus was far too much enlarged for the assigned date of pregnancy, and he describes the cavity of the uterus as being filled with quantities of blood-clot and pieces of after-birth.

One point of interest in this case seems to be the apparent latency of this growth: two secondary nodules in the vagina broke down and healed spontaneously; the third, removed by me, was evidently undergoing a similar process. The absence of bleeding from the cavity, and the microscopic appearance of the growth, showed it to be undergoing necrotic changes.

In the absence of an efficient *post mortem* (which was refused) it is impossible to say whether the hæmorrhage from the lungs and the cerebral symptoms were the result of a secondary growth or a septic fœcus; but, from the marked septic symptoms and such *post-mortem* findings as we were able to obtain, I am inclined to attribute her symptoms to sepsis.

After the removal of the uterus it would have been quite impossible to have diagnosed her condition from a severe and prolonged form of septic infection. One might, *prima facie*, expect this form of growth to assume varying degrees of malignancy, and it is possible that toxins resulting from the septic infection may have neutralised or arrested the growth of the secondary tumors, much in the same way as Coley's fluid sometimes influences a sarcomatous growth.

In consequence of recent theories as to the frequency of an excess of lutean tissue being found in the ovaries in cases of chorion epithelioma, I prepared a large number of sections from both ovaries. To one who does not claim any expert histological knowledge on this subject it seems an extremely difficult matter to decide what constitutes an excess of lutean tissue; and, furthermore, when we are taught that this lutean tissue is undergoing a process whereby the connective tissues proper of the ovary are re-formed, it is evident that there must be stages in this process where it is impossible to distinguish the one from the other. I should be glad to have the opinion of any who have studied this matter on the specimens which I shall show. Of one point, however, I am convinced: it is this—that lutean tissue exists in all ovaries in very considerable quantities during the active periods of sexual life, and in specimens that I shall show, where the ovaries have been removed in cases of early ectopic gestation, the lutean tissue is very greatly in excess of anything that is met with in this special case which I have described.

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PUERPERAL SEPSIS.

By H. Osburn Cowan, M.B., C.M., Glas., of Kew, Victoria.

I have chosen the title, Puerperal Sepsis, in preference to the older term, Puerperal Fever, as removing my subject at once out of the realm of epidemicity and placing it in that of dirt. It thus loses much of its mystery, but at the same time, as I shall hope to show, much of its menace.

CAUSATION.

Puerperal sepsis, like sepsis elsewhere, is due to breach of surface infection by pathogenic organisms. The organisms are various, comprising streptococci, staphylococci, the bacillus coli communis, and others; the sites and circumstances of their entrance vary, and so we find much variety in the clinical expression of microbial invasion of the parturient canal, either during labor or during the puerperium.

Under normal circumstances, the uterine interior in pregnancy and labor is sterile, and even under abnormal circumstances of dirt and disease, the experience of lying-in charities shows that the danger of puerperal infection can be reduced to the vanishing point. For instance, in the out-door practice of the British Lying-in Hospital, for the years 1880-1902, the proportion of deaths from septic causes was only one in 10,000 deliveries. As this covers the cases due to auto-infection, as well as those due to infection from without—and that, too, in the class of patient in which the former might be regarded as most likely to occur—it is evident that, as a cause of puerperal septic disease, auto-genesis may be regarded as a negligible quantity.

The most recent work on the bacteriology of the genital canal confirms this statement. Whitridge Williams says—"Bacteria, likely to be the cause of puerperal infection, are not to be found, except rarely, in the upper part of the vagina"; and Foullerton and Bonney, who have given the fullest consideration to this aspect of the question, say—"The puerperal fever which is likely to follow auto-infection will usually be of a comparatively mild type." Puerperal sepsis, therefore, for all practical purposes, must be regarded as not only due to infection, but to infection conveyed from without. It may be carried from case to case; but of hardly less importance is the fact that pathogenic organisms, which are found in myriads at and around the vaginal orifice, may be carried in on the examining finger, instrument, or douche nozzle. Nature has, in various ways, safeguarded the process of parturition against the unassisted entrance of infective organisms. She had, however, laid her plans long before the introduction of the methods of meddlesome midwifery, and no provision has been made against the actual introduction of infection from without, or of dealing with it when it has occurred. Given, indeed, the presence of pathogenic organisms in the upper reaches of the parturient canal, and no culture medium can compare with the interior of the puerperal uterus. Once the invading army has reached the uterus, it finds supplies and lines of communication ready at hand. The possibilities of extension and absorption are so vast that anything approaching to certainty in dealing with an established infection is manifestly out of the question. On every count we are compelled to the conclusion that the best endeavors of the obstetrician must be concentrated upon the prevention of infection in the first instance. Here prevention is not only better than cure, but prevention is possible and cure is dangerously problematical.

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GENERAL CONSIDERATIONS AS TO PREVENTION.

It has been said that the medical man begins his struggle with infection on laying hold of his obstetric bag. But he must begin it long before this, recognising that successful warfare depends hardly less upon preparation than upon performance. Apart altogether from the added horror of puerperal infection, pregnancy and parturition in themselves involve no small risk and strain. Even under modern conditions there still remains a small proportion of women to whom pregnancy brings improved nutrition and general health; but for the large majority it involves more or less impairment of function—digestive, hæmapoietic, nervous, and mechanical. There results loss of individual resisting power and of tissue vitality. Deficient innervation, for example, and inefficient muscular contractility increase the liability to post-partum hæmorrhage, and the resulting diminution of systemic resistance to microbial invasion and the greatly enhanced activity of the absorbents seriously increase the liability to generalised infection. And over and above its special significance, albuminuria, with the cellular degradation to which it gives rise, has an important bearing in this direction. In short, from the date of his engagement to attend a confinement, everything that concerns the functional and organic health of his patient must concern the practitioner who desires to conduct her with the minimum of risk through her coming trial.

THE OBSTETRIC BAG.

It has always seemed to me that quite an unnecessary amount of abusive criticism has been directed against the obstetric bag. Various measures having been suggested for its reformation, and in vain, its abolition is now demanded. The so-called aseptic detachable lining is a delusion, and may be in a very real sense a snare. As it is impossible to keep such a lining aseptic, it cannot be looked upon as a defence, and after two or three boilings it becomes utterly lost to all sense of size and proportion. The *bag* should certainly be clean; but it is the *man* who must be aseptic. The principle which so much insistence upon the character of the bag is apt to make us lose sight of is that the only safety in obstetric work lies in personal and instrumental sterilisation for each individual case. The obstetric bag should be kept strictly for primary obstetric work, and it should be much larger than that in common use, in order to carry in washable coverings an apron and the necessary instruments and appliances.

PREPARATION OF PATIENT AND PHYSICIAN.

After reaching the sick room, and noting the general condition of his patient, the medical attendant's first duty is the careful sterilisation of his hands. I need not go into details as to this, but need only say that it should be as conscientious and thorough as for any surgical undertaking. If, owing to previous septic contacts, he is in doubt of its completeness, sterilised rubber gloves should be worn. Its use is so desirable, on the grounds of decency and defence, that no careful medical man, who looks at the question from the modern point of view, would nowadays proceed to the conduct of labor without the protection of a sterilised apron. The nurse will, in the meantime, have been engaged in the careful preparation of the skin at and around the vaginal orifice, and the combined abdominal and vaginal examination of the uterus may now be undertaken.

EXAMINATION OF PATIENT.

No part of a paper which I read at Hobart was so adversely criticised as that in which I urged the necessity for retaining the vaginal method of examination. The position which I then took up I still maintain. In a

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large proportion of multiparous cases it may be entirely dispensed with, and in primiparous cases where the descent of the head is already well advanced. But if we keep before us as our ideal not to make more than one vaginal examination, and not to make that if we can avoid it, we can so safeguard the procedure as not only to increase but actually to diminish the risk to mother and child. Much as we may learn from abdominal palpation as to the position of the child, and even as to the progress of labor, there remain points upon which vaginal examination only can inform us, such as the condition of the cervix, the descent of the anterior lip, which not infrequently prolongs labor and renders it more painful, and the exact bearing of coccygeal abnormalities. For the early recognition, too, of prolapse of the cord, upon which the life of the child may turn, we are entirely dependent on vaginal examination.

I am interested to note that, in speaking of abdominal palpation in a recent article, Berry Hart says—"This method is not so suitable for private practice, but there the reduction of internal examination to a minimum, with the use of gloves, should give results as good, and probably *more* sure."

Needless to say, the casual "trying of the pains" by the nurse must be absolutely prohibited.

Vaginal examination must only be performed after thorough preparation of the physician's hands, and the careful separation of the labia minora, in order that the examining finger may not carry infective organisms from the external genitalia to the upper part of the vagina.

ASSISTED DELIVERY.

We have, so far, dealt only with the preliminaries to the conduct of a case of normal labor. The importance of their consideration lies in the fact that, in such a case, they represent the only risk of infection up to the end of the second stage. The same principles will guide us when we have to assist delivery. In any attempt to deal fully with the thesis of puerperal sepsis, something more than a passing reference to the use of the forceps is necessary. They may increase the risk of infection by the direct introduction of organisms, the production of trauma, and the consequent opening of fresh channels for infection, and the diminution of tissue resistance. The nicest discrimination is necessary in their use, so that it be not too frequent, too early, nor yet too long deferred. With a view to the safety of both maternal and fœtal structures, they must be accurately applied, and used to so assist the expulsive pains in their incidence and in their direction as to obtain the maximum of result with the minimum of force and of injury. It is my own practice to remove the blades when the head is well down upon the perinæum, with a view to minimising the risk of laceration.

Important as trauma is from the point of view of mortality, it is even more so from the point of view of morbidity. Broadly, I think it may be said that the morbidity of the puerperium is more largely determined by the traumatism produced during the second stage of labor, and its mortality by the complications of the third stage.

THE THIRD STAGE.

The management of the third stage of labor is the key to the control of post-partum hæmorrhage, which has been already referred to as seriously enhancing the risk of septic infection; but it is in retained secundines that the greatest danger of infection lies.

Nature is deliberate with design in the separation of the placenta, and so far as my experience goes, seldom makes an attempt at actual expulsion within half an hour. It is not until after this period has elapsed that there

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is any justification in ordinary cases for hastening the delivery of the placenta. Cases, however, occur from time to time in which, owing to over-fatigue of the uterine muscle, free hæmorrhage is observable while the placenta is still *in utero*. When confronted with such inertia, great judgment is required to determine whether the danger of delay is greater than that of delivery. It is sometimes impossible to maintain uterine contraction sufficient to control bleeding, and I have not hesitated in such cases to empty the uterus after fifteen or twenty minutes, and give ergotinin and strychnine hypodermically and the liq. ergot. by the mouth, the latter in the hope of continuing the more immediate contraction induced by the former.

Both placenta and membranes should be carefully examined as a matter of routine, with a view to ascertaining, not only whether they are complete, but also whether separation has occurred in the proper plane. If not satisfied as to the integrity of the secundines, my own practice is as follows :—

1. If the placenta is complete, and only a small portion of the membranes is wanting, to leave it.

2. If a considerable strip is wanting, to go as far as the cervix in search of it. If that fails to discover it, again to leave it, and to instruct the nurse to watch carefully for its presentation.

3. If a portion of placenta is wanting, to pass my hand into the uterus. The necessity for this, however, very rarely occurs. It is much easier to remove manually the whole than a small portion of the placenta, and the accoucheur should not persist in attempts to forcibly express a placenta which is presumably adherent in whole or in part. I find, however, that greater patience in the third stage has markedly reduced the number of cases in which either procedure has been required.

Nothing gives one such a sense of security at the termination of labor as the integrity of the placenta and membranes.

THE REPAIR OF LACERATIONS, ETC.

I need not dwell upon the folly of pursuing the policy of the open door with regard to lacerations of the perinæum. If extensive, they should be repaired in the dorsal position, and deep and accurate apposition obtained. I am at a loss to explain nature's apparent indifference to the integrity of the perinæum, even in normal cases, seeing that it constitutes such an important safeguard against puerperal infection.

If for any reason there has been recourse to forceps extraction before the full dilation of the cervix, it is advisable to explore the upper reaches of the vagina and the cervix, as rupture into the posterior fornix is known to occur in instrumental delivery which has left the perinæum intact. In normal cases, however, and even in the large majority of difficult cases, *post-partum* interference is to be strongly deprecated. For this reason routine *post-partum* douching has been abandoned. In my opinion, vaginal douching *post partum* is worse than useless, excepting as a preliminary to intra-uterine irrigation, which will only be called for when there has been manual interference with the uterus itself. For such procedure only a fountain douche should be used, because of the one-way flow, the domestic Higginson, with its two-way suction, being, to my mind, a most dangerous appliance.

CLINICAL CONSIDERATIONS.

We have dealt, so far, only with preventive measures. If, however, infection should occur, how are we to recognise it, and how shall we treat it ?

It is not necessary to dwell at any length on the clinical picture of puerperal sepsis. It is ever present to the mind of the obstetrician, and is as imperative as ever was the writing upon the wall. It should determine and

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control every detail of the conduct even of a normal confinement. No man who has struggled with it for, perhaps, week after week, with despair for his "familiar" night and day, can deem any precaution too exacting which promises immunity from its recurrence.

I wish it were possible for me to define wherein lies the difference between the rise of temperature on the third to the fifth day of the puerperium, which ushers in puerperal septicæmia, and that which, though suggesting ugly possibilities, clears up entirely within twenty-four hours. When it is ushered in by a distinct rigor and a rise to 104° or 105°, the differentiation is not difficult; but it is not always so accompanied, nor does it always rise so high. The febrile reaction after septic infection is often insidious, and may suggest nothing more serious than mammary or intestinal disturbance. There are cases, too, in which a free *post-partum* hæmorrhage or some individual peculiarity robs the pulse rate of much of its significance. In my own experience an acceleration of the pulse, disproportionate to the rise of temperature—as, for instance, a pulse of 100° with a temperature of 99°—has frequently been traced to mammary disturbance, and has cleared up on the satisfactory establishment of lactation. On the other hand, nothing has been so effective in eliminating intestinal disturbance as a cause of high temperature in the puerperium as a dose of calomel. I have known the temperature fall after purgation from 103°.5 to 99°, with the clearing up of accompanying symptoms. If, however, after the elimination of such disturbing factors, the febrile temperature is maintained, and especially if it show a definite evening rise, the presence of infection may be inferred.

It has been usual to distinguish at this point between sapræmia, or septic intoxication, and septicæmia, or septic infection. It is now generally admitted that this is a distinction of degree and of duration only. The attack of pathogenic organisms upon dead tissue producing toxæmia may, at any moment, develop into a general invasion, producing septicæmia. It is more correct, therefore, and it is certainly safer clinically, to regard the so-called sapræmia as a stage on the road towards a general septic infection, and to treat the case from the outset as having all the inherent possibilities of the latter.

Fœtor of the lochia is not regarded as evidence of puerperal sepsis. In some of the most virulent cases of streptococcic infection it may be entirely absent. It is evidence of decomposition of dead tissue, but tells us nothing as to systemic invasion; in fact, given other evidence of general infection, I should regard marked fœtor of the lochia as a favorable symptom. It suggests local, rather than general, bacterial activity, and it promises a satisfactory response to energetic local treatment. Where it is possible, a bacteriological examination should be made of the uterine contents, and, if performed early, this may give invaluable indications for treatment. In the later stages of puerperal sepsis mixed infections so confuse the issue that the indications for treatment are much less definite.

TREATMENT.

Given the presence of infective organisms in the parturient canal during the puerperium, and everything favors their multiplication and distribution. This is no time for hiding one's head in the shifting sands of conjecture. Above all other explanations, let us banish the ever-handy influenza from our consideration of the earlier, and typhoid from that of the later, stages of puerperal sepsis. Every hour is of importance. Acute immediate general infection, presumably streptococcal, is very rare, and the development of puerperal sepsis may frequently be inferred before a general microbial distribution has occurred. Such inference should spur us to immediate action. If we have

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been quite sure of the integrity of the membranes and placenta, it will be sufficient, in the first instance, to douche the vagina, and, subsequently, the interior of the uterus, and to deal with such breaches of surface as our examination reveals. This will remove the uterine *debris*, destroy organisms which have not penetrated the decidua, and, according to Pryor, if performed within twelve hours of the onset of the first evidences of infection, will effect a cure. If it does not, and especially if there has been any doubt about the placenta, my own practice has been to explore the interior of the uterus under an anæsthetic, and to remove adherent tissue and *debris* with a blunt flushing curette; thereafter to swab out the uterine interior with an antiseptic, strongly germicidal, but not escharotic; to irrigate with a normal saline or very dilute antiseptic solution; and then pack the cavity with 10 per cent. iodoform gauze. In the limited field of private practice I have not found it necessary to employ the more radical procedure of the *cul-de-sac* operation so strongly recommended by Pryor.

Such is my attitude towards the much-debated question of primary curettage in cases of puerperal sepsis. Is has given me most satisfactory results, and, carried out carefully, with a rigid regard for asepsis, has proved itself a sound and safe procedure. In cases of direct virulent streptococcic infection, in which there is no question of retained secundines, it is not indicated; but there are cases the true nature of which can only be determined by bacteriological investigation, and in which, more than in any others, good results may be expected from anti-streptococcic serum.

BACTERIOLOGY AND SERUM-THERAPY.

Splendid work is being done with a view to determining the precise bacteriology of puerperal sepsis. It has already established the preponderating importance of the streptococci in the causation of severe puerperal infections. Much, however, still remains to be done, for, as Dr. Foullerton points out, the group probably includes several species, differing in pathogenic action and in the character of their respective toxins, and requiring for their appropriate treatment different specific sera. Until these have been distinguished, the use of such a polyvalent serum as that offered by Burroughs, Wellcome, & Co., represents, perhaps, the best we can do under the circumstances. But just as the bacteriological examination of the lochia must be early to be unequivocal, so the serum-therapy of streptococcic infection must be early to be effectual. There is reason to think that the serum is rather germicidal than antitoxic in its action, and, if so, it is unreasonable to expect much result when the organisms have invaded a large cavity such as that of the peritoneum.

THE EXAMINATION OF THE BLOOD

has, so far, not given us any further indications for the treatment of puerperal septic conditions. It is hardly necessary to say that the demonstration of streptococci in the blood stream indicates definitely the presence of a streptococcic septicæmia; but the leucocyte count has not led to findings of definite diagnostic or therapeutic value. The normal variations during pregnancy and the puerperium are so great that the determination of morbid variation is well-nigh impossible. Besides which, it is not the mere presence of pus which determines leucocytosis, but the passage of bacterial toxins into the circulation; and this passage ceases to take place when free drainage has been provided: consequently, it is likely to be more marked in an acute localised mammary inflammation than in a generalised puerperal infection.

For the same reason, too, when the pus in a pyosalpinx has been definitely walled off, the leucocyte count does not indicate its presence.

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To the clinical obstetrician the importance of the demonstration of the part played by streptococci in virulent puerperal septic conditions is as follows:—

(1) They constitute the most clearly and definitely preventable of all puerperal infections, being always introduced from without.

(2) Their presence may be definitely recognised at an early stage in the process.

(3) There is reason to believe that, at an early stage, they are most amenable to treatment by an appropriate serum.

The principle to bear in mind in dealing with all puerperal septic conditions is, be previous if you can, and if you cannot be previous, be early.

PREVENTABILITY.

I do not propose to go into the statistical aspect of this question, which has been so often dealt with before. The experience of the in-door and out-door departments of the great lying-in charities demonstrates the preventability of puerperal sepsis, and it remains to bring the statistics of private practice into line with these results, for up to the present they have shown no improvement. In the tables of the Registrar-General's returns there is no cause of death so open to attack as that which is covered by the term puerperal septic diseases. Much is said from time to time of the possibility of eliminating tuberculosis as a cause of mortality within three or four generations; but here is a cause which, by combined effort, could be removed within one generation. Much blame is laid at the door of the untrained midwife, and this difficulty is being largely done away with by the Midwives Act of 1903. Some such Act should be placed upon the statute books of all civilised communities. In the meantime, let each medical man in his own sphere demonstrate the fact that puerperal sepsis is preventable by trained intelligence, and an enlightened public opinion will see to it that such training is made available for those who cannot afford to pay for themselves.

If the infirm, the insane, and even the inebriate are made a charge upon the State, surely the mothers, upon whom its very continuance depends, should be not less the object of its care.

From a racial point of view, surgery must often regard its triumphs with doubtful satisfaction; but the healthy mother means so much to the future of the child, and the healthy child so much to the future of the race, that the safeguarding of parturition admits of no such mental reservation.

THE TOXÆMIA OF PREGNANCY.

By F. C. Batchelor, M.D., M.R.C.S., Eng., Dunedin.

My reasons for bringing this subject under your notice are these:—In the first place, the condition to which it is my intention to refer is of great practical import, and is at the present time attracting considerable attention from those interested in our special branch.

So far very little is definitely known on the subject; what we do know, however, promises to considerably modify our views and methods of treat-

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ment of what I take to be a very common complication of pregnancy. Further advance in our knowledge can be best attained by drawing the attention of the profession generally to the condition and to the trend of modern thought concerning it. Further, I am hopeful that a discussion will ensue that may be helpful in eliciting the experiences of others, who must have frequently had to deal with similar conditions.

For clinical purposes the toxæmia of pregnancy may be best ranged under two main types :—

1st. The toxæmia accompanied by that appalling complication, eclampsia. Most authorities seem now to regard this condition as the result of toxic material circulating in the blood: that the condition of the kidneys, often in the past regarded as the primary source of the trouble, was now relegated to secondary importance, and considered due to the irritation, congestion, or inflammation of these organs in consequence of the toxins in their blood supply. The nature of the toxic matter and its source was not clearly understood, the weight of evidence tending to show that it was a form of auto-intoxication resulting from imperfectly oxidised proteid material, which, under normal circumstances, is excreted by the kidneys in the form of urea; but, as the result of failure of oxygenation, poisonous materials or by-products of urea were produced which were highly poisonous, and that the organ chiefly responsible for this failure was the liver. With this form of toxæmia it is not my intention to deal. Many of you residing in the large centres must meet a greater number of cases than fall to the lot of one who lives in a comparatively small town, where such cases are, fortunately, not of frequent occurrence.

The second type of toxæmia to which this paper specially refers is unaccompanied, as a rule, by convulsive seizures, and is but little recognised by the profession generally. The severe cases are not of great frequency; yet, from an experience of over thirty years' professional work, I am confident they are by no means rare, nor to be viewed as medical curiosities. The symptoms may be marked and virulent, and may lead to a fatal issue, and are specially liable to be overlooked or confused with other conditions; nor is this at all surprising when we find the current works on midwifery, almost without exception, ignore the subject, or at most devote a few short paragraphs to the topic. When myself brought face to face with the disease I have altogether failed to find anywhere anything approaching a clear and definite description of it in any of the text-books. Further, although, as I have already stated, the severe cases of this type of toxæmia may not be very common, milder forms, it seems to me, are extremely frequent; many of the minor disturbances, so common in early pregnancy as to be almost looked upon as physiological, are often probably a result of this toxic state. Fortunately, in a large proportion of instances they pass off spontaneously or under treatment, but they are capable of more efficient and satisfactory management if the factor on which they depend is recognised.

Finally, although some of you may have heard or read little of this disease, I am confident during the next few years you will find it taking a prominent place in obstetric literature; once its significance is recognised, as is now being done, from its importance it must excite deep interest in all who undertake midwifery work, and is of great moment to the general practitioner under whose charge these cases invariably come.

The readiest method of bringing this subject before you will be to read you clinical reports of three cases which have come under my own observation during the past few months.

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CASE I. was seen by me in consultation with Dr. Gabites, of Timaru; he has kindly supplied me with a full report, from which the following *precis* was prepared :—

M.C., aged 21, married seven months, had always been healthy, strong, and fairly stout; no previous illness, except heat-exhaustion in Durban nine months before present illness; no history of malaria. Fell pregnant about five months before commencement of the present illness; early pregnancy, uneventful till the second month, when she complained of nervousness; got well under treatment.

August 3rd, 1904.—Went out in the snow in thin shoes; on returning home felt chilly and faint, and had bad pain in the stomach. There was a slight red discharge. Spent a restless night on account of the pain in the stomach.

Dr. Gabites first saw the patient on the morning of August 4th. Temperature, 102; pulse, 100; no cough or coryza; complained of pain in the back and over abdomen. Treated for influenza: gradually improved.

August 7th.—Felt quite well and took her food well; no feverishness and no pain or tenderness anywhere; ravenous, ate heavy meal. At 11 p.m. the same day Dr. Gabites was called urgently, and found the patient evidently suffering from intense pain; pain, referred to the right hypochondriac and the right lumbar regions, did not appear to shoot in any particular direction; there was extreme tenderness on pressure over these regions. Temperature, normal; pulse, 116.

August 8th.—The pain returned, but was bearable; pain referred to the right iliac region and down the right side. Morphia gave relief.

August 9th.—Pain and tenderness less; passed urine frequently, urine clear and high-colored; acid, specific gravity (1015), mucous deposit, no albumen. Temperature, 100; pulse, 90.

August 10th.—No pain. Temperature, 99; pulse, 85; seemed to be improving.

August 12th.—Had a rigor at 2 p.m. Temperature at 5 p.m., 101; pulse, 102. Flatulence troublesome; complained of nausea, no inclination for food. Patient's condition evidently worse. No pain, but extreme restlessness; felt as if her heart would stop.

August 13th.—Consultation with Dr. Drew. Symptoms of pain attributed to pressure of the pregnant uterus; restlessness, feverishness, coated tongue—probably due to a relapse of influenza. Treated for influenza.

August 15th.—Nausea more troublesome during the past two days. Vomited bile-stained fluid this morning; anorexia, faint icteroid tinging of sclerotic. Some return of tenderness and pain in right hypochondriac region. Patient in a highly nervous state; complains of pains first in one part of abdomen, then in another; tongue thickly coated, bowels constipated. Temperature, 101-2; pulse, 110.

August 22nd.—Trained nurse put in charge; from hence temperature is recorded on chart daily. Patient still restless, emaciation marked, complains greatly of thirst, vomiting has been a prominent symptom last three days. Nutrient enemata every six hours and bowel irrigated with warm water every twenty-four hours, half-pint of saline injected per rectum twice daily to relieve thirst.

August 26th.—Patient's condition remains unsatisfactory.

August 30th.—Consultation with Drs. Drew, Hogg, and Unwin; no definite conclusion arrived at. General opinion seemed to be in favor of

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pyelitis, due to pressure on ureter. Temperature, 103.3; pulse, 120; slight rigor for a few seconds. As the patient's condition was becoming critical further advice was decided on.

August 31st, 5 p.m.—I first saw the case in consultation with Drs. Gabites and Drew. The patient looked extremely ill and emaciated, being markedly changed from the plump healthy young woman she appeared in a photograph taken shortly before her illness. Her facial aspect was pinched and anxious; there was a darkish flush over both malar regions, and a slight icteroid tinge of the skin and sclerotic. Her tongue was coated and red, her pulse quick and weak, running up to 120 at the slightest excitement or movement; she complained of indefinite abdominal pains, chiefly located about the right lumbar region; there was general hyperesthesia of the skin. An exhaustive examination failed to localise any distinct lesion; the uterus was enlarged to its normal size for the period of pregnancy, the fœtal heart and fœtal movements were to be detected, the liver dulness in the right mammary line was less than normal: it was noted, too, that there was entire absence of any enlargement of the thyroid. A blood-count showed 9,000 white cells per cmm. The symptoms seemed to me to point to some form of toxæmia; tuberculosis or enteric were possible conditions, and the diagnosis lay between these and some form of auto-intoxication of pregnancy. On the whole I inclined to the latter view. If tuberculosis had been going on for some weeks we should have expected some evidence by physical examination; there was none anywhere, and the family history was good. Any doubt as to the question of typhoid was subsequently excluded by an examination of the blood, which showed negative results tested by Vidal's re-action.

The examination of the urine supplied the strongest argument in favor of an auto-intoxication; the urine had a peculiar brownish-green color, was clouded when passed, was small in quantity, of low specific gravity (1010), with a marked decrease in urea; there was a slight trace of albumen, some leucocytes and small epithelium from the upper urinary tract, and a few granular casts. It gave a strong acetone re-action with the perchloride of iron. It seemed to me that if the condition was due to typhoid, even allowing that the patient was practically starving from an inability to retain food, yet, with the rapid tissue waste going on plus the excretion for a five months' fœtus, there should have been a much greater excretion of urea.

It was decided that, in addition to saline injections by the bowel, the patient should be moderately purged, fed by the mouth on small quantities of peptonised gruel only, and, with the object of increasing the evidently deficient metabolism, 5 grains of thyroid extract should be given thrice daily.

For the following four days there was a decided improvement. The urine was carefully collected and measured daily; the quantity was found to average 30 ounces daily, an increase in the amount passed prior to the administration of the thyroid. The specific gravity ranged from 1109 to 1013. The total average daily excretion of urea from September 1st to September 7th was 144 grains per diem—needless to remark, a very marked diminution from the normal excretion of a woman in the sixth month of pregnancy. During this period the appearance of the urine was much as at the first examination—cloudy, greenish-brown, considerable uratic deposit after standing, slight occasional tinge of albumen, bile acids in small quantities, with strong acetone reaction to the perchloride of iron. No efficient examination was made for leucin or tyrosin, but ordinary evaporation tests failed to show their presence.

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September 5th.—Dr. Gabites reported :—"Patient improved until to-day; was able to take small quantities of peptonised gruel. For three days temperature mostly sub-normal, and pulse rarely up to 100. Very restless all night, slept for a few minutes at a time; thirst troublesome, gruel discontinued on account of urgent vomiting, delirious at times, patient weaker."

September 10th.—Dr. Gabites wrote me patient had had some weak turns, the pulse was irregular and weak, necessitating the injection of strychnine, and, in consequence, it was decided to discontinue the thyroid extract.

September 11th.—After consultation by letter and telegram in consequence of unfavorable report, and in view of the persistent diminution of the quantity of urea—less than 150 grains per diem—we advised induction of labor without further delay.

September 12th.—I again saw the case, in consultation with Drs. Gabites and Drew; her general condition was worse, emaciation more marked, nausea and vomiting persistent, constant thirst and distress with feeling of extreme exhaustion. Temperature rises daily from 101 to 103 about 4 p.m.; occasional rigors and very marked nervous irritability and restlessness: the nurse states it is impossible to please or manage her. Induction of labor decided on; a bougie passed about 10 p.m.

September 13th.—It was decided that it would be unwise to employ slower methods of emptying the uterus, as the delay would probably have been fatal. At 10 a.m. Dr. Gabites administered ethyl-chloride, followed by ether. Patient was moved to operating table and labor induced; the bougie had effected a moderate amount of dilatation of the cervix; os about the size of a half-crown: digital dilatation failing to efficiently dilate for purposes of delivery, Bozzi's dilater was used, and delivery completed in about two hours. The placenta and membranes were perfectly normal. The child died during delivery.

Dr. Drew, during the latter stage of the operation, administered two pints of saline direct into the veins of the elbow. The patient stood these measures well, and was moved back to bed in fair condition; two hours later, however, she was suddenly seized with pre-cordial distress, intense dyspnœa and restlessness. The pulse at the wrist becoming imperceptible, and the beat of the heart fluttering and irregular, we feared she was about to die from pulmonary embolism. After free administration of strychnine, however, the condition gradually passed off, and towards evening there was a decided improvement.

September 14th.—I saw the patient with Dr. Gabites in the morning. Nurse reported she had passed a better night than she had for some weeks past; the nurse was specially struck with the marked decrease in the nervous irritability: she was now quite manageable, whereas previously she had been a perfectly impossible patient.

September 18th.—Dr. Gabites reports patient has been making steady progress; vomited three times after operation, and complained of severe pain across upper portion of abdomen on two occasions; sleeping well, no restlessness, pulse and temperature normal.

From this date the patient gradually improved, and in about a month had fairly recovered.

Specimens of urine were sent to me, from time to time, subsequently to her delivery; there was a gradual improvement in quantity and quality. The return to normal was, however, gradual, it being some three weeks before it could be considered healthy.

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CASE II.—The second case, to which time will only permit a very brief reference, was seen by me in consultation with Dr. Marshall Macdonald, who has kindly supplied me with data prior to my attendance.

B.F., aged 18, married eight weeks. First consulted Dr. Macdonald on February 8th, 1905; was then five months pregnant; had been attacked in the night with severe abdominal pains; there was also some vomiting and diarrhœa. Uterine contractions could be felt, and were accompanied by pain; os undilated. For ten days following she gradually got worse, temperature reaching 101, or higher, daily; vomiting more or less persistent; pulse ranging from 115 to 120. Urine showed a trace of albumen, was scanty, and of low specific gravity. Patient complained of constant thirst and epigastric discomfort.

February 18th.—The case was first seen by me in consultation with Dr. Macdonald. Patient's general condition was very low; pulse between 130 and 140; temperature, 102.5; tongue, brown and dry. Careful examination failed to elicit evidence of any local lesion; patient was suffering from painful uterine contractions, persistent vomiting, and great thirst; she was much emaciated. It was deemed advisable to remove her to the hospital to secure more favorable surroundings and efficient nursing.

February 18th to 23rd.—Patient was treated by mild laxatives, half a drachm of bromide with saline by the bowel thrice daily, small quantities of peptonised gruel only being administered by the mouth; a few doses of virbirnum with liq. opi. sed. soon subdued the painful uterine contractions.

Beyond the relief from pain, there was little general improvement. Very little food could be retained by the stomach; she slept badly, complained constantly of intense thirst and epigastric discomfort, was highly nervous and irritable, and suffered more or less from constant retching and vomiting, the latter being occasionally bilious in type. The patient was still wasting There was a slight icteroid tint of the skin and sclerotics, a marked decrease in liver dullness; the thyroid gland was not enlarged.

The urine was carefully measured and examined daily: during this period it averaged about 36 ounces daily. It was clouded on passing, was a peculiar brownish-green tinge, which formed a heavy deposit after standing; it was faintly acid reaction; specific gravity ranged from 1008 to 1011; there was generally a trace only of albumen; microscopically, a few round cells, small epithelium from the upper urinary tract, and occasional waxy casts. There was considerable deposit of urates, and crystals of uric acid. Bile pigments and bile acid were detected in small quantities.

No efficient examination for leucin or tyrosin was made; there was always a strong acetone reaction with the perchloride of iron; the urea was markedly diminished, averaging 148 grains per diem.

No local lesion whatever could be found to account for her condition, and we concluded the case to be one of auto-intoxication.

February 24th.—Five grains of thyroid extract were administered thrice daily.

February 25th.—The temperature this afternoon rose to 103.5, her pulse rate being 126; there was a marked increase in the quantity of urine, 52 ounces being passed, of low specific gravity (1009); urea, 220 grains.

February 26th.—She felt better; urine to-day measured 50 ounces. From hence to March 6th she seemed better, nausea and vomiting almost ceased, and the urine averaged from 40 to 50 ounces per diem; it still

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retained the characteristics as when first examined, specific gravity being persistently low and the quantity of urea much less than normal, averaging 140 grains.

On March 6th she said she was quite well, and, despite all warnings, insisted upon leaving the hospital.

March 11th.—Re-admitted. Temperature, 102.4; pulse, 124; condition much as when she was first admitted; urine scanty; low specific gravity (1009).

March 12th.—Her condition was evidently becoming critical, and it was decided that further temporising measures were inadvisable.

March 13th.—Chloroform administered by Vernon Harcourt's inhaler; less than 2 drachms used. Cervix dilated by Hægar's, and a medium-sized Barnes' bag inserted. Small doses of chloral given through the day.

March 14th.—No result from Barnes' bag; chloroform again administered. Digital examination failed, in consequence of the extreme rigidity of the cervix; Bozzi's method soon proved efficient, dilatation being effected without much trouble, and delivery completed.

Nothing abnormal with child or placenta. Child died shortly after birth, sixth month. Towards the termination of the operation two pints of saline were injected into the vein at the elbow.

The patient's subsequent progress was satisfactory. There was a marked increase in the quantity of urine immediately following delivery, but for some days the specific gravity remained low and the urine clouded; it was about a fortnight before it became normal.

Case III. was of a somewhat different type; admitted into the hospital February 13th, 1905.

M.S., aged 28, pregnant three months; had been troubled with retching and vomiting for the past ten weeks. Examination failed to disclose anything abnormal; uterus enlarged to about the usual size for the period of pregnancy; no cervical catarrh or erosion. Urine scanty, but the exact quantity difficult to estimate in consequence of the relaxed state of the bowels from medicine.

Urinary examination showed brownish thick cloud, containing epithelium, urate, and a few round cells; no albumen; specific gravity, 1020. The nurse is sure the total quantity is less than one pint daily: urea estimated at 180 per diem.

The gravity of this case was not at first sufficiently realised. I was inclined to view it as one of the severe types of reflex vomiting of early pregnancy, which, as a rule, rapidly improve in hospital with rest, purgation, and careful dieting.

February 15th.—Still vomiting; chlorotone, 5 grains every three to six hours, as required.

February 17th.—Vomiting less, but patient heavy and drowsy; looks worse. Pulse, weak, 120. Chlorotone stopped; bismuth and hydrocyanic acid by the mouth, and saline injections by the bowel, each containing, in addition to the saline, ½ drachm bromide of soda.

February 20th.—Less vomiting, and now able to retain small quantities of peptonised gruel.

February 24th.—Not much trouble with sickness; can take but little nourishment, feels sick, occasionally brings up a little bilious fluid. She is not improving. Pulse averaging between 120 and 130. The urine is per-

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sistently scanty—less than 18 ounces per diem—dark and cloudy; slight trace of albumen; strong acetone reaction with the perchloride of iron; urea, 120 grains.

February 28th.—For the last two or three days the patient has been losing ground. She is not vomiting, but seems excessively exhausted; pulse persistently above 120; she is dull and drowsy. Although the patient has lost much flesh she is not excessively emaciated; her temperature for to-day, for the first time, was raised—100.6. There is a marked ictoroid tinge of the skin, the liver dullness is decreased; the urine is further reduced in quantity, only 10 ounces yesterday and 16 to-day; shows bile acid and pigment in small quantities; total urea in twenty-four hours less than 100 grains.

Consultation with Staff.—In view of the fact that so many cases of vomiting of early pregnancy are relieved by local treatment to the cervix, it was thought desirable to employ local measures before resorting to emptying the uterus. The cervical canal was therefore dilated with ivory glove stretchers, as recommended by Edgar, and carbolic acid applied to the whole cervical canal; this was easily done, without rupturing the membranes. Chloroform was administered by Vernon Harcourt's inhaler, which answered excellently; less than 2 drachms being used.

When the patient was returned to bed she was apparently in very fair condition, and I did not expect she would be much upset by what had been done. The following day, on the usual morning visit, I was shocked to find that the patient had become comatose and died about 11 p.m. the previous evening.

No intra-venous injection had been administered in this case, as her condition at the time I left her did not seem to call for its use. *Post mortem* was refused.

It must, I think, be pretty evident to you that in cases I. and II. we had to deal with very similar conditions. The most prominent objective symptoms were the nausea and vomiting, but in reality they were minor incidents; the rapid wasting, the intense nervous irritability, the constant thirst, and excessive lassitude and debility were, in my opinion, factors of greater importance than the vomiting itself.

In all three cases on some days the vomiting ceased, or caused but little inconvenience; but it was noticed that the cessation of the vomiting was accompanied by no alleviation of the general symptoms. The temperature chart in cases I. and II. is remarkable, and I wish to draw special attention to it, for, although reports of cases of toxæmia are by no means uncommon, nowhere in medical literature have I been able to lay my hands upon any record of temperature. In view of the theory of the causation of this condition, to which it is my intention to refer later, some such elevation in temperature might, I think, be anticipated.

The marked deficiency in the daily excretion of urea is, to my mind, the cardinal diagnostic point in all these cases, and upon which more reliance can be placed than upon any other individual symptom. It might be suggested that the deficiency in the urea resulted from the starvation alone; but when we remember that previous to their illness both these patients were healthy, well-nourished young women, that during their illness they wasted rapidly, had a more or less persistently high temperature, and, further, had to excrete material for a five-months' living fœtus, one is forced to conclude that the consumption of their own tissues, apart from a certain amount of nourishment that had been retained, should, under normal conditions, have produced a much larger excretion.

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Case III., although of a different type, seems to me clearly toxic. The operation of dilatation of the cervix was simple and easy, taking only a few minutes, and should not have caused any serious disturbance. Although the girl was seriously ill, her condition did not seem immediately alarming; after her return to the ward there was no evidence of shock, she quickly recovered from the anæsthetic, and for some hours seemed fairly well; later, towards evening, she became drowsy, and finally comatose, and died. This seems more in keeping with death from toxæmia than from simple exhaustion. It was most unfortunate that no communication was sent me as to her condition; had an intra-venous saline injection been administered I think her life might have been saved.

As remarked earlier in this paper, this condition of toxæmia has received scant attention in our midwifery textbooks, and, apart from the eclampsic condition, is almost altogether ignored. Cases, however, may be found scattered through medical literature; and if you look up Frerich's treatise on "Diseases of the Liver," published in 1858, you will find described under a series of cases of acute yellow atrophy of the liver in the pregnant woman, under observation case No. 14, a report of a very similar condition to my first and second cases. His *precis* reads, "Repeated attacks of lumbago in the seventh month of pregnancy, gastric catarrh, icterus, delirium, convulsions, death under symptoms of blood-poisoning." In the more detailed account of this case, which follows, the convulsions were apparently of a very trivial character, and only occurred towards the termination of the case, when, he remarks, "convulsive movements occurred in the muscles of the face neck, and arms."

In the American Textbook of Obstetrics I have found the only useful account that, so far, I have come across in any obstetric work where this condition is considered apart from eclampsia. Under the heading of "The Toxæmia of Pregnancy" the writer suggests that toxic material which may jeopardise the life of mother and child may result from the deficiency in metabolic processes. The toxins he considers alkaloid in nature, and the symptoms they produce upon the pregnant patient are especially addressed to the nervous system. He quotes Van Santvoord, who, from clinical observation, ascribed toxæmia during pregnancy very largely to deficient action of the liver, by which an insufficient formation of urea causes the patient to retain in her blood toxic material. Further, in diagnosing the toxæmia of pregnancy he points out two clinical signs of special value: first in importance, the amount and character of the urine, the diminution in the output of urea being of great significance; second, the condition of the nervous system; the presence of pain, headache, thirst, lassitude; disturbance of vision, of hearing, of taste; sleeplessness or lethargy, irritability or apathy, melancholia, nausea and vomiting, are all symptoms to be noted.

More recent work by Nicholson and Eden has also drawn the attention of the profession in Britain to this subject, but the most valuable information that I have so far come across is a communication to the "American Journal of Obstetrics," in the February number of this year, by Dr. Ewing, Professor of Pathology, Cornell University, Medical College, New York, and to this I shall specially refer as epitomising recent views and being specially valuable from its wealth of pathological data.

Dr. Ewing records the results of thirteen years' study of this subject in the Sloan Maternity Hospital, under the heading of "The Pathological Anatomy and Pathogenesis of the Toxæmia of Pregnancy." From my reading of this article it seems to me that, clinically, his cases can be ranged under

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two main types:—First, the toxæmia accompanied by eclampsia: second, the toxæmia where convulsions do not occur, but where symptoms may assume varying grades of severity; one extreme being represented by what we look upon as the physiological vomiting of early pregnancy, the other by the disease known as acute yellow atrophy of the liver. Ewing holds that the focal lesion in both these conditions is the same organ, namely, the liver, and in a large number of *post mortems* in fatal cases of eclampsia he found, without exception in every case, marked pathological changes were discoverable in the liver cells, and consequent loss of function. It is not, however, with this class that I want to-day to deal.

The Class II., or toxæmia apart from eclampsia, has been usually described under the term of "pernicious vomiting of pregnancy" in its less virulent type, "acute yellow atrophy of the liver" in the more fulminating variety. Ewing reports autopsies on three cases selected from a number clinically diagnosed as vomiting of pregnancy, and shows that this disease, when fatal, may be associated with (1) acute yellow atrophy of the liver; (2) with the necrotic process in the liver, which is not reduced in size; (3) with less marked degenerative change in the liver which may be overlooked or ignored, but by microscopic examination reveals diffused granular and fatty changes; foci of intense degeneration with disorganisation of the liver cells; foci of partial necrosis, in which the cells showed watery contents and pyknotic nuclei, and areas where the cells are distended with bile pigments, which indicated extensive auto-lysis and profound disturbance of the function of the liver—lesions, moreover, very similar to those to be found in eclampsia.

After dealing at length with the pathological changes in the liver, to which I do not propose further reference, he proceeds to deal with the pathogenesis of the toxæmia of pregnancy:—

"The exact nature of the disturbance of nitrogenous metabolism, which is responsible for the clinical manifestations of the toxæmia of pregnancy, is a failure of oxidising capacity on the part of the liver; for this reason the proteid derivatives, which are normally combined by the liver into urea, are no longer combined, but circulate free in the blood in some poisonous form, and are, to some extent, excreted by the kidneys. The complex nature of the sources of the poisons renders less obscure the fact that the clinical manifestations of the toxæmia of pregnancy vary from mild vomiting to acute yellow atrophy.

"The relation of the mild to the severe cases of vomiting of pregnancy also required very careful consideration. No one doubts that mild and fatal cases of eclampsia are identical in nature, but there persists a definite impression in some quarters that acute yellow atrophy never occurs in a mild form, but is always fatal, while it is still generally taught that the mild vomiting of pregnancy is physiological and the severe vomiting is an exaggerated form of the other—but without definite pathological basis.

"Impressed by the supposed rarity and unfavorable prognosis of acute yellow atrophy, clinicians seldom attempt the diagnosis of this disease; hence only the fatal cases are commonly recognised.

"Recent study of the toxæmia of pregnancy, and many other clinical conditions, furnishes abundant evidence that the morbid process in acute yellow atrophy is of very frequent occurrence, and is often followed by recovery.

"Vomiting is seldom the only symptom present in early cases of vomiting of pregnancy; observation usually shows also striking mental symptoms—

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headache, hysterical tendencies, pruritis, constipation, lassitude, &c., all of which doubtless result from the mild auto-intoxication which is the cause of the vomiting.

"The urinary changes in the toxæmia of pregnancy are of great importance for the diagnosis and prognosis, as well as for their bearing on the essential nature of the disease. These changes indicate chiefly deficient oxidation of proteid derivatives instead of urea, uric acid, ammonia, leucin, and tyrosin, and other unoxidised proteid radicals appear in the urine. Albumen casts are sometimes present, but may be absent in dangerous or even fatal cases.

"The present view of the toxæmia of pregnancy classes the disease as a functional disturbance of the liver, usually, but not necessarily, attended by severe anatomical lesions in this organ; and, secondarily, with functional disturbance and anatomical lesions of the kidney and other organs. The ground for regarding the disease as primarily in the liver is the fact that the synthesis of urea is exclusively a function of the liver."

There are endless theories as to the factors underlying this failure of metabolism in the liver; probably a combination of causes are at work:—

(1) The increased demands of the growing fœtus.

(2) Nervous disturbance. In two of the cases reported the patient had suffered great mental distress from the fact that pregnancy had occurred some time previous to marriage.

(3) Constipation is excessively common in early pregnancy, mechanical factors, &c. Some think in consequence of this loaded state of the bowel intestinal toxins are formed, which, when absorbed, may be a cause of the liver failure.

(4) Then again it has been noted in the fifth month of pregnancy there is physiologically a marked increase in the thyroid gland, with increased functional activity; when this fails to occur the normal metabolic processes may be interfered with.

In cases I. and II. it was specially noted that the thyroid gland could not be felt.

(5) Another recent theory of great interest is: It has been found there was in normal pregnancy synthycial elements, or the proliferating epithelium of the chorionic villi find their way into the systemic vein, and have been deported to the liver. Whether this is a physiological function which, in some instances, may exceed its normal limits, opens up a question of great interest at present but little understood.

One other theory I may mention, which cases I. and II. at any rate strongly negative, is that the toxæmic condition may depend on a diseased condition of the maternal uterine membranes. In both my cases the placenta, membranes, and fœtus were perfectly normal.

As Ewing shrewdly remarks, "It is not necessary to explain the ultimate origin of the toxæmia before recognising its existence and practical importance." One conclusion Ewing draws from the pathological study of his cases is the absence of any necessarily fatal character in the disease:—"We are not dealing with an uncontrollable bacterial infection, nor with a hopeless anatomical lesion, but with a disturbance of function which only secondarily leads to organic lesions. If the poison can be eliminated, or its further production prevented, there is nothing in the majority of the lesions which is incompatible with life, and there is demonstrative proof that extensive lesions of the liver of this type are sometimes followed by spontaneous recovery."

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I fear it may seem that I have taken a great deal of time in describing these cases; but, personally, being so impressed with the importance of the condition and with the fact that it is so largely overlooked, and the symptoms are so often highly misleading, I feel justified in dealing with it at some length.

Do not for a moment think it is my intention to infer that every case of vomiting in pregnancy is the result of a dangerous toxæmia; clinical experience must have taught most of us how frequently this condition is relieved by comparatively simple measures: as, for example, unloading distended bowels, careful regulation of diet, quiet and rest; or, maybe, replacement of a displaced pregnant uterus. What, however, I do strenuously contend is this, that when all such sources of extraneous irritation have been removed, and when the vomiting and nausea persist, especially if accompanied by a train of severe nervous disturbances, a careful examination of the urine is demanded with special reference to the daily output of urea. If such examination serves to confirm the suspicions of toxæmia, the less the treatment is directed to the arrest of the vomiting the better for the patient; the sickness may be but an attempt to eliminate the poison. The bowels must be freely opened and kept open, any food by the mouth must be of the most digestible character and in very small quantities. Saline injections by the rectum, if absorbed, are of great value, by diluting and washing the blood and relieving the troublesome thirst. Where there is marked nervous irritability half-drachm doses of bromide of soda may be given two or three times daily with the saline by the bowel. If the sleep is broken and disturbed, half a drachm of chloral by the bowel at bedtime is the best remedy I know, and the least injurious.

The thyroid extract is well worth a trial, and is useful by increasing metabolism and by its diuretic action on the kidneys.

When a reasonable trial of these measures fails, and when the maternal system seems unable to accommodate itself to the demands of reproductive life, the only resource left is to empty the uterus; nor should this be delayed till the patient is *in extremis*, but, with that valuable aid to our resources—namely, intra-venous saline injections—I do not anticipate that fatalities attending the operation of emptying the uterus will be so frequent in the future as they have been in the past.

Further, it seems to me it is no longer justifiable to make a return of death as a result of the pernicious vomiting of pregnancy unless the liver has been subjected to thorough macroscopic and microscopic examination; and, if the alterations to which I have just referred are found, the death certificate would more correctly record the fatality as due to the toxæmia of pregnancy.

One further point before concluding, that is, to refer to the temperature chart in cases I. and II. Nowhere have I been able to find any data dealing with this point. In both cases I. and II. careful and repeated examinations failed to find any concurrent condition to account for the elevation in the temperature; but viewing these cases in the light of Ewing's article, and looking upon them, as I certainly am inclined to do, as a type of disease closely resembling acute yellow atrophy of the liver, might we not expect this condition to be attended by a rise of temperature? In many of the cases reported of acute yellow atrophy of the liver considerable elevation of temperature has been noticed in their early and late stages, and in a less virulent form there is no reason why a similar rise should not occur.

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In cases I. and II. the symptoms in many respects resemble acute yellow atrophy of the liver, and its existence was seriously discussed at our first consultation; the decrease in liver dullness, nervous irritability, the marked lassitude and debility, the slight ictoroid tint, and the character of the urine were all highly suggestive of the disease.

ACUTE DILATATION OF THE STOMACH FOLLOWING ABDOMINAL SECTION.

By J. A. G. Hamilton, M.B., &c.

Acute dilatation of the stomach is a mysterious, and most fatal, condition. There is very little literature on the subject. None of the leading authors on gynæcology mention it as a possible complication in abdominal surgery. Campbell Thomson, of the Middlesex Hospital, has collected forty-four cases. No less than twelve were associated with surgical operations; only four, however, were associated with abdominal section. I have been doing abdominal work for the last twenty-five years, and have only quite lately been able to diagnose two cases of this condition; but probably I, in common with others, have overlooked many cases in the past. Patients, with constant vomiting more or less immediately following an abdominal section, which were put down as due to peritonitis, were in my opinion due, in many cases at any rate, to acute dilatation of the stomach. One case in particular I can recollect which followed an operation for pus tubes. I was at a loss to account for the symptoms, which somewhat differed from those usually seen in peritonitis; but, with increased experience, I have little doubt this was a case of acute dilatation of stomach. Pepper and Stangel suggest that the immediate cause is spasm of the pylorus; but it seems more probable that the symptoms are dependent on the absorption of toxins from the cavity of the stomach. Campbell Thomson, in an article in the *Lancet*, 1902, inclines to the belief that a primary paralysis of the organ must be regarded as the underlying cause in the great majority of cases, and not, as has been suggested, a constriction caused by tension on the inferior mesenteric artery under certain circumstances. In most cases the first part of the duodenum shares in the dilatation, the distension ceasing abruptly with collapsed bowel further on. Thomson relates a case in which he had made a necropsy. The acute dilatation of stomach was accompanied by distension of 8 feet of small intestine, and says in this case the superior mesenteric artery could not be considered as the constructing force, nor were there any other signs of constriction present. We have all met with cases of paralytic distension of the intestines which occurs during cases of acute peritonitis; in these cases we often find, during life or after death, the stomach has participated in the paralytic condition.

This is important, when we remember that peritonitis is clinically the condition which most resembles acute dilatation of the stomach, and for which many primary cases of the latter have been mistaken. As in every case where acute dilatation of the stomach appears after an abdominal operation, we have to decide whether the dilatation is a primary symptom or only the predominating feature of a general peritonitis. The symptoms are usually sudden in onset. A patient may seem to be doing well for some days or weeks after operation, when she complains of a fullness and discomfort in

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her abdomen; this is quickly followed by the vomiting of huge quantities of greenish or brownish slimy fluid, with a sour odour; at the same time the pulse rate rapidly rises, the temperature falls or remains normal; the urine becomes scanty and loaded with urates; the bowels may act normally, or there may be diarrhœa, and the patient becomes progressively weaker, and may die in a short time. Physical examination shows distension of the abdomen, and on palpation a sense of fluctuation and a succussion splash may often be obtained, both of which are valuable aids to diagnosis when they are present. A peculiar fact about this condition is that the thinning of the walls of the stomach is not commensurate with the enormous dilatation. One would expect to find the walls of the stomach very thin, as in acute dilatation of a previously healthy bladder; but such is not the case, the walls appear to retain their normal thickness. If an attempt were made to blow out a stomach to the size it would certainly rupture at its lower curvature. From this it would seem as if the toxins have a softening effect on the stomach which permits the enormous stretching. As regards treatment, as soon as the condition is suspected lavage of the stomach should be started. This little operation is not a pleasant one, and usually patients strongly object to it; but, in acute dilatation of the stomach, they not only do not object to the passage of a tube but they usually beg for it, as it always relieves the constant vomiting—for a time at any rate; and in milder cases one or two washings out may cure the patient. All nutriment should, as far as possible, be given by the rectum; the thirst which is sometimes so distressing is best relieved by saline rectal injections and sips of hot water. Strychnine should be given hypodermically. I have tried salycilate of eserine, but without any good effect; if necessary, normal saline may be given intravenously. Drugs are generally useless, as there is no reasonable prospect of their being absorbed. If this milder treatment does not give relief, then some surgical treatment is called for. Cases in which the stomach has been opened, drained, and closed again have not done well. Robson and Moynihan, in their book on "Diseases of the Stomach," suggest doing a gastro-enterostomy, but it has struck me a jejunostomy would be more to the point: a gastro-enterostomy will only pass on the toxins, which nature is trying to eliminate by the mouth. If the patient cannot bear the stomach tube, which in my experience they generally bear well, then a very minute opening might be made in the stomach after Witzell's method—that is, an inch incision made in the stomach with a plication round a small tube, through which the stomach can be washed out, and, *a fortiori*, a similar opening made in the small intestine, as high up as possible, and the patient fed through this opening in the bowel, and then allowing the stomach a chance of eliminating the poisonous toxins. As these patients are generally in a bad condition, I think the operation would be better done under local anæsthesia in preference to a general one. There are no recorded cases, as far as I know, where gastro-enterostomy has been put to the test; but a friend of mine assures me he tried it in a non-post-operation case, which was diagnosed as "spasmus pylori." On opening the abdomen the stomach was found filling the abdomen like an immense ovarian cyst, with distension of the duodenum. A gastro-enterostomy was done, but the patient died. Although I have only seen two cases of acute dilatation of the stomach in my own practice, two other cases have come under my notice—one in an old man who had an operation for a double hernia; with frequent lavage of the stomach he made a good recovery. The other case occurred in a man who had an acute gonorrhœa, with pus in his epidydimis. The abdomen was opened, and a stomach reaching down to pubes found; it was opened and drained, but the patient died.

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The following are the notes of my two cases:—CASE I.—The history of this case is interesting. E. W., *æt.* 22 years, admitted to Adelaide Hospital, May 5th, 1905. Menses, which have been regular, ceased in February. On April 20th she jumped off a washstand: after that a blood-stained discharge came on which has lasted ever since. Complains of sharp pains in lower part of the abdomen, which come on frequently. P. 126, small; temp 99.8. On examination, cervix softened; body of uterus enlarged slightly, in good position; sound passed 3¾ inches; no discoloration of vagina; some milky fluid in breasts; no areola; nothing to be felt in fornices; both lateral fornices very tender. May 7th, uterus curetted. Some thickened endometrium removed. I thought it might possibly be a case of ruptured tubal pregnancy, and told my house surgeon to let me know at once if any sudden change occurred.

May 19th.—Got up for a short time to-day; feels well. No discharge. Pulse and temp. normal. On the evening of the same day she had a sudden rise of temp., 101, pulse 136. Feels as if something had given way. My house surgeon telephoned me her symptoms at 8 p.m., and I thought my original suspicion of ruptured tubal pregnancy was confirmed. I went down to the hospital at once and opened her abdomen. No blood escaped when the peritoneum was opened. The right tube was found thickened and inflamed and œdematous, but contained no pus. The left tube was in the same condition, but from the ostium abdominalis, which was patent, a few drops of pus escaped. This explained her condition. There had evidently been a sudden leakage of pus from the left tube. Both tubes and ovaries were removed, and abdomen washed out with large quantities of normal saline solutions.

May 20th.—Feels comfortable. P., 106; temp., 99. Flatus troublesome. Ordered eserine salycilate, one-forthieth gr. t.d.s.

May 21st.—P. 110; temp., 98.6. Slight distension. To have calomel, one gr. every hour, for five doses. In the evening bowels were well moved.

May 22nd.—Feels better. P., 120; temp., normal. Bowels moved. Abdomen a little distended. Eserine, one-fortieth gr. every six hours. Vomited greenish fluid. Ordered strychnine, one-thirtieth gr., hypodermically, every four hours.

May 22nd.—Been vomiting large quantities of greenish fluid all night. Temp., normal; p., 120; only passed 8 ounces of urine during last twenty-four hours. Abdominal wound looking well. Some horsehair sutures removed. Stomach washed out night and morning with two quarts of saline solution: a large quantity of greenish fluid returned. She expressed herself as much relieved after each lavage. On palpation, stomach distended down below umbilicus. Succussion splash felt. She went on pretty much the same until 10 a.m. on May 26th, when she suddenly collapsed: pulse imperceptible, temp. sub-normal; abdomen more distended. At 3.30 on same afternoon abdomen was re-opened. A dilated stomach was found reaching down to pubes, like an immense ovarian cyst. Small intestine collapsed. An opening was made in stomach, and several pints of dark-green sour-smelling fluid evacuated. Opening in stomach closed in two layers; abdomen closed. Two pints of intravenous saline solution given; but she never rallied after operation.

Post-mortem.—Stomach found extending down to umbilicus. Walls not much thinned out. The dilatation extended throughout whole course of duodenum, and ceased abruptly at duodeno-jejunal flexure, below which the intestines were collapsed. There was no constriction at duodeno-jejunal flexure.

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CASE II.—Miss B., aged 42, August 8th, 1905. Supra-vaginal hysterectomy, for myoma of uterus. The operation was a difficult one, as she had a marked anterior curvature of spine, with a contracted right hip, which lay partly across the abdomen and very much hampered the operation. She made an excellent recovery. Pulse ranging from 90 to 100, temp. not over 100. She did well until August 28th, twenty days after operation, when she complained of pain in epigastrium, and vomited 6 ounces of dark-brown fluid: vomited continuously all that night; temp., 98.4; pulse, 126. Urine scanty, in fact almost suppressed—only passed 5 ounces last twenty-four hours. Rapid dilatation ensued, and the stomach not only formed a large swelling, filling up the superior abdominal region, but also extended well below the umbilicus towards the pubes. Stomach washed out with saline solution, three quarts; a quantity of brownish fluid came away. Ordered strychnine, one-thirtieth gr. four-hourly, rectal feeding; nothing by mouth but sips of hot water, to relieve thirst, which was excessive.

August 29th.—Feels comfortable. No vomiting since wash-out. Distension less. P. 100, temp. normal.

August 31st.—P. 90. Distension almost disappeared; able to take slight nourishment. Since then she has made good progress.

In both these cases the condition was recognised early, and stomach lavage commenced soon after vomiting appeared; but evidently the toxine infection was more severe in the first than in the second case. Whether the original infection had any hand in the cause in the first case I cannot say.

THE DIFFERENTIAL DIAGNOSIS OF APPENDICITIS IN YOUNG WOMEN.

BY J. W. DUNBAR HOOPER, L.R.C.P., et. S. ED.

During the last five years, in private practice alone, sixty cases of inflammation of the appendix in women have come under my notice; of which forty were clearly associated with, or mistaken for, diseases of the reproductive organs. Of these latter cases thirty-one occurred in women under 30 years of age, and were operated upon; and in the following notes I have limited myself to these instances, entirely omitting children under 10 (four cases), and women over 30, of which the oldest was 58. For sixteen years I had under observation all the boys at Scotch College, East Melbourne, and during the whole of that time only two cases of inflammation of the appendix occurred, of which only one submitted to operation. Yet in five years, while medical officer to a large public girls' school, seven cases of appendicitis have come under my notice, of which five submitted to operation. Eleven cases were brought to me for opinion on a (supposed) purely gynæcological condition. Eight of these eventually proved to be an appendicitis, which, on operation, was associated with some lesion of the right ovary, or fallopian tube.

It seems to me that, until Howard Kelly's recent publication (1905), insufficient attention had been paid to the diseases of the appendix in young women. One is apt to regard all abdominal and pelvic complaints in women as arising from one or other of the many gynæcological lesions. It is, however, a very serious matter to allow a school girl, month after month, to dread her

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menstrual period, to become morbid, invalided, unable to work for competitive examinations, to always have headache and malaise, when one thorough examination might elicit certain facts, viz., that either the appendix itself was to blame or adhering to one or other of the pelvic organs; and I am sure that, to-day, no parent or guardian would hesitate to allow the necessary operation on such a girl, once assured of its justice by a careful and competent medical man. Many girls, of course, suffer from anæmia, ovarian neuralgia, malaise, and headache, uric acidæmia, or even hysteria— "and some may have appendicitis on the brain" (Howard Kelly); but I do earnestly say that, of my own knowledge, some sad mistakes have occurred; as when a girl has been pronounced to be suffering from one or other of these complaints, whereas all the while she has had an inflamed and dangerously situated appendix. And when one thinks of it, such a state is not to be wondered at. School girls, certainly until quite recently, were never allowed sufficient muscular exercise; nearly always suffered from constipation, and it was even regarded as "unladylike" to own to such a common complaint. It is a matter of daily observation how little fluid, simple water, is drunk by school girls as compared with the ordinary boy who plays football, tennis, and cricket; and that water is necessary to wash away micro-organisms and flush the kidneys, and induce peristalsis. And when the resistance is lowered by constipation, anæmia, and copræmia, then bacteria, especially the B.C.C., travel into the appendix, and there or elsewhere, by their toxins, generate chronic blood-poisoning. This has been so frequently demonstrated that it seems a wonder to me how we can ever overlook the incidents.

Furthermore, we occasionally get those difficult cases where a young woman is said to suffer from gravel, urinary or renal calculi, and has to endure severe attacks of pain and vomiting, when probably the application of the X-rays would demonstrate the presence or absence of such calculi, and leave one free to investigate the condition of the appendix. I admit that it is exceptionally hard, in some cases, to undertake the responsibility of advising an operation for the removal of the appendix after only one mild attack of inflammation, when we are bound to say that such an attack *may* never be repeated. But, on the other hand, the laity is well educated in popular medicine, and many fully recognise the seriousness of the condition.

We now regard appendicitis to be due to micro-organisms which reduce the vitality of the patient, and quite possibly at any moment (and that a most inconvenient one—on a journey, or a visit to friend at a distance) these cocci cause toxæmia, inflammation, which may result in rupture of the appendix, suppuration, septic peritonitis, and death. I doubt very much if there is one medical man in this Section who, during the last ten years, does not know among his own acquaintance of some young life laid low before an operation had been carried out. It has occurred three times to me, within two years, to be called to little children under 5 years of age when they were *in extremis*, and subsequent examination proved suppurative septic peritonitis, due to perforated appendices, and in each case there was a history of frequent attacks of colic, for which a medical man had not been consulted. I feel very strongly that if, in my limited experience since I left the Women's Hospital, such a number of cases should come to me as a gynæcologist, many more must have come under the notice of my brethren in general family practice.

I will not trouble you with the various methods of operation, or of treating the wound; these will be dealt with in the Surgical Section. I merely wish here to draw attention to what, in my opinion, has been an unrecognised fact, namely, *that appendicitis, acute and chronic, occurs more frequently in young girls than we hitherto have believed.* Yet I do not want to become a

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mono-maniac on appendicitis in girls! It would be only fair to say that, if I have sixty cases of appendicitis to report in five years' experience, I must, in these same years, probably have had nearly 600 gynæcological cases. In this vast continent so many of our people send their daughters to the cities, Melbourne, Sydney, Adelaide, Perth, and for their education, and these girls return home, many of them, to live in districts far away from medical advice. During these important years of their life they come under the attention of the medical officer of their schools, or have the advice of some practitioner in active family practice, and these are the years during which a diseased and disordered appendix can often be recognised, the parents or guardians warned, and on them rests the responsibility if the modern surgical treatment be not availed of while it is procurable.

I have reduced my experience to a table, for convenience and in order to make my paper as short as possible. If that paper is worthy of your notice, you will, I hope, find something of interest in every case; and I will ask you to specially observe that every appendix removed has been submitted for examination to an independent pathologist. And here I have to thank Dr. Mollison, Pathologist to the Melbourne Hospital, and Dr. MacKeddie, Clinical Pathologist to the Alfred Hospital, for very careful and interesting work. I do not feel that I have once needlessly exposed a young woman to an unnecessary operation, and I can look on these thirty cases as justifying the carefully considered advice given, namely, that, having once had an attack of inflammation in an organ "awkwardly placed" (as Worcester puts it) they should have that organ removed while it is fairly safe to undergo the operation under favorable circumstances, rather than await—it may be for a month or for many years—a second or third attack, when the inflammation may be so rapid as to preclude the chances of operation; indeed, the inflammation may occur at such a place, or such a date, that the sufferer is unable to even get the chance of an operation. I would venture to emphasize the fact that every one of these sixty cases—and I suppose that my professional brethren have had the same experience—has given me infinitely more worry, more anxiety in dealing with friends and relatives, than in managing the patient herself, or in considering the operation or its possible complications.

I do not allow time to be wasted in useless discussion, but seize the earliest and best moment for relieving a condition which at any moment may end in disaster. These cases require knowledge and decision, and, perhaps, above all, abundant confidence in aseptic surgery—though, as is well known, the peritoneum, if involved, leaves a large margin for error (Edmund Owen).

Inflammatory diseases of the appendix in women are frequently found associated with pelvic peritonitis, most often complicated with lesions of the right fallopian tube and ovary. The appendix itself may adhere to the bladder, the ureter, or broad ligament. In many cases the appendix is adherent to the posterior wall of the cæcum, or its tip may be fixed to the kidney or gall-bladder. The accompanying toxæmia of appendicitis is most dangerous when it exists without well-defined inflammation, being due to an excessive number of micro-organisms shut up in the appendix, and absorption of their toxins into the system by the lymphatics and veins. Pozzi and Tallamon and Landau have urged the importance of recognising the relationship between gynæcology and appendicitis. Dysmenorrhœa is, undoubtedly, in many cases wholly due to chronic inflammation of the appendix, with adhesions in the right pelvis. Howard Kelly records the practice of the John Hopkins' Hospital, that in five major gynæcological operations four may probably have these complications; and in all diagnoses of dermoids, ovarian cysts, parovarian cysts, uterine myomata, and ectopic gestations,

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possible complications with the appendix are to be borne in mind. Therefore, we have to decide whether the case is one of appendicitis alone, or of pelvic disease, or of both : *and it is partly due to errors in diagnosis that appendicitis, being often overlooked in women, is, apparently, more common in men.* To-day Howard Kelly invariably removes the appendix in all abdominal operations, unless the condition of the patient absolutely forbids the extra strain. Duhressen and Rosenblum declare that chronic appendicitis is frequently the cause of uterine displacements, and Howard Kelly states that, out of eleven cases of appendicitis with adhesions, five certainly caused displacement of the uterus.

Some observers have claimed to find a definite arterial supply from the right ovary to the appendix, but Professor D. J. Cunningham failed to do so, and Howard Kelly denies its existence. It is mentioned in Quain's Anatomy. There is an undoubted relationship between pain in the appendix and the menstrual period, not only when the appendix is in the pelvis, but also when it is retro-cæcal. At these periods the splanchnic area is congested, and induces a favorable soil for the micro-organisms ; also, there is a definite relationship between rheumatic fever and appendicitis, and the latter is certainly influenced by the influenza bacillus. All the vascular supply of the appendix is from the right ilio-colic branch of the superior mesenteric artery, and undoubtedly the relatively strong muscular coats of the appendix render it capable of producing active peristalsis, inducing typical appendicular colic when inflamed or containing concretions.

It has been found possible to demonstrate the presence of a thin peritoneal fold passing from the mesoappendix to the adjacent iliac serosa in a median direction to the infundibulo-pelvic-ligament, also called the vascular pedicle of the ovary. On traction, these folds can be recognised distinctly with their blood-vessels and lymphatics, and thus there would be a connection between the vessels of the appendix and ovary, or between the portal and systemic circulations ; yet Kelly positively denies the existence of any considerable vascular or lymphatic connection between the ovary and the appendix, for he points out that the blood-vessels of each are completely formed before they become neighboring organs. He doubts whether the ovary can be injected from the appendix through the ligament, and, in certain experiments, not one single lymph channel was seen to pass in the peritoneum towards the ovary. But Lafforgue is satisfied that he found the appendicular ovarian ligament in seventeen cases out of ninety subjects. Treves denies that the cæcum has a mesentery. As a matter of fact there are nine anatomical directions to which the appendix may point, and there is no region, from the liver to the pelvic floor, where the appendix may not be found, the malposition being due either to pelvic adhesions or to arrested fœtal development.

The lymphatics of the appendix are subject to great variations, and at the base of the mesoappendix, in the angle which separates the appendix and cæcum from small intestines, there lies a gland—the appendicular gland—and it attains to considerable size. It may be considered the last of the misnamed mesenteric glands (Claudo). The nerves of the appendix are supplied from the superior mesenteric plexus, and divided into the plexus of Auerbach and that of Meissner. There is a small number of ganglia in the mucosa connected with one another, and forming a plexus with the glands of Lieberkuhn, and a careful microscopical examination of apparently healthy glands in the neighborhood of an inflamed appendix has revealed B.C.C. in those glands ; and in a case of suppuration, after removal of diseased appendix, the wound was reopened and the glands removed, and found infected with B.C.C., and the patient recovered.

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The primary existing causes of an acute attack of appendicitis are constipation, and an undigested meal, setting up acute catarrh of the colon and cæcum.

Has the appendix any functions? It exists in man, in rodents, and in apes, and is peculiarly rich in lymphoid tissue, like the tonsils, and has been called the abdominal tonsil. The carnivora have no appendix. Many observers are convinced that the appendix has some unknown influence on digestion in the cæcum, and certainly it has regular and powerful peristaltic action. Whether the appendix has any healthy function or not, unquestionably we know of certain factors which give rise to pathological conditions in the organ. For instance, deficient mastication, bolting the food, chronic dyspepsia, and the infection of a bolus of food by numerous micro-organisms which find their home in dental caries (William Hunter on "Oral Sepsis and Anæmia"). And in the aged, or gouty, or syphilitic, an endarteritis may favor gangrene of the appendix, and the danger of kinking is that it may effectively cut off the entire blood supply to the appendix. The inflammation, or catarrh, stimulating the nerve endings in the lumen thus causes hyperalgesia. Mayo Robson doubts whether appendicitis has actually increased, though he admits our methods of diagnosis have vastly improved. In 1892 many cases of suppuration in the iliac regions were operated upon in the London Hospital, and the proportion of cases was 2.5 males to 2.1 females.

Recent bacteriological research has proved that, in inflammation of the appendix, there are found within the lumen of the tube, and along the lymphatics, streptococci, more rarely staphylococci, the bacillus coli communis, various other micrococci, Weiselbaum's intra-cellular diplococcus, the bacilli of pneumonia, influenza, and diphtheria; actinomycosis, and the bacillus of amœbic dysentery (Osler, 1890). There is a distinct analogy between catarrhs of the nose, the urinary and gall-bladders, in the formations of various calculi, rhinoliths, or gall-stones, and those concretions of the salts of lime and magnesia, with inspicated masses of bacteria, and some little fæcal matter, which are found within the appendix itself (Sherren, in *The Practitioner*, June, 1905). Sir Frederick Treves regards the presence of foreign bodies as occurring only in 3 per cent. of the total number of cases of appendicitis.

Signs and Symptoms.

(1) Onset of pain just over McBurney's point.
(2) Hyperæsthesia and muscular rigidity of the right rectus.
(3) Nausea and vomiting.
(4) Pyrexia; rigor seldom.
(5) Altered pulse rate.
(6) Constipation, almost amounting to obstruction.
(7) Right knee slightly drawn up.
(8) A tumor or swelling in the right iliac region, cylindrical, size of a finger, steadily enlarging and painful on pressure, and defined by rectal examination under anæsthetic.
(9) Thoracic and quickened respirations.
(10) General appearance much altered, face flushed or livid, nose sharply pinched, and tongue thickly furred.
(11) A rising leucocytosis.

Complications.

Adhesions between the tip of the appendix and bladder cause dysuria, and, to the rectum, cause proctitis. Careful examination is necessary to differentiate from renal lesions, especially calculi in kidney or ureter, often

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detected by the X-rays; from pneumonia, by thorough use of the stethoscope; and from typhoid fever, by the Widal agglutination test; from ovarian tumor, with twisted pedicle, or from movable kidney and torsion of the ureter and renal vessels, by vaginal or rectal examinations under an anæsthetic. Intestinal parasites are often present, especially the ascaris lumbricoides and thread worms.

In *children* acute thoracic disease may stimulate, or be masked by, abdominal symptoms. With renal calculi we generally have dysuria, hæmaturia, vomiting, and collapse. If the calculi are lower than the brim of the pelvis, there may be much difficulty in detecting them by the skiagraph. A peri-renal abscess may travel along and down the psoas muscle to the thigh or inguinal region, and it is well known that peri-appendicular suppuration can burrow up and behind the kidney, and even penetrate the pleura or the lung tissue.

Tenderness in the right iliac region is due to increased tension within the lumen of the appendix, or in the adjacent parts; and the disappearance of superficial tenderness, *without* other evidence of improvement—in aspect, pulse, temperature, and mobility of the rectus muscles—calls for immediate operation, as it so often follows perforation or rupture of the appendix (Bottomley, *The Practitioner*, June, 1905).

Appendicitis is very rare in infancy In 2,000 autopsies of children under 2 years old appendicitis was never once found (Holt). I have thrice seen it in children aged 3 years, and from this age, and up to 10 years, we have to distinguish it from simple colic, constipation, intussusception, psoas abscess, tubercular peritonitis, and pneumonia at the base of the lung, when a daily leucocyte count would assist.

Howard Kelly (1905) states that biliary calculi have never been found in the appendix. Many surgeons have mistaken certain facets on the fæcal concretions as evidences of gall-stones.

Chronic appendicitis has been mistaken for hip-disease by surgeons of excellent reputation and large experience, and Kelly records two graphic instances. It is obvious, therefore, that, in cases of possible appendicitis, one has to sit down at the bedside and cautiously eliminate many other lesions, some very obscure.

Lesions Likely to be Mistaken for Appendicitis.

(1) Tumor of the right ovary, or inflammation of the same.
(2) Extra-uterine pregnancy.
(3) Worms in the intestinal canal.
(4) Meckel's diverticulum, causing obstruction.
(5) Stone in right kidney or ureter.
(6) Movable right kidney and torsion of pedicle.
(7) Intestinal or omental adhesions (? tubercular).
(8) Calculi in biliary tract.
(9) Obstruction, due to impaction of fæces in cæcum.

Once the diagnosis is established, every clear case of appendicitis should be operated upon within the first twenty-four hours; in the meantime hot fomentations being constantly applied, as long as the skin does not become tender; an enema of hot oil and soapsuds administered—and I agree with those who do not give calomel, but rely on the castor oil draught. At the end of twenty-four hours, the indications for operation would be—

(1) Pains about the umbilicus or McBurney's point.
(2) Muscular rigidity in right iliac fossa.

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(3) Tenderness in right iliac fossa; the sudden diminution or actual cessation of pain in that region *without improvements* in other directions, is an indication for immediate operation, because so frequently preceded by perforation or rupture of appendix.

(4) Increase of the localised swelling.

(5) Continued nausea and vomiting.

(6) Constipation, the rectum being emptied by enema.

(7) Elevation of temperature and pulse.

(8) Rising leucocytosis.

(9) Sudden seizure, with pain in the abdomen, with or without collapse.

(10) Ileus, the gravest symptom, and admitting of no delay.

It is most important not to dawdle or to wait for the presence of relatives, guardians, or friends, not only in the patient's best interests, but, as has been well put—a surgeon is never so unhappy as when watching a case of appendicitis, and noting progress or retrogression when it has not been operated upon; and even the most desperate cases of suppurative appendicitis, with peritonitis, sometimes pull through.

RETRO-DEVIATIONS OF THE UTERUS.

By T. G. Wilson, M.D., Ch.M., Syd., F.R.C.S., Edin.

One of the most striking features in the recent literature on the subject of retro-deviations of the uterus is the number of writers who maintain that those symptoms, which we have hitherto been accustomed to look upon as being due to some backward displacement, are caused not by this displacement but by some existing complications. One writer (1) at the recent Pan-American Congress at Panama, maintains that there is no normal position for the uterus, and that, whatever the position of this organ, it is only on the supervention of some complication that symptoms manifest themselves. On the other hand, there are authorities who maintain that a retro-deviation is essentially a pathological condition, and requires treatment whether there are any symptoms directly referable to it or not. Thus, W. D. Haggard (2) Professor of Gynæcology at the University of Tennessee, in a recent address on this subject, says:—"To disregard a case of retro-deviation simply because it is giving rise to no symptoms at the time is comparable to disregarding many cases of renal and cardiac disease, gallstones, and errors of refraction, &c., which are often discovered by accident, and to assume that because they are causing no symptoms at the time that they will never do so . . that, inherently, the causes which produce the retro-deviation will also engender complications . . . . that there is always the element of infection to be considered in the female pelvis, and it is this element of infection that determines the presence or absence of complications, such as adhesions, etc."

Certainly when a uterus becomes fixed in its retro-deviated position we may consider that the fixation is an important element in the symptomatology, and, whichever standpoint we take, the question of whether the uterus is fixed or free is a most important factor from the point of view of treatment.

On going through the notes of 900 consecutive gynæcological cases seen recently, I find that the uterus was in a position of retro-deviation in 153 cases, or a frequency of 17 per cent. Of these 153 cases I have noted that in 39 cases the retro-deviation was a result of, or coincident with, some degree of prolapsus

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uteri secondary to pelvic floor weakening. Of the remaining 114 cases, in sixty-nine the retro-deviation was complicated, leaving forty-five cases, or 5 per cent. of the total number, in which the uterus was freely mobile and could be replaced in a forward position. I have noted the symptoms in these forty-five cases, with a view of determining whether they were to be considered as due to the retro-deviation or to complications present. In nineteen cases the symptoms the patients complained of were more directly referable to some accompanying condition—endometritis, retained decidua, &c.; while of the remaining twenty-six cases, in whom there was no other abnormal condition present except the retro-deviation, I found that in twenty-three cases the symptoms complained of were apparently directly referable to the retro-deviation itself, some at least being produced mechanically by the position of the uterus and adnexa.

In regard to such symptoms as menorrhagia, metrorrhagia, &c., while we must consider these as being directly referable to the condition of the endometrium, we must admit that, in the majority of cases, such complications cannot be effectively treated without at the same time rectifying the backward displacement of the uterus; and whether we are to consider a retro-deviation without symptoms as pathological or not, it would seem as if the patient's general health gets below par, and if any infective condition —endometritis, &c.—arises, the retro-deviation becomes an important factor in exaggerating and keeping up the condition, and therefore demands active treatment.

The treatment of those cases which are coincident with prolapsus uteri will naturally be from the point of view of the prolapse, and any method which aims at correcting the retro-deviation, without at the same time restoring the integrity of the pelvic floor, will be doomed to failure; and, for this reason, I purposely exclude such cases altogether in considering the treatment of retro-deviation proper. In regard to the treatment of mobile retro-deviations by mechanical means—*i.e.*, pessaries—I am quite aware that amongst many gynæcologists pessaries are considered a thing of the past, and, if the condition requires treatment at all, that operation is advised; but I cannot help thinking that there are certain cases where the pessary has a distinct sphere of usefulness, and should be the method of choice. I refer especially to cases of mobile retro-deviation occurring after confinement, in whom a well-fitting pessary will keep the uterus in a forward position, and help to prevent a long convalescence due to sub-involution. I have followed up enough of these cases to convince myself that, after a time, the pessary may be discarded without the retro-deviation recurring. This period after confinement has been aptly termed the "psychological period of the pessary."

With regard to the treatment of fixed retro-deviation by massage, after the combined vagino-abdominal method of Brandt, this method has never gained any foothold in English-speaking countries, and the objections to its use are fairly obvious, but it has been extensively practised in Continental hospitals; and while working in Professor Chrobak's clinic in Vienna I had some chance of seeing the method of procedure, and examining the patients before, during, and after the treatment. Naturally it is all-important that the cases should be carefully chosen, and when this is done it is quite certain that good results can be obtained. I have seen many cases with fixed retro-deviations who would certainly here be advised to have operative interference, in whom, after several sittings, the uterus has been got into forward position, and kept there temporarily by means of a pessary, and after a time it has been possible to discard the pessary without recurrence of the retro-deviation.

The operative treatment of retro-deviation presents a large scope, and the number of operations suggested is a fairly good index that no one of them

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is generally applicable. Indeed it is rare to go through a gynæcological journal without seeing some new operation, or modification of some old operation, designed for the cure of this condition. Probably the most usual and most favored operation at the present time is some form of ventro-suspension or ventro-fixation; and, as regards the after-result, the cases I have had a chance of following up would indicate that the distinction between these two operations depends chiefly on whether the ligatures used for the suspension or fixation are absorbable or not, as a ventro-fixation done with an absorbable material, as catgut, in the course of a few weeks becomes practically a ventro-suspension. Apparently tendon, either the formalin prepared or the ordinary biniodide tendon, is very often not absorbed, or at any rate for such a time as to make it justifiable to consider it as an unabsorbable material. I have frequently seen tendon, which has been buried in the abdominal cavity for periods up to two and even four years, which was apparently unaffected at the time of a second operation, and on cutting sections through such tendons little or no leucocytal infiltration was to be seen, and as regards tensile strength it did not seem to have suffered at all. Catgut is always absorbed quickly, so that probably the adhesions formed round an absorbable ligature like catgut will tend to be less dense than round an unabsorbable ligature like silk or tendon, round which the adhesions, being denser, will naturally persist longer and not tend to disappear while the foreign body remains. As a result of these operations the uterus is changed from a pelvic to an abdominal organ, for a time at least, and from being a freely mobile organ it is made into a more or less fixed one; and we have to consider what the result of this fixation will be should the patient become pregnant, and also whether the operation will permanently keep the uterus in its forward position. In considering this operation there are such a number of modifications of doing it that it becomes difficult to compare them. While some operators insert their sustaining sutures on the anterior surface of the uterus, others use the fundus; and others, again, following Kelly's original suggestion, insert the sutures on the posterior surface of the uterus, in order to allow the intra-abdominal pressure to act on that surface. Then some operators use unabsorbable sutures, silk, silkworm-gut, &c., and others use catgut always. There are very different views, too, as to the ultimate result of the operation. The question of what really does happen to the suspensory ligament after a ventro-suspension is an interesting one, and I have kept notes of cases I have examined at varying periods after this operation, and also of cases where it has been necessary to reopen the abdomen at a subsequent operation. In thirteen of these the second operation was done at the Adelaide Hospital, where I have assisted at the operation, and I am indebted to Dr. Hamilton for these cases. First, in those cases where the ventro-suspension was done with tendon or silk: I have notes of eight of these, six done with tendon and two with silk, and all of whom I have examined at periods from six months to five years after the first operation. In six of these cases the uterus was still in a forward position, and a definite suspensory ligament was palpable on bimanual examination. In three of these six cases the abdomen was reopened, and the actual condition of the ligament could be observed. Two of the ligaments were solid and firm, suggesting that they might cause trouble by preventing the expansion of the uterus during pregnancy. The third was much thinner, and stretched out to a length of 2 inches, and did not apparently take any active part in keeping the uterus forwards. In the other two cases no suspensory ligament could be felt, but the uterus was again in a position of retro-deviation. One of these patients had had a full-time child since the original operation, and the abdomen had to be again reopened on account of further adnexal inflammation. In this case also there was a thin,

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stretched-out, suspensory ligament, stretching from the old abdominal incision to the fundus uteri. So that in seven out of eight of these cases there was a suspensory ligament left as a result of the operation, though in two cases it had apparently stretched to such an extent as to cease to be of service in holding the uterus forwards.

These are the only cases I have been able to follow up which have been done with tendon or silk, as for the last four years most of the ventro-suspensions at the hospital have been done with catgut. I have notes of twenty-seven cases in whom a ventro-suspension was done with catgut, and whom I have examined at periods varying from six months to four years after this operation.

First, in regard to the position of the uterus. In eleven cases the uterus was in good position at the time of the second examination; in sixteen cases it was again retro-deviated. In none of these cases examined after six months was any suspensory ligament to be felt on bimanual examination. Of these twenty-seven cases the abdomen was reopened in seventeen of them, and the actual condition of the pelvis could be ascertained, and in none of these cases was any sign of a suspensory ligament discovered. The shortest period after the first operation that the abdomen was reopened was four months. Six of these cases had had the anterior aponeurosis included in the suspensory ligatures, so that the original operation was a fixation. In the ten cases which were not reopened it is impossible to say for certain whether any sign of a suspensory ligament would have been found, but it is certainly remarkable that in all the cases that were reopened there was no suspensory ligament found. In some of these cases the catgut used was formalin catgut, in others biniodide catgut, and in others iodine-prepared catgut. While I have recorded these results as I have found them, I naturally realise, from hearing of the experience of others, that sometimes a suspensory ligament does persist after a ventro-suspension done with catgut, and especially if there has been any suppuration in the abdominal wound. Taking these two series together, we get thirty-five cases who had had a ventro-suspension done, and at varying periods from six months to five years afterwards the uterus was found not to have retained its forward position in eighteen of them. While agreeing that it is not a fair criterion to take eighteen out of thirty-five cases, as during this period there were a great many other cases who did not report themselves, and who were, presumably, well, and also admitting that in some of these cases the retro-deviation had recurred as the result of a fresh inflammatory process, the fact of finding that the operation had not fulfilled its object in such a number of cases indicates that a ventro-suspension, as generally done, and especially when absorbable ligatures are used, cannot be regarded as really effective in permanently maintaining the forward position of the uterus.

The above results would indicate that a ventro-suspension done with catgut does not fix the uterus permanently to the abdominal wall, but merely holds it in a forward position for some months, and so gives the uterine supports time to regain their tone; while when an unabsorbable ligature is used the uterus, as a rule, is definitely suspended by a cicatricial ligament. It seems rather difficult to believe the statement we often see that such a ligament will stretch with pregnancy, and involute after delivery, and so still hold the uterus forwards. I have attended five cases in confinement who have had a ventro-suspension done, one with silk, one with tendon, and three with catgut, and in all these cases the confinement was easy. Four other cases who had had ventro-suspensions done, and who have had what they described as normal labors, are included in these thirty-five cases. Of these nine cases in only three was the uterus in a forward position after the confinement, which is instructive, as indicating the importance of parturition

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as a causative factor in retro-deviations, and also how unlikely it is that the suspensory ligament, if one should be present, will stand the strain of the growing uterus during pregnancy and still hold the uterus forwards after confinement.

It is generally admitted that a ventro-fixation proper is not a justifiable operation in a woman who is likely to have children, though even now one constantly sees accounts in the journals of cases where Cæsarean section has been necessary when such patients have gone to term. In regard to this, it is of interest to note that Andrews (3) in a recent review of the effects of ventro-fixation and ventro-suspension on subsequent pregnancy, has collected 395 cases of labor following these operations, and of these there were twenty cases where Cæsarean section was performed as a direct result of difficulties caused by the previous operation. Of these twenty cases fourteen followed ventro-fixation, and six followed some form of ventro-suspension. He came to the conclusion that the method which caused least difficulty with subsequent labor is a ventro-suspension, though he adds that this operation has the theoretical objection that the suspension may not be permanent. In a great number of these cases the original operation was done on account of retro-deviation accompanying prolapsus uteri.

While the position of the uterus after the removal of the ovaries seems to be of little consequence, if it is wished in such a case to put the uterus in a forward position there would appear to be no objection to a ventro-suspension done with catgut; but in those cases in which the abdomen is opened, and the ovaries not removed, what is to be done to correct a retro-deviation? If a suspension with catgut be done, judging from these cases, we can never tell exactly what will happen afterwards. If the uterus remains forward, well and good; but if not, can we ask the patient to have another suspension done every few years, or after each confinement? Or are we to use an unabsorbable ligature, and aim at having a firm suspensory ligament left, which may cause trouble with a subsequent pregnancy, or, if not, may stretch so as to cease to be effective as a sustaining ligament, and which will remain as a potential source of danger from giving rise to internal strangulations, &c.? That this is a real possibility we have only to read the accounts of such cases recorded by Jacobs (4), Leopold (5), Rufus B. Hall (6), Thomas (7), Lindtors (8), and many others. Is there no other method by which the uterus can be kept forwards without leaving a permanent source of danger in the abdominal cavity? The different methods that have been suggested by the vaginal route do not seem to have been very effective, though they all have their warm advocates. Shortening of the so-called utero-sacral ligaments, vaginal shortening of the round ligaments, Pryor's operation and vaginal fixation may be mentioned, though this last method is now generally considered as unjustifiable in a child-bearing woman. The abdominal route is at present the favorite one, at any rate when the condition is complicated; for, as a rule, it is these complications, and not the retro-deviation, that demand a section. For an uncomplicated case, if it requires surgical treatment, the Alexander-Adams operation is certainly the method of choice; and some operators (9) apparently prefer to finish up with this operation after having dealt with the pelvic condition through a median incision, though the extra element of the time required must be a disadvantage. Noble's suggestion of using a transverse incision, after the method of Pflannelstein, in these cases, and, after dealing with the pelvic condition, shortening the round ligaments extra-peritoneally from the extremities of the original incision, would seem to be the ideal operation, at any rate where a large abdominal incision is not required. The different methods of shortening the round ligaments intra-peritoneally—Wyllie's, Mann's, and Baldy's operations—

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while they all have the, to my mind, theoretical objection of leaving the weakest parts of the ligaments—the part in the inguinal canals—as one of the points of fixation, certainly do maintain the forward position of the uterus, and apparently do not interfere with subsequent pregnancy. Gilliam's operation, which has been warmly advocated by many American gynæcologists, is open to the objection that an opening is left on either side where a coil of gut may become strangulated, though it has been suggested that the parietal peritoneum may be sewn to the round ligament in order to obliterate these two spaces. All these operations are more or less on trial, and until the ultimate results as regards their permanency, and especially the effect of subsequent pregnancy, are more fully recorded, it is impossible to lay down any fast rule; but, granting that they do not cause interference with gestation and parturition, when surgical interference is adopted for the cure of a retrodeviation in a child-bearing woman the utilisation of the round ligaments to effect this certainly seems to be the most rational procedure.

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A RARE FORM OF PELVIC TUMOR—OPERATION—RECOVERY.

BY FRANK A. NYLUASY, M.B., CH.B., MELB.

My reason for reporting this case is that the condition—cyst of Gartner's duct—is such an exceedingly uncommon one. It is not even referred to by Howard Kelly, for instance. Bland Sutton is the only authority, so far as I am aware, who has described it. Herman and Jellett both mention the subject, but their accounts are evidently derived from Sutton's. I have myself never previously seen a case like it, nor have the medical men who assisted me at the operation.

The patient in the present instance is a young married woman, aged 27; slight in build, with a somewhat anxious expression. She has, for the past four years, complained of constant aching pains in the back, tenderness in the left ovarian region, severely painful scanty menstruation—worse since her marriage, four years ago—constipation, and occasional difficulty in urination.

On examination I found the cervix lying up under the pubic arch, and the narrow end of a fluctuating tumor dipping down behind the cervix, and in close contact with it. A distinct fulness in the right iliac fossa could be detected on palpation, but was not obvious on inspection, which probably accounted for the fact that the tumor was completely overlooked by previous medical attendants. I had the greatest difficulty in convincing the patient that she had anything so serious as a pelvic tumor: all sorts of reasons but the right one had been given to account for her symptoms. It was only

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after I called in the aid of another medical man, to make a conjoint examination under chloroform, that I succeeded in convincing her (almost completely) that she had a pelvic tumor at all. (She was really only fully convinced when she was shown the tumor after operation.) The fact that she had no startling symptoms, and that the tumor grew downwards into the pelvis, without producing obvious enlargement of the abdomen, and that previous medical attendants had asserted that there was nothing seriously wrong, explains the reason of her incredulity. After some weeks' consideration, however, she submitted to the operation. This was performed on February 17th, 1904.

Operation.—An incision, 3 inches long, was made in the midline below the umbilicus. A part of the cyst was seen lying snugly in the right iliac fossa. There was a little ascitic fluid surrounding the growth, and its peritoneal covering had a dark deeply-congested appearance. The fallopian

tube and upper part of the meso-salpinx were quite free from the cyst; the abdominal end of the tube was closed. The ovary lay on the anterior and outer part of the cyst, but was not attached to it anywhere. The cyst extended right across, behind the uterus, to the opposite side, and lay in contact with the left broad ligament, which was wrapped firmly round it. The left fallopian tube was dilated into a hydrosalpinx as large as small intestine; the corresponding ovary was dilated into a cyst the size of a small mandarin orange; and both were bound down by universal adhesions. The uterus was displaced forwards by the main cyst, and had also been pushed over to the left as the growth enlarged. [Even after the main cyst had been removed, the uterus retained its left-sided position until its multiple adhesions with the left broad ligament had been severed.] On cautiously passing the fingers behind the tumor, as far as they could safely reach, they dipped right down

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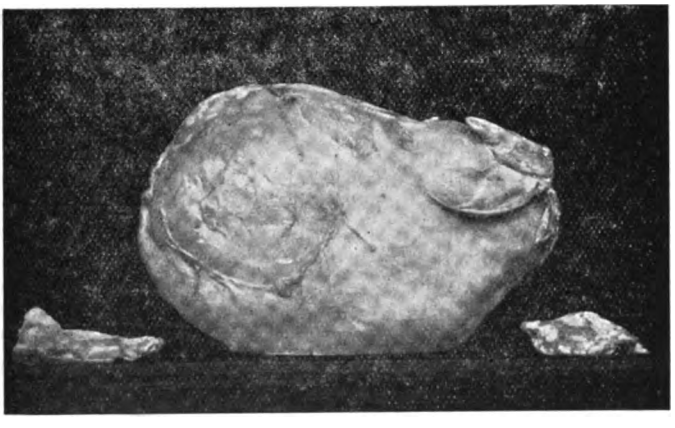
into the pelvis, where many bands of adhesions were felt crossing in an antero-posterior direction, between the pelvic and tumor walls. The tumor itself was situated between the walls of the mesometrium, and was everywhere densely adherent to it. The tumor, moreover, had lifted up the posterior wall of the peritoneum, and there effected some important attachments. The bladder, as far as could be felt at this stage of the operation, was free from the cyst. However, the absolute impossibility of releasing the growth, without previously emptying it of its fluid contents, was thus made clearly evident. A number of flat sponges were thereupon placed in the abdomen, so as to completely surround the cyst, from which two and a half pints of perfectly clear watery fluid were now evacuated by tapping. Then began a process of enucleation, the difficulty of which can probably be best appreciated by those who have had experience in the removal of an adherent cyst that has burrowed deeply into the pelvis between the layers of the broad ligament (and this growth had extended down to the cervix uteri, as felt per vaginam). The collapsed cyst was drawn up as far as possible, and the process of separation began at its outer side, and was continued towards the front. Many of the adhesions on the anterior part of the cyst were so dense that they could not be torn through with the fingers, but had to be cut with scissors.



The right ureter was flattened out into a broad band, firmly attached to the base of the cyst wall, the peritoneum having been lifted off the ureter by the growing cyst. The ureter was separated from the cyst by the aid of dissecting forceps and scissors. This being accomplished, the enucleation of the rest of the cyst was comparatively easy; but, at best, such an operation tries one's patience greatly. Fortunately, hæmorrhage was not severe, so that it was unnecessary to ligature the infundibulo-pelvic ligament. As soon as the cyst was removed, attention was directed to the other side. It was difficult to get hold of the ovary here, owing to the adhesions and displacement of the uterus. This ovary was at length reached, and was found to be about three times its normal size: it burst during removal, discharging clear fluid. The same thing happened with the hydrosalpinx, owing to the density of the adhesions and the difficulty of their separation, but the clear fluid was caught by the surrounding sponges. The fallopian tube, which formed this hydrosalpinx, was dilated to quite the size of small intestine, and was at least 6 inches long, so that its separation was also tedious and troublesome. Ligatures were placed on both ends of the corresponding broad ligament. The

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patient was now left with one ovary and one fallopian tube—on the right side—but, unfortunately, this tube had been sealed up by inflammation, so that all chance of the patient realising her hopes of future pregnancy was out of the question, unless the closed condition of the tube could be rectified. The abdominal end of the fallopian tube was, therefore, slit up, and a few silk sutures put in to keep it open. The patient has now a possible chance of one day becoming a mother.

With all the manipulations required to separate these very adherent growths, it was a little difficult to decide whether drainage should be adopted or not. Having made sure that there was no bleeding into the pelvis, and feeling convinced that there was nothing septic to be feared, either from the growths or the sponges, instruments and ligatures, it was decided to dispense with drainage altogether, and the wound was, therefore, closed in three layers and dressed with iodoform gauze and a flannel roller bandage.

I may remark, in passing, that it is quite unnecessary to suture the separated walls of the broad ligament, as recommended by Howard Kelly, Dudley, and many others, inasmuch as these walls fall together nicely after the removal of the contained cyst, and there is no great amount of oozing between them. As Bland Sutton states, "Enucleation is usually accompanied by more loss of blood than simple ovariotomy, and the prolonged manipulation is often responsible for severe shock."

Although this tedious operation, lasting an hour and three-quarters, was done at the patient's own house, on one of the hottest and most oppressive days of the year, she did not suffer much from shock, and she had little or no vomiting.

In speaking of Gartnerian cysts, Bland Sutton further remarks, in his work on the "Diseases of Women," that "cysts of this character, which burrow deeply, often entail risk in removal, as they lie in intimate relation with uterus, ureter, and bladder. The cyst, when large, will come in contact with the iliac arteries and veins at the brim of the pelvis, and even rest upon the inferior vena cava. Gartnerian cysts arising in the terminal segment of the duct project into the vagina. In some instances these cysts may be treated surgically through the vagina with greater success than by cœliotomy." For my part, I should like to know what those instances are, and how to differentiate them. In the meantime, and after the experience of my own case, I should view the advice to operate on Gartnerian cysts through the vagina as likely to lead to the most disastrous results.

After the operation I saw the patient at six o'clock in the evening, when she was complaining of a good deal of pain, as was only natural after the separation of so many adhesions. I therefore varied my usual rule of withholding morphia, and gave her her first and only dose, of one-quarter of a grain, hypodermically. But she was otherwise remarkably well, and, in fact, made the easiest recovery I have ever seen, either in my own practice or in that of others. She had one rise of temperature to 100° on the day following the operation, and thereafter everything was normal. She never required a catheter, and menstruation came on at the expected time—two days after operation—without pain. She vomited twice slightly after the anæsthetic. The bowels acted freely after an enema on the third day. She was given sips of hot or cold water whenever she desired, as I altogether disapprove of the quite unnecessary practice, initiated by Martin, of absolutely withholding drinks shortly after abdominal operations. This practice is cruel in the extreme, and I agree with Professor Mummery that it has absolutely nothing to justify it. In fact, the more I see of these operations the more convinced I am of the harmfulness of a rigid adherence to what Treeves calls "the abdominal ritual."

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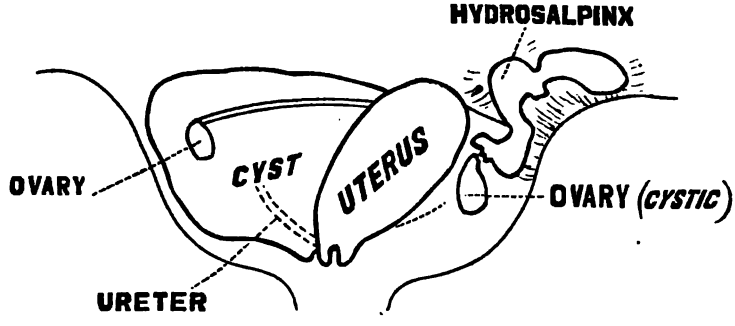
My patient was allowed to get up at the end of the third week, the external wound having united perfectly; and she has not only remained quite well, but has greatly improved in health since the operation, eighteen months ago.

I must here express my thanks to Dr. Rennie for his able assistance at the time, and to Dr. Trood for his skilful administration of the anæsthetic—chloroform, followed by ether.

Appended is a rough sketch of the growth *in situ*, and a photograph of the same after its removal. The main cyst measured 8 inches by 5. The shaded photo. exhibits some of the numerous adhesions very clearly. The peculiar tube-like prolongation at one end is also seen, but it appears much shorter in the photograph than it really is. The remains of the removed ovary, and one-half of the previously dilated but now shrunken fallopian tube, are also depicted.

The rarity of the affection is my excuse for reporting it at length.



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SECTION OF EYE, EAR, AND THROAT.

EYE DIVISION.

INTRODUCTORY ADDRESS.

By W. Odillo Maher, M.D., Ch.M., Dub., M.R.C.S.

President of the Ophthalmological Section.

Gentlemen—My first duty is to express my appreciation of the marked distinction conferred upon me by the members of the Executive Committee of the Australasian Medical Congress in electing me to the position of President of the Section of Ophthalmology, and to thank them for what I consider to be one of the highest honors they had in their power to bestow. The invitation came to me as a surprise, and my first inclination was to decline, fearing I should not be able to adequately discharge the duties of this office. On reflection, however, I deemed it my duty to accept, for I felt that the compliment was intended, not only for me personally, but also for the State to which I belong.

The first difficulty which presented itself was the selection of a subject for an address worthy of your attention. After much consideration, it occurred to me that, as it was nearly a quarter of a century since I first commenced the study of Ophthalmology, a brief sketch of some of the principal advances that have taken place within my own observation would not be wholly devoid of interest.

It was in the year 1881, when I was appointed Clinical Assistant to the Royal London Ophthalmic Hospital, Moorfields, that I commenced the study of diseases of the eye. At that time the consulting staff of the Moorfields Hospital consisted of Drs. Critchett, Bowman, Dixon, and Hutchinson; and the visiting staff of Drs. Wordsworth, Streatfield, Hulke, Lawson, Adams, Lyell, Cooper, Tweedy, and Warren Tay. Of the former only one, Mr. Jonathan Hutchinson, and of the latter only the last three mentioned, survive. Then, too, it was my good fortune to meet our courteous and energetic Secretary, Dr. Symons, with whom I was intimately associated for two years—first as Clinical Assistant, and afterwards as House Surgeon.

Looking back, one cannot but be deeply impressed by the vast amount of research and useful original work performed by many arduous and earnest workers in this department of medical science. Still we must all feel how much remains to be accomplished; how little we can do to arrest the progress of many of the cases of optic nerve atrophy, how ineffectual is our treatment of some of the diseases of the eye—as, for instance, detachment of the retina—and how limited is our knowledge of the direct causes of many of the changes met with in the fundus oculi.

Owing to the varied details, and to the limited time at disposal, it would be impossible to give more than a brief—and, I fear, a somewhat disjointed—retrospect. I therefore propose confining myself to some of the advances I consider most noteworthy.

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I well remember the first demonstration of retinoscopy at Moorfields. It was given by a former House Surgeon, who had just returned from a trip to the Continent. Its simplicity, approximate accuracy, and its advantage as an objective test were at once appreciated, and within a very short time it was adopted by all. Retinoscopy was first suggested in 1874 by Cuignet, who called it keratoscopy. Bowman, however, about ten years before this, drew attention to the possibility of diagnosing astigmatism by means of the ophthalmoscopic mirror; but, unfortunately, he did not follow the subject up. Retinoscopy affords us an invaluable means of estimating the refraction of children, and of those who are mentally deficient, or whose powers of observation are of a low order. By this means the refraction of the whole of the media can be estimated. In this respect it differs from, and is far superior to, keratometry, another objective method of estimating refraction to which much attention has been given of recent years. The most improved keratometer, or ophthalmometer, is probably that of Javal Schiotz. It is an excellent instrument for determining the amount of corneal astigmatism, and the axes of the greatest and least corneal curvatures; but, beyond this, affords no information.

During the period under review our views as to the treatment of myopia have undergone a material change. Formerly it was unusual to prescribe the full correction for constant use, and it was thought that the strain of the ciliary muscle during the effort of accommodation produced an increase of the myopia with its deleterious effects; now it is believed to be the convergence of the optical axes which plays the most important part in causing this increase; and many cases have been recorded which show that, when the full correction has been worn constantly for all purposes for a number of years, there has been no increase in the myopia in the majority of instances, while in many the increase has been slight, and in but few has it been marked. Hence the practice nowadays to order, when the eyes are healthy, the full correction for constant use except in very high degrees, and if the myopia increases, to increase proportionately the strength of the glasses. In myopia of twelve dioptres and more the removal of the crystalline lens, when successfully performed, improves the vision immensely. This operation was first described and recommended by Desmonceaux in 1776, and later, in 1817, advocated by Beer; but it remained for Fukala to bring it prominently under the notice of the profession. Many cases of detachment of the retina following this operation have been recorded; still the advantages derived by the majority of patients are so great that there can be little doubt but that this operation will long continue to hold a place in ophthalmic surgery.

Errors of refraction as a cause of headache are now more generally recognised by physicians than formerly, and, consequently, such cases are more frequently referred to ophthalmic surgeons.

Comparatively recently Helmholtz's theory of accommodation has been questioned by Tscherning, who maintains that during accommodation the anterior surface of the lens, instead of becoming spheroidal, assumes a more or less conical shape. This he considers is caused by a tightening of the zonula. Karl Grossman more recently has had an opportunity, in a case of aniridia with small opacities on the anterior and posterior poles of the lens, of studying the changes which take place during accommodation, and his observations confirm Helmholtz's view as to the relaxation of the zonula; but also show that the anterior surface of the lens, instead of becoming spheroidal, becomes conical at the centre, and flattened towards the periphery, as stated by Tscherning. Grossman also states that a slight posterior lenticonus is developed during accommodation. It is difficult to understand

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how a relaxation of the zonula could result in these conical changes in the surfaces of the lens, unless, as suggested by Priestly Smith, there is an arrangement of lens fibres causing resistance to be progressively greater towards the periphery of the lens.

In referring to some of the very valuable drugs now in general use in ophthalmic surgery, and which were for the most part unknown twenty-five years ago, cocaine is deserving of first mention. In the year 1884 Carl Koller, at the Heidelberg Congress, read his memorable paper on Cocaine, and demonstrated its marvellous power of producing local anæsthesia. This wonderful discovery marked an eventful epoch in the history of ophthalmic surgery. Cocaine has facilitated enormously the performance of most eye operations, and has saved patients an incalculable amount of inconvenience and pain. Several other excellent local anæsthetics have since been discovered, and of these I shall mention only eucaine and holocaine. The latter, a synthetic product, is probably the best local anæsthetic known. It is an antiseptic; produces a deeper anæsthesia of the iris than cocaine: does not affect either the pupil or accommodation; causes little, if any, dryness of the corneal epithelium; and does not produce any toxic effects unless introduced sub-conjunctivally. Euphthalmine is probably the best mydriatic to use to dilate the pupil for the purposes of ophthalmoscopic examinations, as it does not affect the accommodation, or only to a slight degree, and the mydriasis, which is very marked, passes off in a few hours. Homatropine, whose action is also transitory, is not only a mydriatic but also an excellent cycloplegic, and, combined with cocaine, is very generally used to paralyse the accommodation before estimating errors of refraction in adults. The cycloplegia, however, is not so complete as that produced by atropine, which is the best drug to use to paralyse the accommodation of children.

The stypic properties of suprarenal extract have proved a great advantage in many of the minor operations on the eye, and it is said to be useful also in the treatment of certain eye diseases. Of late attention has been directed to the use of the organic silver compounds in the treatment of conjunctival affections, especially in the treatment of purulent and gonorrhœal ophthalmia, and also in the treatment of chronic dacryocystitis. The advantages which are claimed for them over the inorganic salts are that they are less irritating, less coagulating, less caustic, and more penetrating; and at the same time, they are strongly bactericidal in their action. The organic preparations of copper and mercury are also highly spoken of. Another drug to which I shall briefly refer is fluorescin, which beautifully maps out with its green and yellow staining corneal abrasions and ulcers. The Rontgen rays are of great value in ophthalmic surgery: they not only enable us to diagnose the presence of foreign bodies in eyes, but also materially aid in locating their positions. These rays have also been successfully employed in the treatment of lupus and epithelioma, and cases of trachoma have been recorded as cured by them.

I now propose to briefly review some of the more recent operations in ophthalmic surgery.

H. Knapp, of New York, in 1892, drew attention to the marked benefit derived by expressing trachoma by means of a roller forceps. In many cases the improvement following expression far exceeds that obtained by any other mode of treatment. Previous to the introduction of the roller forceps, granulations were often expressed by crushing the lids between the thumb nails, but the expression of the trachomatous substances by this means was very imperfect, and the results were often unsatisfactory.

The simple extraction of senile cataract has been revived, and warmly advocated by many distinguished ophthalmic surgeons. The most serious

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drawback to this operation is the tendency of the iris to prolapse; but, when this does not take place, the results are most satisfactory, and the cosmetic effect all that could be desired. Formerly it was the practice not to operate on senile cataracts until they were mature; now, when a patient becomes incapacitated by senile cataracts, one does not hesitate to extract, washing out, if necessary, the soft matter, as was first suggested by Professor Horner of Zurich, in 1881, and more prominently brought under notice some years later by the late Dr. McKeown of Belfast; or, to artificially ripen the immature cataract by Forster's method, and extract a few weeks later. Recently the extraction of senile cataract in its capsule has been advocated by Major Smith, who has recorded excellent results.

Berlin, in 1868, reported a number of cases of extirpation of the lachrymal sac for dacryocystitis. This paper attracted little attention at the time. but the operation has since been revived, and, for the treatment of chronic cases—especially among the laboring classes—is a distinct advance. The epiphora which follows is surprisingly little, but should it be such as to cause any inconvenience, it can be readily cured by the removal of portion of the lachrymal gland.

About twenty years ago Mules described the operation which bears his name. After a few years it fell into disuse, owing to the large proportion of glass balls, which failed to be retained. Later, with the advance of our knowledge of asepsis and with improved technique, it again came into favor; and my experience is that it seldom ever fails. The great improvement in the appearance and in the mobility of the artificial eye, after a Mules' operation, is such that in my opinion in all suitable cases it is to be preferred to enucleation. A few cases of sympathetic ophthalmia following it have been recorded, and the probability is that there is a slightly greater risk of sympathetic after this operation than after enucleation. I have never had the misfortune to see such a case, although I have seen several following enucleation. In order to minimise the risk of sympathetic, and still retain the advantages of Mules' operation, the glass ball has been enclosed in Tenon's capsule, but the results have not been satisfactory. As a substitute for the glass balls, balls of gold, silver, and paraffin have been used.

Regarding strabismus the operative treatment is less in vogue than formerly, and advancement continues to grow in favor as compared with tenotomy.

The galvano cautery is now much more generally used in the treatment of conical cornea and corneal ulceration, especially in that variety which is associated with hypopyon; and its use in the treatment of corneal ulcers is much facilitated by first staining them with fluorescin. Sub-conjunctival injections are also highly spoken of by some authorities as treatment for hypopyon ulcers: they are also employed in the treatment of other diseases of the eye. Donders was the first to call attention to their action.

The removal of the superior cervical sympathetic ganglion for the relief of glaucoma was first performed by Jonnesco, in 1897. Since then it has been tried in a large number of cases, but the results cannot be said to be encouraging. In chronic and hæmorrhagic glaucoma it has been followed by temporary good results, but in cases of acute and sub-acute glaucoma the benefits derived from this operation, if any, are not to be compared with those obtained by iridectomy. Sympathectomy is suitable only in cases of hæmorrhagic glaucoma, or when a patient will not submit to an operation on the eye. The ciliary ganglion has also been removed for glaucoma, but the results were disappointing. This ganglion can easily be got at, by means of an operation devised in 1887 by Professor Kronlein, of Zurich, for the removal

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of orbital tumors and foreign bodies within the orbit without sacrificing the eye. Briefly, it consists of temporarily turning back the outer wall of the orbit, and thus exposing the orbital contents. Several brilliant surgical triumphs have been achieved by means of this operation: notably one, in which a tumor was removed from the optic nerve by Dr. Antill Pockley a few years ago, the patient retaining excellent vision.

Of the many useful instruments invented within my own knowledge, it is my intention to mention only a few, namely, Morton's ophthalmoscope, Priestly Smith's perimeter, Haab's electro-magnet, Prince's forceps, Knapp's roller forceps and cutting needles, Lang's twins, Ayers' chalazion forceps, Maddox glass rods, the Geneva lens measure, and Javal Schiotz's ophthalmometer.

Thanks to Listerism and the subsequent development of asepsis and antisepsis, the results in ophthalmic surgery have materially improved. Panophthalmitis and severe iridocyclitis, leading to phthisis bulbi following operations, are practically things of the past. In the early days of my practice I can well remember the great dread one had of these complications; but now one approaches operations on the eye with greatest confidence, feeling assured of a happy result in almost every case. Following the advent of Listerism came the study of the bacteria, several of which—the Kock-Weeks bacillus, the diphtheria bacillus, the tubercle bacillus, the xerosis bacillus, the diplo-bacillus of Morax-Axenfeld, the gonococcus, the pneumococcus, the streptococcus, the staphylococcus, and a few others, are found associated with eye diseases. Much yet remains to be discovered before we can claim to have a definite knowledge of the exact relation of many of these micro-organisms to the different diseases of the eye, and the fact that the infections are so often mixed adds to the difficulty. According to the present state of our knowledge it would appear that the Kock-Weeks bacillus is the most common cause of acute contagious conjunctivitis, the diplo-bacillus of Morax-Axenfeld of one of the commonest varieties of chronic catarrhal conjunctivitis, the diphtheria and tubercle bacilli are the causes of diphtheritic and tubercular affections, the gonococcus of gonorrhœal ophthalmia, and the pneumococcus of hypopyon keratitis and pneumococcic conjunctivitis. The xerosis bacillus—which, morphologically, so closely resembles the diphtheria bacillus—is frequently found in the normal conjunctiva and in far greater numbers in xerosis, of which, however, it is not considered to be the cause. Streptococci, on the other hand, are said not to be found in the normal conjunctiva: they occur in two varieties of conjunctivitis—a simple catarrhal and a pseudomembranous form, while staphylococci are found in the normal conjunctival sac, but probably do not produce any inflammatory condition.

Before concluding I desire to pay homage to the memory of those heroes of our science who, during the past quarter of a century, have passed away, leaving behind them works which will for ever be revered. The names of Graefe, Donders, Arlt, Helmholtz, Bowman, Critchett, Desmarres, Sichel. Jager, and of the other great men who have laid the foundation of the proud edifice of modern ophthalmology, will always remain intimately associated with its history; and we, as oculists, must deeply feel how great is the debt of gratitude we owe them.

Let me thank you for the patient hearing you have accorded me. I am well aware of the many imperfections of my retrospect, and of its manifold omissions, but I feel I would not be justified in occupying more of your valuable time. And in conclusion, gentlemen, permit me to express the hope that reviewing the past may stimulate us to greater efforts in the future.

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A DISCUSSION ON THE PRESENT DAY TREATMENT OF TRACHOMA.

INTRODUCED BY F. ANTILL POCKLEY, M.B., C.M., M.R.C.S.

When invited by the President of the Section to open a discussion on some ophthalmic subject, it was only after some hesitation that I accepted, because of the importance of the task, the difficulty in selecting a subject, and my inability to do it justice. Relieved, however, by the reflection that discussion implies difference—or, at least, modification or amplification—and that the introducer may be regarded merely as one who tosses the ball into the arena, leaving it to others to keep it moving, or as the advance guard that opens an engagement and then retires under cover of the heavy artillery, I accepted the honor—though still with misgivings.

The initial difficulty of the choice of a subject was met by the consideration that it should be one of wide-spread interest, of which all the members should have experience and could speak with benefit to us all. From this point of view it appeared to me that our present position in regard to the treatment of trachoma would be a useful matter for discussion.

I do not propose to go over the wide field of the treatment of trachoma generally, but merely to elicit the opinions of those present as to the comparative merits of the older and the more recent methods. Neither is it my intention to touch on the treatment of the numerous complications and sequelæ of the disease. All this would furnish enough controversial material for a week's discussion.

It seems fitting to take, as an arbitrary dividing line, the period of the second Intercolonial Congress held in Melbourne, 1889, when, in excellent papers by two ophthalmic surgeons of ripe experience—Dr. Aubrey Bowen and Dr. R. B. Duncan—we had given us what we may take as the recognised best methods of treatment at that time. I would inquire whether our present methods give speedier, better, or more lasting results than those then in vogue, or whether we have to confess that trachoma still remains as formidable and intractable a disease as ever? At that time treatment was mostly by local application (medicinal), and practically all the external remedial agents in the Pharmacopœia had been tried (and some not in it—such as inoculation with gonorrhœal pus), with the result that, as topical applications, silver nitrate and copper sulphate—the remedies of the ancients—remained our best friends. Perchloride of mercury, which was just coming into fashion, was going to do great things. Has it justified those expectations, or have we found that its usefulness, and even its safety, can be challenged? Our friend, Dr. Symons, was, I think, then using ethylate of soda: does he still pin his faith to it, or has he found anything better? In the sixteen years that have elapsed we have had new medicinal agents galore put into our hands, such as the organic salts of silver (protargol, argentamine, and argyrol), adrenalin and hemicine, cuprocitrol, ichthyol, various naphthol compounds, and many others (not forgetting "mud eels' blood"). The mechanical and surgical methods of treatment have come more into favor: and we have also to learn the results of our experiences with X-ray, high frequency, and radium treatment.

Let us consider first some of these medicinal agents, and see whether in them we have anything better or more reliable than the time-honored (if somewhat barbarous) silver nitrate and bluestone. The most satisfactory of these newer reagents appear to be the organic silver salts, and especially argyrol. To what extent have they realised expectations? They certainly are useful in some conjunctival and lachrymal sac troubles; and in trachoma also they

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often do good. In all but the more severe cases of trachoma, and in cases that improve slowly or hang fire under other treatment and call for a change, they may be used with advantage; and in such their painlessness renders them specially suitable for children and nervous or sensitive patients. Though I have used them for years, however, personally I have not the same confidence in them in severe cases of trachoma that I have in nitrate of silver, bluestone, or lapis divinis. I find also that patients themselves feel that the latter do them more good, and prefer them in spite of their painfulness. Neither (as personal experience has shown me) are the organic salts free from the risk of staining the conjunctiva (though I have seen it stated that they are safe in this respect): for this reason it is unwise to give patients these preparations to take away for their own use. Another drug that for a time had a certain (though I think over-rated) reputation is ichthyol, or ichthargin. It never appeared to me to do much good, though it has a place for a change, especially when silver salts are beginning to stain. Adrenalin and its congeners greatly impress the patients by their effect of bleaching (though only temporarily) the conjunctiva, and this seems to be their only virtue.

Though the continued use of weak lotions of perchloride or biniodide of mercury appears to be of no particular advantage, and may be injurious to the cornea, an occasional swabbing with 1 to 500 perchloride in glycerine, with or without previous mild grattage, is, I think, undoubtedly useful.

Some years ago I tried the effect of nascent iodine. I had a clamp made, like a Snellen's lid clamp, but with both blades of solid metal. To the surface of the inner blade was stitched a piece of lint soaked in a saturated solution of iodide of potassium. A current of two or three milliamperes was passed through it for a few minutes. It has a remarkable immediate effect on the sago grain granulations, making them white and friable, so that they either loosen and fall out or disintegrate and disappear; it also causes a fair amount of general inflammatory reaction. I consider it a valuable method of dealing with cases in which the sago-like bodies are numerous. One objection to it is that it is painful. Recently Roselli has used nascent iodine, which he obtains by the simultaneous application of potassium iodide and peroxide of hydrogen.

Cuprocitrol, in the form of a 5 per cent. ointment, appears to be another useful introduction, and may be used without apparent injury by the patient himself; but, as I have been using it for a few months only, I cannot speak with any certainty.

Mechanical and Surgical Treatment.—Though the methods employed to-day are, with some modifications, as old as the time of Hippocrates, it is only quite recently that they have been revived. The modern plan of expression by means of the forceps of Knapp, Grady, or Kuhnt, is, in suitable cases and in experienced hands, one of the most valuable methods we have of shortening the treatment; though I do not see the immediate cures recorded by Knapp. Possibly this may be because of a difference in the Australian type of the disease. This method is chiefly indicated where there are large, soft, or "ripe" sago bodies, without much previous inflammation or thickening, or when the inflammation and discharge have subsided. In most other conditions it does harm. Simple as the operation is, however, it must be done with judgment and caution, for one frequently sees disastrous results, in the form of extensive laceration and deep scarring of the conjunctiva and partial symblepharon caused by a too vigorous use of the forceps by inexperienced operators. In some cases a light subsequent brushing with a tooth brush and boracic acid adds to the efficacy of this method. Where the tougher papillary condition prevails, light scraping with a sharp spoon expedites matters. Scarification by numerous longitudinal incisions, often

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recommended, appears to me to be fraught with danger and apt to be followed by too much scarring; though a few incisions when the tissues are greatly congested and engorged gives relief by local depletion.

Excision of the Retrotarsal Fold.—This is an operation believed in and practised by Dr. T. K. Hamilton when most of us regarded it with suspicion, but latterly it has come into more general favor, and is, I consider, a most useful operation in suitable cases where the retrotarsal fold is exuberant. It should be carefully done, avoiding excising the sub-conjunctival tissue or the ocular conjunctiva. A good plan is to raise the conjunctiva on a thread passed longitudinally along the fold, or on two threads passed through either end of the fold. There will be contraction afterwards, of course, but not more than (if as much as) results eventually from a long and tedious treatment by local applications. Suitable cases for this operation, however, do not often come my way. It appears to be a feature of the Australian type of the disease (as was first suggested to my notice by Dr. Gordon McLeod) that the retrotarsal fold is not so prominently affected as it appears to be in the European form. Of the combined excisions of the fornix and tarsus, at present popular in Germany, I have no experience, and hope to hear something about it from some of those present who may have practised it or seen the results.

X-Ray Treatment.—As was to be expected, trachoma has been submitted to this, and many cures have been recorded by Mayou and Sydney Stephenson, in England, and by others on the Continent and in America. I have used it in both hospital and private work, choosing for purposes of comparison cases where both eyes were equally affected, and using the X-rays on one, and bluestone, silver nitrate, &c., on the other eye. I cannot say that I ever saw it do more good in active trachoma than can be more readily effected by more ordinary and simpler methods: in some cases it made matters worse. It has, however, appeared to be useful in reducing the hyperplastic and cicatricial condition found in old chronic cases.

High Frequency Currents.—My experience of these is too slight and too recent to be of any value. Of treatment by radium I have had no experience whatever, nor have I employed the method of "massage."

To sum up briefly, of local applications I should say silver nitrate and copper sulphate still hold pride of place, such as it is; the former to be used in moist discharging conditions, and the latter in the hard, dry, less vascular cases. Save in exceptional cases the nitrate should not be employed stronger than 10 grains to the ounce, and the copper sulphate should be applied lightly and evenly, and well into the fornices; the object being to excite a moderate reactionary inflammation, and not to cauterise the conjunctiva. Argyrol is a very efficient substitute, in the milder cases especially. As variants, swabbing with 1 to 500 perchloride tannin ointment or glycerine of tannin, or ichthyol 10 per cent. solution, or cuprocitrol ointment, may be used with benefit; nascent iodine is also distinctly useful. For the patient's own use a simple lotion of boracic, or borax, or hazelene, or weak sulphate of zinc, is as good as anything else.

Of mechanical and surgical methods, expression, in suitable cases, and when properly performed, is most useful; and so is excision of the retrotarsal fold in suitable cases. It is in the surgical treatment of trachoma that the most beneficial advance has been made in recent years.

X-Ray treatment, high frequency current, and radium emanations are only on trial, and appear of very minor value.

Whatever routine treatment we adopt, its usefulness in long-continued cases is increased by temporary intermission, at suitable times and for suitable periods, of all local treatment except simple lotions and ointments; and of more importance than we are apt to keep in mind is the improvement, as far

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as possible, of the patient's general health, and the establishment, as far as practicable, of better hygienic conditions—especially good food, fresh air, cleanliness of surroundings, and change of air or residence.

Finally, we have all seen cases of what has evidently been severe trachoma that have got well after lasting for years (as far as such cases ever do get well—that is to say, with a pale, smooth, glistening conjunctiva, delicate cicatricial lines, and normal shape of lids), when the patient has had absolutely no treatment whatever. The lesson of this is not to be unnecessarily energetic in our methods, but to hasten slowly, using the mildest effective methods when the patient has the requisite time and patience and confidence in his attendant. Unfortunately few patients can afford the time, or expense, or loss of employment entailed by long treatment; and there is, moreover, the risk that the longer the disease exists the greater the liability of its being communicated to others. There is no disease in the whole round of ophthalmic work that demands so much patience, experience, and resourcefulness on the part of the surgeon in its treatment as does trachoma, with its numerous complications and sequelæ, and we have, doubtless, all prayed for the discovery of the trachoma coccus and its antitoxin. But that day is not yet.

Dr. Barrett (Victoria) said that it seemed to him that the discussion had assumed too limited a form. The weakness of the position was that we did not clearly understand the cause, or, in general, the pathology of trachoma, and we were simply discussing the effect of a number of remedies applied to the surface of the conjunctiva in a purely empirical way. When one recollected that the lid was enormously thickened and infiltrated, and that the immediate effect of a remedy applied to the conjunctiva could only be for the moment to the depth of a fraction of a millimetre, the crudity of the proceeding appeared tolerably obvious. So far as the merits of the various local applications were concerned, he must confess that nothing gave him more uniform results than the free expression with Grady's forceps, followed by the alternate use of sulphate of copper and 1 per cent. cyanide of mercury solution, with occasional alternations to preparations of silver. With Dr. Orr, he had been for some time engaged in collecting evidence as to the conditions under which trachoma seemed to take rise in Victoria, and, though the work was far from complete, they had found that on the whole the trachoma came from the dry, hot, and dusty districts of the State. Very few cases came from the fertile western district. He understood that it had recently been discovered that the anthropoid apes could be inoculated with syphilis. Possibly they could be inoculated with trachoma also, and, if that were once done, the pathology of the disease would probably become well known.

Dr. Lockhart Gibson (Brisbane) said he expressed in suitable cases, but not in all. He used a 5 per cent. solution of nitrate of silver, carefully protecting the cornea, and neutralising it almost at once. Corrosive sublimate, of the strength of 1 to 5,000, when poured very freely into the conjunctival sac, was found by him to be a very useful mode of treatment. It was impossible to be sure of a cure until a case had been well for, say, six months.

Dr. Hogg (Tasmania) remarked that cases of trachoma in patients who had never lived out of Tasmania were extremely rare, most of the cases which he saw in Tasmanians having been acquired in the other colonies; and he had no doubt that hygienic and climatic conditions had a distinct bearing upon the disease. Expression was of great value. He thought too much had been made of the dangers of incision. In some bad cases he practised incision with grattage and brossage with mercuric chloride or cyanide,

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1 to 500: this shortened the period of treatment, even if it were not sufficient in itself. He had no doubt that relapses occurred, and that patients should be advised to report themselves from time to time.

Dr. Symons (Adelaide), in reply to a question, said he still had faith in ethylate of soda for cases where the granulations were fleshy and vascular, and especially if pain were a marked symptom. Of late ethylate of soda had been set aside in place of more recent drugs—as protargol, cuprol, &c. Dr. Pockley has not mentioned acetate of lead in the treatment of trachoma. He had recently treated a case throughout the whole course of the disease, the duration of which was less than six months; a result which he considered good, though he could not compare it with the results of cure in three months obtained by Dr. Barrett.

Dr. Gault, Dr. Kent Hughes, and Dr. Webster also spoke.

Dr. Antill Pockley, in reply, said that one noteworthy point brought out by the discussion was the existence of very different opinions as to the seriousness of trachoma; and it came as a surprise to him to hear ophthalmic surgeons say they regarded trachoma with complacency, and could nearly always guarantee a cure in about three months. His experience of eighteen years, as ophthalmic surgeon to the Prince Alfred Hospital, led him to believe that in most cases of trachoma one could never "guarantee" a cure, and in many cases it was impossible for any treatment to cure. Could it be maintained that cases such as this: chronic trachoma of long standing, with infiltration, thickening and drooping of the lids, perhaps contraction, entropium, and pannus could be guaranteed a cure in three months? He could say that cases had been coming to the Prince Alfred Hospital on and off for many years, and, in the intervals, having a turn of treatment at other hospitals, and still were not cured. The explanation of the difference in opinion probably lay in the fact that some surgeons' experience was gained chiefly from private patients, who generally came early, and who could generally be cured in a short time; whereas other surgeons, with a majority of hospital cases at a more advanced stage, and with various complications, were not so optimistic, but found that the treatment often ran on for many months, or even for years, and even in the end were not "cured."

KERATITIS DENGUE AND POST DENGUE.

By J. Lockhart Gibson, M.D., Edin., M.R.C.S., Eng., of Brisbane.

Although my experience of these cases has not been large, they appear to form a very definite double group, and to have been unrecorded before. Their diagnosis appears to me important for prognostic reasons. Treatment in the cases of keratitis dengue appears to be unproductive of direct benefit, and in the post-dengue cases it resolves itself into an effort to palliate symptoms. Keratitis dengue is one of the most serious affections which can attack an eye, and I am unaware of any other condition which makes one feel more impotent. Considering the very large percentage (probably 75 per cent.) of the people who in Brisbane were attacked by dengue, the cases of keratitis dengue were extremely rare. Three cases came under my notice, all of them in patients suffering from prolonged or relapsing attacks of the fever. I have heard of no others. Doubtless the post-dengue cases would have been as serious had the febrile attacks continued in them.

I am indebted to the post-dengue cases for being in a position to offer an explanation of the etiology of both varieties. The post-dengue cases, of which

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I saw five, are affected with patches of keratitis neuroparalytica. The dengue cases also appear to start as keratitis neuroparalytica; but to this is added rapid infective ulceration of the affected cornea.

A case of keratitis dengue.—Male patient, aged 42, referred to me by Dr. Jackson. Attack of dengue began on a Monday night. On Wednesday pain commenced in the right eye. On Thursday night pain in right eye was unbearable. When Dr. Jackson saw him on Friday morning, pain and photophobia were so intense that morphia was injected subcutaneously.

I saw him two hours later. Photophobia was still great. Very little injection of the conjunctiva. In the lower outer quadrant of the cornea there was a 3mm. to 4mm. patch denuded of its epithelium, suggesting that this had first been raised as a bleb, and then thrown off, leaving a sharply-defined shallow ulcer, with a transparent floor except in its very centre, where a small pin's head of deep opacity existed.

Being my first case, I did not regard the outlook as very grave. The pupil was easily dilated, and a disinfecting lotion of quinine ordered. Pain subsided quickly, and, probably because the peripheral neuritis had resulted, after an initial stage of hyperæsthesia, in insensibility of the cornea. In thirty hours the change for the worse was very remarkable: the affected patch had enlarged to include one-half of the cornea—it was covered with white macerated necrotic débris, and its margins were dense white and raised. The swollen margin apparently included the eipthelium and immediately subjacent interstitial substance. Temperature was 102°. It was quite evident that the eye would be lost unless radical treatment could stop the apparently infective ulceration. It was explained that the cautery point might do this, but that the outlook was not hopeful. The whole area was cauterised with a dull red electro-cautery point. For twenty-four hours ulceration appeared to be at a standstill, making one regret that the cautery had not been applied when he was first seen. During the succeeding twenty-four hours it spread again rapidly to involve the whole cornea. The whole surface was then again touched with a dull red cautery point, but without beneficial result. The conjunctiva became inflamed, but apparently as a secondary affection. There was very little mucopurulent discharge after the first day, and at the end of a week some chemosis of the conjunctiva.

The cornea, when the eye settled down, was represented by little, if any, more than its posterior limiting membrane with the iris adherent to it behind. On the second day the eye was so hopelessly going to the bad that I do not regret having applied the cautery point as a forlorn hope; but I am more than glad that I did not do so on the first day, as its use then would have left a doubt in my own mind as to whether the cautery was not a contributing factor in the advance. If my belief that the cases are primarily neuroparalytic be correct, cauterising the ulceration, even though itself of an infective nature, could not stay the progress.

Cases of keratitis post dengue are also very important for prognostic reasons. Treatment is merely palliative. It was these cases which cleared up to me the pathology of the cases of keratitis dengue. I found the area of cornea affected to be, after the first few hours of hyperæsthesia, anæsthetic, and that the anæsthesia persisted in one case for two months; that after three months sensibility had begun to return; after four months it was quite restored.

The eye becomes affected after the actual attack of dengue has subsided, generally during the first week of convalescence. In its most typical form a triangular area of the cornea is affected, with its base at the periphery and its apex at or beyond the centre of the cornea. At its onset there is acute pain with photophobia and lachrymation. The corneal epithelium is possibly

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raised as a bleb at the extreme outset, but this I have not seen; it, or a superficial layer of it, appears then to slough, and to leave a very shallow ulcer, in whose floor appear a number of small pin's head opacities, apparently situated in the more superficial layers of the interstitial substance of the cornea. In the worst case the triangular area involved a fourth of the cornea; the least severe had a very narrow triangle. In the worst case there was swelling and inflammation at the corneo-scleral margin, not unlike that seen in cases of acute inherited interstitial keratitis. In this case photophobia and lachrymation continued until the corneo-scleral margin cleared up, and until the pericorneal injection had disappeared. In the three severe cases repeated and continued use of a mydriatic was necessary, to control the irritability of the iris. I found in all cases that the use of bisulphate of quinine was followed fairly quickly by some healing of the superficial ulcer, but that the interstitial opacities persisted for months, and that the epithelial surface of the area—especially that part in the centre of the cornea—became thickened, and glistening white in appearance. This glistening white central portion, composed chiefly, I think, of thickened epithelium, gives place to a much slighter opacity, and results in comparatively little disfigurement. How completely it will clear up I cannot say. It is still (September, 1905) decreasing in my first case, which began five months ago, and sees now 6-24. After two months sensibility begins to return in the severe cases, but very slowly, and after three months the sensibility of the affected area is still markedly less than that of the surrounding cornea.

The post-dengue cases are evidently due to peripheral neuritis of the portion of the corneal nerve plexus corresponding to the affected area, with resulting keratitis neuroparalytica. The initial stage of extreme hyperæsthesia, the subsequent absence of sensibility, the very prolonged character of the affection, and the fact that peripheral neuritis in other parts has been not uncommonly observed during convalescence from dengue all point to this diagnosis.

It is important to recognise the true nature of these cases, if only because the patient can be informed that a severe attack will persist for months, and may permanently impair sight.

I have given strychnine internally, in the hope that it may have some influence in hastening the recovery of the paralysed nerve filaments. It certainly raises the general health, and possibly helps recovery of the nerves.

In the cases of keratitis during the actual attack or attacks of dengue, we have, I believe, added to keratitis neuroparalytic infective ulceration of the cornea. This may, of course, be due to infection from without; but as, in the patients observed by me, it occurred in all those who at the time were still suffering from the fever, and in none of those whose temperature had fallen to and remained at normal, there seems reason for concluding that infective ulceration was due to the organism of dengue itself acting upon the portion of cornea unprotected by a nervous supply.

A CAUSE OF PSEUDO-MYOPIA.

By J. Lockhart Gibson, M.D., Edin., M.R.C.S., Eng.

I have recognised for many years that nasal obstruction, especially post-nasal obstruction, emphasizes the presence of errors of refraction. My attention was first called to this by finding that children requiring the correction of, say, one and a half dioptres of hypermetropia prior to the removal of adenoids were able after their removal to relinquish their glasses and make their extra

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accommodative effort without discomfort. A second observation in this connection is one which most of us must have made, even if the explanation given it has not coincided with mine, viz., that children with comparatively small amounts (say 1½D.) of hypermetropia often hold their books very close to their eyes when reading, much closer than emmetropic children—although they, as a rule, see well in the distance. I have found that such children have nasal obstruction, and that it is more important to remove this than to correct their error of refraction; *i.e.*, after removal of adenoids the extra accommodative effort for near work is often made without discomfort, and is not made, as formerly, in excess of requirement. My explanation of the mechanism by which this excessive accommodation is brought about will not add another to the already too swollen list of reflex nasal neuroses. I have considered it a result of the distinct and constant increased effort required in respiring through an obstructed nose—of the nature, in fact, of an overflow stimulus from the respiratory centre.

The cases which have induced this paper are cases which exhibit to test-type examination all the characteristics of myopia, and sometimes those of myopic astigmatism. They are generally children, and are not common. I have observed the condition in one adult. I will quote, shortly, two cases; one because he was my first case, and because his pseudo-myopia was astigmatic, and the other because he turned out to be actually hypermetropic. Each was cured of discomfort and of short sight by the removal of adenoids. In each case the cure has been permanent.

E. B., aged 11 years. First seen, August 20th, 1900. History—Eyes troubling for a year. They feel weak, and he cannot look at anything for any length of time. Has a nervous spasmodic habit of moving the skin of his head, and his ears, and of frowning. Reads 6-12 and with an effort some of 6-9. To direct ophthalmoscopic examination, no myopia. With correction—Right eye — 1 D. cyl., axis vert. Left eye — 0·50 D. sph. — 0·25 D. cyl., axis vert.—reads 6-5 and eyes are equal, and fan in each case is equal.

A considerable mass of adenoid hypertrophy on naso-pharyngeal roof, hiding upper one-third of septum. Drum membranes are indrawn and opaque. Breathes heavily at night. Hearing natural. Myopic astigmatism diagnosed to be spasmodic. Advised removal of adenoids and no correction of refraction. Removed adenoids on September 19th. Facial spasm steadily decreased and myopic astigmatism gradually decreased, until in a month from operation his facial spasm had disappeared, and he saw 6-5 without any correction. There was a doubtful 0·25 of hypermetropic astigmatism in his right eye, axis horizontal, and no astigmatism in his left eye. He has remained well and quite free from eye trouble. No mydriatic was employed.

S. N., aged 14 years. Sent to me on January 15th, 1904, by Dr. Lilian Cooper. For months has held book very close to his eyes and has seen badly in the distance. No headaches. V. Right eye, 6-36; left eye, 6-24. To direct ophthalmoscopic examination, neither fundus requires a — glass.

With correction—Right eye — 1 D., left eye — 0·75. Sees 6-5. Has adenoids on naso-pharyngeal roof, hiding base of septum. Had not been noticed to breathe with difficulty.

January 19th.—Removed a considerable quantity of adenoid hypertrophy from his naso-pharynx.

January 22nd.—Reads 6-9 with both and each.

January 26th —Reads 6-9 and some of 6-6.

February 6th.—Reads 6-5.

February 27th.—Reads 6 5, and can be made to do so also with + 1·25 D. sph.

He has kept perfectly well and has no trouble with his eyes or head. No mydriatic was employed.

Dilatation of the pupil has been observed (De Schweinitz) to occur in vomiting, and in forced respiration; and if forced respiration can induce

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dilatation of the pupil, there can be no reason why it should not be associated with spasm of accommodation. The overflow stimulus explanation appears to receive some support from the cases we get of facial spasm due to accommodative effort. Twitching of the lids associated with accommodative effort is of course common, but I have seen some cases of very marked facial spasm on the side corresponding to a more hypermetropic eye, the spasm being at times clonic, and at other times continuing for quite half a minute or more, and then relaxing to be soon repeated. When this spasm has not been of very long standing, correcting glasses cure it. This spasm has evidently been an overflow stimulus from the accommodation centre, first to the orbicularis centre and after to the muscles of expression. It has been a result of prolonged overactivity of the accommodation centre, and is apt in adults not to show itself until some presbyopia has supervened. It is not very infrequent in children.

Galassi and Harold Gifford describe what they call an orbicularis pupil reaction occurring when a forcible effort is made to close the lids. The contraction of the pupil which then occurs they ascribe to an overflow stimulus from the nucleus of the orbicularis fibres of the facial to the pupil contracting centre We would all admit that the muscles of expression are implicated in forced respiration, and there is no reason why, in some people, a further overflow should not implicate the accommodation centre. I have myself described marked facial spasm in children simulating chorea, and cured by the removal of adenoids—which induced it, either by interference with free nasal breathing, or by acting as a foreign body in the nose: the facial spasmodic contractions being then part of an unconscious effort to move or dislodge them.

DISCUSSION.

DR. POCKLEY said *(à propos* of remarks by Dr. Kirkland)—"We cannot let that pass." Surely it was the general experience of ophthalmic surgeons that it was often the slighter degrees of ametropia, especially of astigmatism, that caused headache, which was promptly banished by appropriate correction. He practically never found it necessary to vary the prescription of another oculist within a few weeks or months, although opticians' prescriptions almost invariably needed alteration; and as for changing his own prescription three times in as many months, he could say positively that he had never done such a thing—except, perhaps, in the case of a neurotic patient, for whom one often found that no correction gave permanent satisfaction, though a very trifling alteration of the glasses gave comfort for a time.

PARAPHYMOSIS PALPEBRÆ.

DR. LOCKHART GIBSON showed a photograph of a case of paraphymosis palpebræ, occurring in both eyes at the onset of acute trachoma. The case came under his care at the Children's Hospital after it had existed for some weeks. Eversion of the upper lid was complete, its lid margin forming a tight constricting band above. The swelling of the constricted lid was extreme. Incisions and a canthoplasty gave some relief to tension. The complete eversion lasted for months. Had it not occurred at the onset of attack there can be little doubt but that both eyes would have been lost, as no cornea could have withstood such pressure from so rough a conjunctival surface. After treatment of the trachoma for some months, it became possible to replace the lids. The cornea remained clear throughout. The boy ultimately became cured as to his lids, and regained normal vision. He was in hospital for more than twelve months.

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INCREASED INTRA-CRANIAL PRESSURE AS A CAUSE OF OPTIC NEURITIS.

By F. Wallace Mackenzie, M.B., C.M., Edin.

Increased intra-cranial pressure has long been recognised as a cause of optic neuritis.

Schmidt-Rimpler expressed the opinion that the neuritis is due to the cerebro-spinal fluid being forced into the space between the optic nerve and its sheath.

Parinaud extended this theory by suggesting that the accumulation of fluid under pressure in the ventricles interferes with the lymphatic circulation, and that the neuritis is caused by the œdema thus created.

Sourdille suggested that the actual mode of production is somewhat different. He pointed out that the chiasma is actually, anatomically, almost portion of the wall of the third ventricle, and is closely incorporated with it. An œdema, therefore, of the walls of the ventricle would directly act on the chiasma, and so down the optic nerves to the discs. The swollen nerve would thus be compressed in the optic canal.

Probably none of these theories is correct. Reasoning by analogy, it is difficult to understand how an inflammatory process can be set up, either by interference with the lymphatic circulation or by compression of the nerve alone. I am inclined to think that the increased tension leads to a change in the fluid secreted, and that a toxin is formed which excites the neuritis.

The following cases illustrate the belief that optic neuritis is one of the consequences of increased intra-cranial pressure.

J. N., aged 23 years, was in good health till October, 1903. At that time he had a severe blow on the back of his head, from a fall off a horse. In December following his sight began to fail. He consulted his doctor in the country, and was told that he had optic neuritis. He went to Wellington, where he was treated by two doctors. Then he went to Dunedin: there he was treated by electricity for some weeks. He got a little better at first, but soon relapsed, and gradually got worse. He then went home, and had no treatment for 10 months. All this time the sight gradually failed, and he suffered daily from headaches, which made him stupid.

I saw J. N. first on December 31st, 1904. At this time he walked with his head erect and his eyes wide open. The pupils were partly dilated, equal, and reacted to light. Vision was only perception of light; hearing was considerably impaired; and he complained of almost continuous headaches. There was atrophy of both optic nerves, with some blurriness of the outer edge of each disc.

I took him into hospital, and had his head shaved. There was a depression in the skull, extending from half an inch below and behind the left parietal eminence nearly to the lambda.

On January 6th, 1905, assisted by Dr. Faulke and Dr. Gilmer, I trephined the skull over the outer part of the depression, taking out an inch disc of bone; the disc had a well-marked ridge on the inside, and the ridge could be felt to be continued downwards. The dura mater showed no pulsation. I cut the ridge off the disc, and filed the surface smooth; then cut a groove out of the skull, in the position of the rest of the ridge, with nibbling forceps. The dura mater was opened by a curved incision, when a large amount of cerebro-spinal fluid escaped. There was normal pulsation of the brain, and it was of the usual color and consistence. The dura mater was sutured with catgut, the disc of bone was returned to its place, and the scalp wound sutured. A small hank of celluloid string was put into the lower end of the wound for

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drainage: this was removed next day. At the end of six days the wound was dressed again, and the scalp sutures removed. The patient was kept in bed for a month. He had no more headaches after the operation, and there has been a gradual improvement in his sight. From having perception of light only, he can now—six months after the operation—distinguish large objects, such as a watch or a pocket-knife, and he can walk about the streets alone.

Brudenel Carter pointed out, some years ago, that optic neuritis could be cured by opening the optic nerve sheath; and Gowers has observed that, even in cases of incurable cerebral tumor, optic neuritis cleared up after trephining the skull and opening the dura mater.

At the Intercolonial Medical Congress, held in Sydney in 1892, I reported a case of "Atrophy of the Optic Nerves with Dropping of Watery Fluid from the Left Nostril." This patient's trouble began with headaches and progressive blindness, due to optic neuritis, together with attacks of unconsciousness, which were probably due to gradually increasing intra-cranial tension. At the end of a year a dropping of cerebro-spinal fluid began from the left nostril, and, coincidently with this, the headaches and attacks of unconsciousness ceased. The dropping from this man's nose continued for four years, and he remained in good health. Then he consulted an American gentleman, who had established a reputation in nasal affections. A few brisk applications of the galvano-cautery cured the dropping from the nose, and the patient came under me again, as the headaches and fits had recurred. After this the patient went from bad to worse, and ultimately died in a general convulsive attack.

I have seen more than one case of optic neuritis clear up after a radical operation for mastoid suppuration. In these cases I believe that the inflammation in the neighborhood of the cerebral membranes sets up increased secretion of fluid within the dura mater. In such a case, in which I explored the middle fossa of the skull for an abscess which was not there, I noticed that there was no pulsation of the brain until the dura mater was opened. The patient had marked optic neuritis, which subsided and left quite useful sight.

It is my opinion that, even where no local lesion can be found to account for the optic neuritis, it is good treatment to open the dura mater and relieve the increased tension inside that membrane.

ACCOMMODATION IN APHAKIC EYES.

By R. H. Jones, M.B., B.S.

If the members of this Section of the Congress were asked to give an account of the mechanism of "Accommodation," I suppose most would quote Helmholtz's views, and others perhaps would give Tscherning's. Both theories require that the lens should be present, and in correct anatomical relation to the surrounding parts. It is more than likely no one would speak of the possibility of accommodation taking place in an eye from which the lens had been removed. That such a possibility exists is shown by the cases I am going to report.

In January, 1902, I was consulted by a young man 17 years of age, who wished to know if it were possible to prescribe glasses for him that would give better vision than those he was wearing. Seven years previously he had been operated on for cataracts. He was under observation for four months, and had a second operation on the left eye.

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Examination:—Right Eye—There is a small scar in the cornea, up and out, such as might be caused by a discission needle. Anterior chamber is deep; pupil active, oval, with longer axis down and out. Left Eye—Anterior chamber deep; pupil active, oval, with longer axis down and out.

In both pupils are the remains of lens capsule, but in each there is a clear opening.

R.V. $\bar{c}$ +7.D Sph.
+2.D Cyl. axis 15° = 5-9.
L.V. $\bar{c}$ +11.D Sph.
+ 0.75D Cyl. axis 75° = 5-5 2 letters.

My patient expressed great pleasure at the result, and said he could see everything, even at close range, quite distinctly.

Testing him, I found with this correction he could read J4 from 15 to 60 centimetres with the right eye, and J4 from 12 to 65 centimetres with the left. (His vision was still further improved with +3.D. Sph. added to this correction; he could then read J1 comfortably.)

This power to read small type after the loss of the lens was quite new to me; and I regret that I did not find out how the accommodation was affected by mydriatics and cycloplegics. I have not seen any account of this condition in the text of Ophthalmic Surgery, but I have a recollection of seeing its occurrence mentioned in one of the journals. I spoke to Dr. Symons when he was last in Sydney about this case, and he told me then that he had seen similar cases. Since then he has kindly sent me brief notes of another patient who has preserved the power of accommodation after the loss of the lenses. Dr. Symons' patient was a woman 32 years 8 months of age. In each eye there was congenital cataract, and vision was less than 6-60. The operation for the removal of the lenses was needling.

For the right eye—+5D Sph. for distance and +9D for reading, and for the left eye +5D Sph.
+1.25D Cyl., axis 160° for distance, and +9D Sph.
+1.25D Cyl., axis 160°
for reading were ordered. With these vision was 6-6 and J1. Afterwards, however, the patient stated that she could read better with her distance glasses than with those prescribed for reading, or any intermediate strength. She wrote, under date August 14th, 1905, that she "cannot see near work better with reading pair than distant pair." This patient would seem to have preserved greater power of accommodation than mine.

Case 3. Senile Cataract—Extraction with Iridectomy.—In September, 1904, Mrs. B., aged 58 years, consulted me. Five years before she had been told by an ophthalmic surgeon that she had in the right eye a dark cataract.

Examination.—Right eye, mature cataract. Left eye no fundus reflex. Lens becoming opaque.

On November 9th I did an iridectomy and extracted the right lens. Recovery normal.

On December 20th.—
R.V. +10.D +1.50D Cyl. 30° = $\frac{5.5}{18.}$ $\bar{c}$ +14.D +1.50D Cyl. 30° = J2 slowly
The distance glasses ordered in No. 2 neutral tint.

January 19th, 1905—
R.V. +10.D +1.50D, Cyl. 30° = $\frac{5.5}{12}$ (3 or 4 letters) +14.D +1.50D, Cyl. 30° = J1.
Ordered +13.D
+1.50D, Cyl. 30° for reading

August 24th—
R.V. +10.D +1.50D 30° = $\frac{5.5}{6}$ nearly all, and with these glasses reads J4 fluently from 45 up to 17c.m. and J2 slowly at 23c.m.

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The common features of the first two cases are that they lost their lenses early in life, and that the irides were not mutilated. Any attempt by me to explain how accommodation was brought about would only be of the nature of guesswork; but I report the cases because of their comparative rarity, and in the hope that some members may be able to throw light on them.

DISCUSSION.

Dr. Lockhart Gibson mentioned the case of a very hale and accomplished lady, now 72 years of age, for whom he had removed two cataracts—one in 1898 the other in 1901. Her corrected vision in each eye was and is 6-12. With the glasses, which enable her to see 6-12, and which she wears constantly, she can read J1 at 12in., and very comfortably at 15in. A secondary membrane had required needling in each eye, but the opening in it was not very small in either. She has correction for near work, but often uses her distant ones for it. For intermediate distances she is very comfortable with her distant glasses.

EXOPHTHALMOS IN RELATION TO DISEASE OF THE ADJACENT BONY CAVITIES.

By F. Wallace Mackenzie, M.B., C.M., Edin.

Exophthalmos, apart from exophthalmic goitre and pulsating exophthalmos, is a symptom which is not at all commonly met with; even in eye hospitals, where a large number of patients are seen daily, cases are quite uncommon. On account of this comparative rarity the actual cause of the exophthalmos in a given case is often difficult to determine, and as the cases which occur are generally those in which one is called on to give a prognosis, and to treat, the matter of finding out the cause is one of great importance.

My present purpose is to confine my remarks to the experience I have gained in studying the few cases which I have myself had to deal with, and to indicate the lines upon which I have classified them, for the purpose of diagnosis.

The bony cavities adjacent to the eye are the orbit, the frontal and sphenoidal sinuses, the ethmoid cells and the antrum of Highmore, and the cranial cavity.

I have seen one case in which the exophthalmos was due to a sarcoma which invaded the orbit from the base of the brain. The patient, who was 33 years of age, came as an out-patient at the Wellington Hospital. He complained of blindness in the left eye, with severe headaches. The left eye was slightly more prominent than the right. On exploring the orbit with a hollow needle, a bony mass was discovered deeply seated to the outer part. The external palpetral angle was extended outwards by an incision, and a square piece of bone removed with saw and chisel from the outer angle of the orbit: this enabled the bony mass to be palpated with the finger. It was smooth, rounded, and quite hard, and it was evidently a bulging outwards of the wall of the orbit. The bone was replaced and the soft parts sutured. The man remained in hospital for a month, getting gradually worse. He was then sent home to the Wairarapa, where he died. I secured a piece of the growth, which proved to be a small spindle-celled sarcoma. While in Wellington Hospital this man was treated with iodide of potassium in large doses and mercury.

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The next case I saw was a married woman aged 35, who had borne several children. The right eye had gradually become more prominent than the left—there was no other symptom, except a degree of discomfort on turning the eye upwards—the protrusion was straight forward and the lids could cover the globe. There was good movement in every direction. The patient was treated first with iron and arsenic, as she was anæmic; but there was no improvement locally. Then she was ordered iodide of potassium in 20 grain doses, with 1-50th grain of perchloride of mercury. The discomfort on moving the eye upwards disappeared in a few days, and at the end of a month the bulging of the eye was decidedly less. At this time 3-grain doses of quinine hydrochlorate were given three times daily, in addition to the iodide and mercury. Improvement went on slowly, and I lost sight of the patient after three months. She reported a year after coming under treatment, and the position of the eye was normal. There was probably a gumma in the orbit.

The following year a boy of 14 years, and a married woman of 52, came into hospital in the same week, both with well-marked exophthalmos. I exhibit their photographs (figures 1 and 2). The boy, a quarter-caste Maori,

FIGURE 1.

had his left eye displaced downwards fully half an inch below its fellow. All movements were good, sight was normal, and he felt no discomfort. The upper and inner bony wall of the orbit was prominent, and could be dimpled in on pressure. This was evidently the cause of the proptosis. The boy's father said the condition began shortly after an attack of measles, and had gradually got more marked during the past six years. At first he had had a considerable discharge of pus from his nose, but this had disappeared for some years.

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I first removed the anterior end of the left middle turbinated body with cutting forceps and snare, because it was swollen and rough; but it only contained a large bony air cell. Then, under chloroform, I opened the frontal sinus. It contained a thick, sticky, yellowish, transparent fluid; when this was washed out it appeared fairly healthy. I passed a curved probe into the nostril and drew a silk thread through the infundibulum, and then drew a rubber drain into the canal, leaving one end out at the forehead, the other at the nostril. This remained for a fortnight, and then it slipped through itself, and the wound was allowed to close. The eye did not go back into its proper place. The boy is now a man of 24 years, and the eye is still down his cheek, but gives no trouble, and he has binocular vision.

The woman (figure number 2) had the left eye bulging prominently between the lids, but not displaced downwards; vision was good and the movements quite free, the lids closed over the eye with an effort. She stayed in hospital for two months, and got gradually worse. She was treated with

FIGURE 2.

iodide of potassium and mercury, together with iron and arsenic. She suffered very much from headaches and vomiting latterly, and ultimately went home and died with symptoms of brain tumor. This woman had no nasal trouble, nor any evidence of involvement of the accessory sinuses. She probably had a sarcoma of the orbit, which spread to the interior of the skull.

Orbital cellulitis is, in my experience, the most frequent cause of the variety of exophthalmos under consideration. All of my cases—of which I have records of seven—were associated with a suppurative process in one of the adjacent cavities. One of these cases followed directly on the extraction of an upper molar on the same side. The patient was a delicate lad of 21 years of age. He had suffered from, and was still affected with,

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diseased bone in the left shoulder joint. Two months before I saw him—October, 1903—he had a right upper molar tooth extracted for toothache. The right eye became bulged and painful, and remained so till I saw him. At that time he complained of a dull pain in the bone above the eye, and the discharge of a thick, clear, jelly-like material from the right nostril. On examination the bridge of the nose was somewhat broadened, and the right ala collapsed on inspiration. The breath had a bad smell, and the patient looked ill. The inferior turbinated body was swollen; pus came from under the anterior part of the middle turbinate and trickled down over the septum from the roof of the nose, which was smeared with pus. In the mouth the right second upper molar was missing; the wisdom tooth on the same side was half erupted and loose. A probe passed from the socket of the second molar into the antrum of Highmore disclosed the fact that a considerable part of the alveolar margin was loose. An incision through the gum allowed the partly erupted wisdom tooth to be removed, along with much loose dead bone, leaving a large opening into the antrum. The eye still retained its position of proptosis downwards and outwards, so I excised the anterior end of the middle turbinate. The cells in it contained a glairy mucopus. Far back in the nose, and attached under the middle turbinate, was a large polypus, blocking the nostril. I removed it with a snare. It had probably been much more than two months growing, and may have been a result of the same disease which set up the acute necrosis in the jaw. Next day the bleeding from the nose had ceased; a probe passed through the alveolus showed that there was bare and brittle bone under the orbit. Pus was discharging freely from the nose, both from the middle turbinate and the roof, and the eye had returned almost to its normal position.

The lad was in very poor health, so, having given an outlet for the pus in two of the diseased cavities, I allowed him to go to his home in the country. Six months afterwards his family doctor reported that the patient had improved considerably, but that he had lately had an attack of orbital cellulitis which caused marked exophthalmos and required an incision under the lower lid, where he discovered that the orbital margin was necrosed. I have not seen the case since, but I suspect there had been an attack of acute necrosis of the superior maxilla when the tooth was extracted, and that the patient has a dangerous course to pass through yet. I think that, in addition to the trouble in the superior maxilla and ethmoid bone, there is pus in the frontal sinus, and that very radical treatment will be required if an attempt is to be made to cure the disease. In the maxilla I expect sequestra will gradually separate and require assistance to get away, while the ethmoid cells and frontal sinus—having suitable outlets for the pus—may not demand active interference until the patient's health is better.

Another instructive case of orbital cellulitis was a boy of 14 years of age. He came first in April, 1898, complaining of pain and swelling of the right side of his face from a blow with another boy's head at football ten days before. He had lost a night's sleep from the pain. The skin over the lower margin of the orbit was greenish colored from the blow, but it was also swollen. The upper lid was swollen hard and partly closed—the eyeball bulged forward. There was a boggy feeling above the inner part of the upper eyelid, and both orbital ridges were intensely painful on pressure. Temperature 101° F.

I advised incising the tissues over the bones in these places to relieve tension, and the same afternoon operated under an anæsthetic. The periosteum over the lower orbital ridge was healthy, but an incision half an inch long was made into it to be sure. The skin wound here was sutured and covered with collodion and cotton wool. An incision down to the bone was then made from the supra-orbital notch towards the inner canthus. Here

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the periosteum was separated by pus over a considerable area of the roof of the orbit, and a probe readily passed backwards for fully half an inch. A strip of gauze was put into this wound and a dressing applied. The boy spent a bad night: he was very sick—probably from the chloroform—and pain in the eyebrow was bad enough to keep him awake all night. Next morning his temperature was 101° F. The wound was dressed: a good deal of pus was on the dressing, and putting a strip of gauze into the wound caused great pain. I decided to explore the frontal sinus, and did so the same afternoon under chloroform anæsthesia. I gouged a hole through the roof of the orbit internal to the supra-orbital foramen and just under the arch. The bone is thin here, and the small mastoid gouge went through very easily. The sinus was evidently full of pus under some pressure, for it squirted out when the gouge went through. The cavity was washed out with boiled salt water, and a dressing applied with a gauze tape in the wound, but nothing in the sinus. The boy had a good night and no more pain. The wound discharged for six weeks and gradually closed up. No bone came away, and the frontal sinus seems to have suffered no inconvenience, but is apparently quite healthy again. The proptosis only cleared up gradually, taking about a month.

Of the next case I have attached a photograph. At the time the photograph was taken he was a married man of 33 years with three children. (Figure 3.) The condition began at 4 years of age, after an attack of measles.

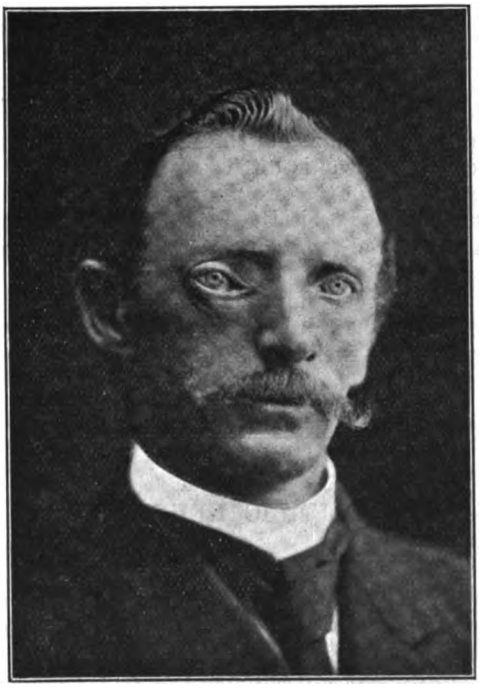

FIGURE 3.

His father told me it got gradually more marked for about a year, and then remained stationary. It has caused no trouble since. He has binocular vision, and all movements are normal. He has + 2 D astigmatism, ax. v., in each eye, and—with correcting glasses—his sight is normal. There is a

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soft elastic swelling occupying the inner angle of the orbit. Its centre is a little above the centre of the pupil, and it is evidently what pushed the eye out of its position. The man consulted me about pain in the eyes, which glasses relieved, and was quite satisfied with the position of his eye. I am of the opinion that it is just as well left alone as long as it remains quiet. I think there is no doubt it is a case of ethmoidal mucocele.

In classifying the extrinsic conditions which I have observed to lead to protrusion of the eyeball, I have excluded pulsating exophthalmos, as I consider it to be a subject of sufficient importance to demand separate consideration, and it is not a condition likely to be mistaken for any of the other causes. I have divided the cases into seven classes :—

1. In tumors pushing the eye forward the expression is very striking: the eye has a peering, intent, or frightened expression, which gives one a generally correct impression of the cause of trouble at first sight.

2. In exophthalmos due to a chronic thickening of the periosteum, in addition to the forward position of the eye—which is not very marked—I have observed a thickening of the eyebrow, with a smoothness of the skin of the forehead and temple on that side.

3. In acute periostitis of the orbit, which I believe always involves the subjacent bone also, the patient looks ill and is in great pain. The eye will have a fixed appearance, and the upper eyelid will be thickened and brawny if the pus has invaded the cellular tissue of the orbit. I have not met with acute periostitis apart from the suppurative form involving the subjacent bone.

4. In acute orbital cellulitis the lids are swollen and thickened. The skin is red over the lids. The upper lid partly covers the eye, and cannot be raised. The eye is bulging and fixed.

5. In ethmoidal mucocele the eye is pushed forwards and to the outer side; there is a smooth rounded swelling just above the inner canthus, and the expression of the patient is often rather sheepish. There is a marked difference in the distance of the two pupils from the middle line.

6. In distension of the orbital wall of the frontal sinus there is a fulness in the upper inner corner of the orbit, and the eye is displaced downwards and outwards.

7. In an acute case of suppuration in the left frontal sinus, which proved fatal from an abscess in the third left frontal convolution, there were swelling and redness of the upper eyelid, closure of the eye, and swelling above the eyebrow.

PLUMBIC NEURITIS.

By J. Lockhart Gibson, M.D., Edin., M.R.C.S.

When asked by our President to fill a gap by opening a discussion on a subject of my own choice, I could agree to do so only if permitted to adapt an already promised paper on Lead Neuritis. I hope by summarising the present position of our knowledge of the action of lead on the optic and ocular nerves, and by going into some detail upon my own observations on the numerous cases in children which have come under my notice, members may be induced to give the Section the benefit of their own views on this interesting and important variety of toxic neuritis.

Since discovering the source of the lead which poisons Queensland children, and its method of ingestion, I have had no opportunity of bringing

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the subject before a meeting of oculists. I am, therefore, anxious to do so now, and have hopes that my matter may be food for discussion. If my own observations bulk largely, I trust to be forgiven on account of the very unusual opportunities I have been fortunate enough to have.

I am anxious to establish certain peculiarities regarding ocular neuritis due to lead which may be helpful in leading to an at least provisional diagnosis of lead-poisoning, in cases whose symptoms would justify not only the thought of almost any form of toxic neuritis, but which would even more forcibly suggest a diagnosis of basal meningitis, or of cerebral tumor. The whole question of toxic neuritis would have been too large for this paper. Still it is possible that some members, whose opportunities of observing other varieties of toxic neuritis have been greater than their opportunities of observing the variety due to lead, may give us their experiences.

Regarding the frequency of lead neuritis in general ophthalmic practice—of ninety cases gathered from literature by De Schweinitz, sixty-five were males and twenty-two females: in three the sex was unstated. The age was unstated in eighteen, but ranged from 12 years to 79 years in the remaining seventy-two. He concludes that it is a comparatively rare condition, even when other symptoms of lead-poisoning are present. In 1897 I recorded the fact that, between 1891 and that date, twenty-four cases had been admitted to the Brisbane Hospital for Sick Children; of these sixteen were girls, and eight were boys: the youngest was aged 4 years, the eldest 8 years. Seven of these cases became quite blind, one or two others recovered to count fingers at 2 yards, the others appeared to regain natural sight. From 1897 to June, 1905, thirty cases have been admitted; making in all fifty-four cases of ocular neuritis due to lead. This number is exclusive of cases seen in private practice.

The total number of cases of plumbism admitted to the Brisbane Hospital for Sick Children since 1891 reaches the respectable total of 200, *i.e.*, including cases of wrist and ankle drop with the fifty-four ocular cases.

Amongst forty-eight lead workers examined by Packard, twenty-four had at one time or other symptoms of lead-poisoning; but no case of amblyopia directly traceable to the metal could be discovered. Among 15,000 case records in the General Ophthalmic Hospital practice of De Schweinitz for five years, only three undoubted cases of lead neuritis presented themselves. Uhthoff found, amongst 130 cases of toxic amblyopia, only one case of lead amblyopia. The conclusion, therefore, has been that only a small proportion of the toxic amblyopias are attributable to lead. This conclusion appears to require reconsideration, at least in climates where conditions making children liable to lead-poisoning obtain.

The Queensland cases have demonstrated the peculiar fact that only very rarely is a child suffering from the wrist and foot drop forms of lead palsy affected at the same time with ocular neuritis, and *vice versa*. I have, indeed, observed only two or three cases. But several of my ocular cases have had attacks of wrist and foot drop antecedent to or subsequent to their ocular attacks.

Lead neuritis may be either acute or chronic. Acute cases, Swanzy says, appear to be often associated with acute cerebral disturbances—due probably to the effect of the lead poison on the brain. These effects include convulsions, delirium, and coma. In those cases in which acute cerebral disturbance occurs, Gowers says that optic neuritis is often observed, with considerable swelling of the discs, and accompanied by hæmorrhages. Even some such severe cases of optic neuritis have been observed, Swanzy says, to recover complete vision, while others, of course, have resulted in atrophy of the nerve. To quote Swanzy:—"Inasmuch as the symptoms of bad cases of lead encephalopathy usually consist of severe headache, vomiting, and convulsions, the spasm being

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in some cases epileptiform, it is evident that, when intense double optic neuritis is added, the diagnosis between this disease and cerebral tumor has to be considered."

Byron Bramwell, whose experience of both lead encephalopathy and of cerebral tumors has been extensive, says—"So closely do the two conditions (tumor and lead encephalopathy) in some cases resemble each other that I never commit myself to a positive diagnosis of intracranial tumor without having previously excluded lead-poisoning."

According to Swanzy—"The aids to the diagnosis will, of course, be the presence or absence of the characteristic blue line on the gums, as also of anæmia, colic, constipation, wrist drop, and lead in the urine, together with the previous history of the patient's occupation."

The greater number of our Queensland ocular neuritis cases has been of the acute variety, and I think that you will agree with me that they are much more likely to be mistaken for basal meningitis than for cerebral tumor; and that the diagnosis, in isolated cases at least, is not, necessarily, made easy by as many of the aids mentioned by Swanzy as we could wish, although, if we take a group of cases, all these aids are available—blue lines in most, anæmia in some, colic in most, constipation in some, wrist drop (in children generally combined with ankle drop) in some (either at the time or previously), and lead in the urine of some. And we shall find that evidence corresponding to the patient's occupation is also now available.

The disease, shortly, as observed amongst Queensland children, is characterised by the sudden onset, with or without preliminary colic, vomiting, and constipation, and with or without severe headache, rigidity of the neck, or retracted head and acute pains at the back of the neck, of paralysis or paresis of one or both external recti, accompanied by optic neuritis or choked disc, and unaccompanied by rise of temperature or by albuminuria: accompanied also by the well-known blue line on the gums, produced by the deposit of the black sulphide of lead; by the finding of small but distinct quantities of lead in the urine; by the fact that the patient has access to painted surfaces, which have been either recently painted, or, more often, whose paint has become powdery and easily detachable—this powder being the very soluble carbonate of lead: accompanied further by the fact that the patient bites his or her nails. The ages of the children affected range chiefly between 2 and 8 years. Only too frequently older children are seen with signs of slight or marked optic atrophy, and with signs and histories supporting a diagnosis of former lead-poisoning.

Acute lead neuritis has received two chief explanations. First, it has been ascribed to the mechanical action of lead inducing effusion into the ventricles and subarachnoid spaces, resulting in distension of the sheath of the optic nerve. In favor of this explanation, according to Clifford Allbutt, is the fact that it is invariably met with in cases of lead encephalopathy where there are signs of increased intracranial pressure, the convolutions of the brain being found flattened *post mortem*. The second explanation is that of Leuber and Deutschmann, who have suggested that the lead particles cause direct irritation to the nerve head by being carried there in the vaginal sheath of the optic nerve itself.

In most of the cases observed by me I have favored direct irritation, not only to the vaginal sheath of the optic nerve, but also to the connective tissue framework of the nerve itself. This explanation would accord with the usually accepted one of chronic lead neuritis.

The very severe cases, however, certainly suggest distension of the ventricles, and it is not unlikely that some of the obscure brain cases which in Queensland children have not been attributed to lead have been of this nature.

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The ocular cases do not die; but, to temper our gratification at this, is the absence of *post-mortem* evidence of the exact nature of the involvement of the retrobulbar portion of the optic nerve.

The explanation that the optic neuritis is due to a uræmic state of the blood receives little support. It receives none from my cases, as none of the purely ocular cases had albumen in the urine.

The fundus changes in acute lead neuritis have been variously described. Gowers and Byron Bramwell describe swollen papillæ. The peculiar feature of the Queensland cases has been extreme swelling of the discs; five and six dioptres of swelling being quite common, less than three dioptres uncommon. The outline of the papilla is entirely obliterated, the disc is represented by an elevated area, marked by radiate striæ fading off gradually into the surrounding retina. The veins are large and tortuous, the arteries relatively smaller, but not actually reduced in size in the acute stage. When the swelling is due to non-solid exudation, enlarged capillaries may be seen below its surface; when the exudation is solid and opaque white, as it often is, merely the coarse radiate striæ of the—at parts—hidden vessels are seen.

My measurements of the elevation of the discs have been taken by comparing the refraction of the surface of the disc with that of the retina during the attack, and more especially with the refraction of the retina and disc after all swelling has subsided. Six dioptres means an advancement of the disc to the extent of nearly two millimetres. There are sometimes hæmorrhages, but not very often, and never large ones. Occasionally the neuro-retinitis extends to and includes the macular region.

I have seen only one case when the optic neuritis was practically entirely post-bulbar, with almost no pinchings in the discs until atrophy supervened. In this case paraplegia externa was complete.

A symptom observed in one case is worthy of record. E. M., a peculiarly intelligent girl of 8 years, was admitted in January, 1904, with a typical attack of acute lead neuritis, with lead in the urine and blue lines on the gums. She recovered quickly, and ultimately regained perfect vision. The child had been ill for three weeks, and had squinted for one week. Our house surgeon, Dr. Lucy Gullett, found that her statement that she saw double with one eye was correct. I confirmed this, finding that she saw double with both eyes open and also with the right eye closed, but single when the left eye was closed. For this reason she covered the left eye when she wished to look at anything. The swelling of the discs only amounted to two dioptres. I noted that there appeared to be relatively more exudation in the horizontal meridian of the left fundus than in the vertical. The case, however, was examined a little hurriedly, and a more careful examination left for two days later. The monocular diplopia had, however, then disappeared: it had lasted for several days. I believe the above to have been its explanation, by causing temporary astigmatism.

A peculiarity of the Queensland cases has been the fact that optic neuritis has never been observed unassociated with some paraplegia externa. As a rule one external muscle is paralysed or paresed; often both. In only one case, where only one muscle was paralysed, was it other than an external rectus: in that case it was an internal rectus. In the most severe cases all the external muscles are paralysed, and the eyeballs are quite stationary. The levator I have not seen implicated. As a rule, where ophthalmoplegia externa is complete, there is opthalmoplegia interna also. Such cases have resulted in complete blindness, with post-neuritic atrophy of the discs. The ophthalmoplegia externa has always been recovered from.

The excellent sight possessed by some of the cases, with several dioptres of swelling in the discs, is astonishing. If the cases can be removed from their

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surroundings and confined to bed before the swelling has existed for long, and sight has remained good, the outlook is hopeful—although not without reservation. Fortunately the presence of squint draws attention to the child even more perhaps than the complaint of defective sight—unless this last is very defective. If the cases are to be explained by interstitial neuritis of the optic nerve itself, it is not difficult to understand that the optic nerve fibres, which, from their possession of no neuri-lemma or grey sheath, must be more vulnerable to the pressure of an inflamed interstitial tissue than the fibres of any other cranial nerve, should be damaged beyond recovery, although the sixth and other ocular nerves invariably recover from a neuritis which is surely interstitial.

Lead, of course, is a recognised cause of paralysis of the external ocular muscles; and there are definite records of its choice of an external rectus, of a levator, and of the whole motor oculi. I have found no record, however, except in my own papers, of a group of cases where the optic neuritis due to lead was constantly associated with paralysis of at least one external ocular muscle. I have seen only one case in which the optic neuritis was unilateral, and in that case the only external muscle paralysed was the external rectus of the other eye. The sudden appearance, indeed, of paralysis of an external rectus accompanied by optic neuritis, and unaccompanied by temperature, and sometimes accompanied by no other prominent symptoms, is sufficient in Queensland to justify a provisional diagnosis of lead.

Of the chronic variety of optic neuritis due to lead, there appears to be more record than of acute; and it is generally accepted as retro-bulbar neuritis, resembling somewhat that produced by other toxic agents, although a central scotoma appears to be less commonly observed when the neuritis is due to lead, and a concentric narrowing of the field is more often spoken of. Appearances in the disc have been described as follows:—"There may be no findings followed by atrophy, doubtless due to retro-bulbar neuritis, or the disc may be pale in its outer half, and the inner half greyish-red and opaque, with ill-defined borders such as is described in alcoholic neuritis; or a diffusely reddened and swollen papilla, without notable swelling, and sometimes with hæmorrhages, may be found."

The chronic cases which I have observed in children have a history of defective sight at least for months, and where the optic discs are not atrophic they are pale, with somewhat irregular and blurred edges. They present, indeed, a half-way appearance between a normal disc and a disc in a state of post-neuritic atrophy. The acute cases, also, which do not run an entirely favorable course, present, ultimately, appearances which vary according to the amount of damage done: the worst being examples of complete post-neuritic atrophy—the partial recoveries presenting pale discs with somewhat irregular edges. The defective vision in both acute and chronic cases has been more a concentric narrowing of the fields than a loss of central vision; but the patients have been young for accurate observations in this respect. As a rule their color sense is not relatively more reduced than their light sense. A child with vision reduced to fingers at five or six yards will show no central scotoma for color—in fact will have a color sense most acute at the centre. Some of the apparently chronic cases were probably cases which at the outset were acute: occasionally the history suggests this conclusion, and sometimes tells of more than one successive summer attack. The following is a case in point:—

A girl, E. B., aged 8½ years, came as an out-patient, with the following symptoms and history. Defective sight: gets nervous twitchings at times. Two years ago was in bed for three months during the summer, with abdominal pains and weakness in the arms and legs. Suffered from a repetition of this attack last summer. Marked blue lines on the gums. Partial atrophy of each

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optic disc. Evidently the defective sight dated from the second attack. Probably keeping the child in bed prevented greater damage to the sight by preventing further ingestion of the poison.

To recapitulate and extend the proofs I published in 1904 of the source of the lead which poisons Queensland children, and its method of ingestion, would make my paper too long. Further observation has confirmed those proofs. It will be sufficient if I emphasize one or two points. Verandah railings, in southern Queensland, at least, after having been painted for two or three years, are covered not by an oily but by a powdery substance, which easily rubs off and adheres to clothes and hands. This powder adheres specially well to sweat-moistened hands. It is nearly entirely composed of the very soluble carbonate of lead. Of eighty-five cases of lead-poisoning, all varieties, from 1898 to 1903, sixty-nine were admitted during the seven summer months—*i.e.*, during the months in which their hands may be expected to be sweat-moistened—and sixteen only during the five winter months. The poisoning is practically confined to children of an age likely to contaminate their fingers, for these play and hang and run round verandahs and fences, and clutch the railings and painted corners of walls in running past. The fact that more girls than boys are affected may be attributed either to a possibly greater prevalence of the nail-biting habit amongst girls, or to the fact that they are more confined to verandahs and houses than boys of the same age.

It may be accepted, I think, that in warm climates easy access to painted surfaces—especially to such as have been exposed to sun and weather—in conjunction with a habit of biting the nails, may be expected to tend to the ingestion of lead and to consequent lead-poisoning, and that the danger to young children is very considerable.

One difficulty in diagnosing these cases when isolated is the fact already indicated, that every case does not furnish every aid to diagnosis. Sometimes a blue line is absent from the gums, though possibly present in an unaffected member of the same family. Sometimes the sample of urine sent fails to yield lead on testing. In one case this occurred until after the administration of iodide and of pilorcarpin, when lead was found by the analyst. It must be remembered that hospital cases have been removed from opportunities of further ingestion of the poison before any collection of urine for analysis begins. The urine has not always been collected immediately. If the child were left amongst its surroundings the lead would doubtless always be found.

Of four cases under treatment at the same time in 1904—all typical, all presenting the same symptoms, hardly differing in degree, all recovering perfect sight—two failed to give lead in the only sample (a week's urine) sent to be analysed, while the urine of the other two cases yielded lead. One of those whose urine yielded lead had no blue line, and one of those whose urine failed to give lead had no blue line. Three bit their nails; the fourth, aged 2, sucked his fingers. In each case abundant lead was available in the form of powdery paint—except in one case, where the paint was still sticky from its freshness.

To illustrate shortly three degrees of neuritis:—

1. A mild attack. E. B., aged 2 years, sent to me by Dr. Clowes, with history of squinting for five days and of defective sight. Symptoms came on suddenly; otherwise in good health, though a little fretful. Slight tendency to retraction of the head. Paralysis of right external rectis: paresis of left external rectis. Double optic neuritis, with several dioptres of swelling in each disc. Pupils not enlarged, and re-act well to light and accommodation. No blue line in gums. Sucks his fingers. Special opportunities for

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powdery paint to adhere to his hands from verandah railings and verandah floor, upon which he spent his days Lead, .32 millegrammes found per litre of urine examined. Result—complete recovery.

2nd. K. R., aged 6½ years, A more severe case. In hospital at present time. History on admission :—Ill two weeks; began with vomiting, violent abdominal pains, and constipation; eyesight began to get weak a few days ago; to-day cannot see at all. Examination :—Very restless, continually crying out as if in pain; head slightly retracted, and neck held stiffly; pupils sometimes large, at others small, re-act slightly to a lighted match when large. The only indication that ophthalmoplegia externa is not absolute is a right eye very slightly directed outwards as compared with the left. Six dioptres of swelling in each disc. For the first week her pulse was irregular at times. Four days after admission she was still unable to see a lighted match, and ophthalmoplegia externa was absolute. Twenty-five days after admission only one external rectis remained quite paralysed, the other muscles all acted slightly; some fairly well. Counted fingers at 1 yard with her left eye; three weeks later, fingers at 7 yards with left eye; very little sight with right eye. Now (three months after admission) left eye sees certainly 6-24, probably 6-18; right eye cannot count fingers. The swelling in the discs had practically disappeared a month after admission. Discs both present an appearance of partial post-neuritic atrophy. It is possible that this case had a little lead encephalopathy; she is now very well.

3rd. A. R., aged 5½ years. A still more severe case. Was seen in consultation in 1900. History :—He had then been ill for two or three weeks, and his case had been diagnosed by Dr. Meek as lead neuritis; with this I concurred. Illness began with pain down the arms, spasmodic, and not always in the same arm—no paralysis; no temperature; retraction of head and pains about head and back of neck. Cries out with these pains on movement; no colic, and eats well. Lead has been found in the urine. Both external recti paralysed. Five to six dioptres of swelling in each disc. Counts fingers at 18 inches; even this amount of sight was put in my notes with a mark of interrogation. When seen two months later he had in the meantime suffered from wrist-drop, but not from ankle-drop, and he was still unable to dorsi-flex his wrist. Pupils natural. Paralysis of external recti has nearly disappeared. In good health. Each disc atrophic. Four years later he counted fingers at 2 feet with one eye, and at 6 inches with the other.

4th. To illustrate the gradual improvement in sight which may occur. M. S., girl, aged 5 years, an attack of severe lead ocular neuritis in 1899. Five dioptres of swelling in the discs and much neuro-retinitis encroaching on the macular regions. Paralysis of right external rectis, paresis of left external rectis. Recovered, with damage to the retinæ and some post-neuritic atrophy of discs. A year later counted fingers at 6 yards and saw 6-60; two years later right eye saw 6-18; left eye 6-24 partly. Now (1905) sees—right eye 6-9, left eye 6-12. Her discs still show marked evidence of previous neuritis, and are pale and irregular. There are marked evidences of old retinitis between the disc and the macula in her left eye. In each fundus the veins about the macular region are somewhat dilated. She has no scotoma for color, and her fields show only slight concentric contraction, central vision being most acute. A sister of this patient suffered from colic and very marked blue lines in her gums, also from twitchings of shoulders and arms. Not uncommonly in families of the lead cases observed by my colleagues in Brisbane have there been histories of infants, just old enough to crawl and hang on to railings, dying from unexplained convulsive seizures. These have long been attributed by Dr. A. J. Turner and myself

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to lead; and, with the explanation of paint as its source, such a diagnosis receives much support. Treatment has consisted in removal from their homes, confinement to bed, and hypodermic injections of pilocarpine daily for six weeks, iodide of potassium after the first few days, in the proportion of five grains thrice daily to a child of 8 years. Sulphates by the mouth. Latterly I have had anterior splints applied to both arms for some weeks, in an endeavor to break the children from the habit of biting their nails.

As prophylactics, zinc white instead of white lead should be used as a basis for paint where the painted surfaces are intended to be of a whitish color, but where a dark color is desired the red oxide of iron should be used.

A prophylactic more easily enforced is correction of the nail-biting habit.

Dr. Pockley considered the Section was greatly indebted to Dr. Gibson for his paper. It was a most valuable contribution, and, as far as he knew, Dr. Gibson's views as to the frequency of lead-poisoning as a cause of blindness in children, and his ingenious and apparently correct explanation of the way in which it was brought about, were quite new and original.

Hearing the paper recalled a case he had had fifteen or sixteen years ago. The patient, a child, had been staying with relatives in Queensland, when she was seized with convulsions from which she quickly recovered, but was found to be totally blind, and unable to stand or hold her head up without support, and her hand-grip was very feeble. The eyes showed total paralysis of all the oculo-motor muscles of both eyes, and also of the sphincter iridis, and probably of the ciliary muscle. There was most intense optic neuritis, and, eventually, atrophy and permanent blindness. The patient is now alive and well, an accomplished musician, but quite blind. Two other men saw the case in consultation, but could not decide on a cause for the trouble. It was thought there might be a growth below the floor of the aqueduct of sylvius, but there were many objections to this, the only proffered explanation. Looking at the case in the light of Dr. Gibson's paper, he had little doubt that it was really a case of acute lead-poisoning.

Dr. Barrett said that Dr. Lockhart Gibson's paper on the causation of optic neuritis by lead-poisoning was extremely interesting, and it would be a necessity in the future to carefully examine the urine for lead in every case of profound optic neuritis. He suggested, however, that the chemical method adopted for the detection of the lead should be scheduled in the publication, so that it might be beyond cavil, since the diagnosis of the causation clearly rested in some cases on this presence of lead in appreciable quantity in the urine.. He congratulated Dr. Gibson on the work done.

Dr. Lockhart Gibson, in replying, furnished the following explanation of the comparative infrequency—or, in places, absence—of lead neuritis in the States of Australia south of Queensland. A large proportion of the dwellings in Queensland are built on piles or stumps, and stand consequently a few feet from the ground. They are all supplied with verandahs, and these with verandah railings—often of wood, sometimes of iron. These railings and the wooden walls of the houses are painted with lead paint. Children, especially those between 2 years and 8 years of age, play mostly on the verandahs. Each time a child touches the railings, or, indeed, the verandah walls, some of the powdery paint adheres to the fingers—especially in summer when these are sweat-moistened. If the child neither bites his nails nor sucks his fingers he escapes, but if he does either he cannot fail to ingest the lead in considerable quantities. I have taken particular notice that in the southern States the houses are not built on piles or stumps, but on the ground; that they are less often of wood, and therefore less often

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painted; and that the verandahs—which are, indeed, less common—are comparatively seldom supplied with verandah railings. In other words, the children have comparatively few opportunities of contaminating their fingers. The summer, also, is shorter in the southern States than in Queensland.

After the discussion it was proposed by Dr. Lockhart Gibson, and seconded by Dr. Kent Hughes, that the following resolution be forwarded to the Congress:—"That the danger of blindness from lead-poisoning amongst young children in Queensland has been recognised; that it is highly probable that painted verandah railings and other painted surfaces, especially those exposed to weather influences, are the source of the lead: that since the paint is carried to the mouths of the children when they bite their nails or suck their fingers, it is important that paint applied to surfaces within the reach of children should contain no lead: that zinc white is suggested as a suitable substitute."

Lead-poisoning in Young Children.

Home Secretary's Office, Queensland, December 13th, 1905.

1. Lead-poisoning in young children, resulting in paralysis, blindness, and in some cases death, is a not uncommon disease in Queensland; 200 cases having been observed in the Brisbane Children's Hospital alone since 1891.

2. It has been ascertained that the source of the lead is the paint of painted railings and walls of houses. It is well known that paint, after being exposed to weather influences, is in a powdery condition, and easily removed by rubbing the surface with the hand, especially a sweat-moistened hand. It has been shown, through specimens supplied to the Government Analyst, that this powder consists of an almost pure carbonate of lead in a soluble form, so that should a child rub off some of it, and afterwards eat with unwashed hands, or otherwise put his fingers into his mouth, he absorbs lead into his system. It has, indeed, been demonstrated not only that the disease is chiefly prevalent in summer, when the hands are likely to be moist with sweat, but that without exception children so poisoned either bite their nails or suck their fingers.

3. It is urgently desired that no lead be used as a basis for paint applied to surfaces within the reach of young children. Zinc, or other paints without lead as a basis, are suitable substitutes.

4. Meantime parents should see to it that children do not bite their nails or eat with unwashed hands.

B. Burnett Ham, Commissioner of Public Health.

Process used in Government Laboratory, Brisbane, for Detection of Lead in Urine.

Government Chemical Laboratory, Brisbane, January, 1906.

At least half a litre of urine is evaporated nearly to dryness in a porcelain evaporating basin with potassium nitrate (20 grams to the litre of urine) over a Bunsen burner, and then to dryness on the water bath. The residue is transferred in small portions at a time to a red-hot porcelain crucible (6cm. diameter), and the whole heated to quiet fusion. The mass must be white when allowed to cool.

On cooling, it is dissolved out with the smallest possible quantity of water, twice its volume, of 1.19 specific gravity, hydrochloric acid added, and evaporated to dryness on the water bath. The mass is soaked in hydrochloric acid, and the heating and soaking in hydrochloric acid kept up till the nitric acid is all driven off.

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The mass is then dissolved in boiling water, which is just acid with hydrochloric acid, the solution saturated with sulphuretted hydrogen while hot, and sulphuretted hydrogen kept passing till the solution cools.

The color obtained is compared with that produced by known amounts of lead in the usual way. It is always advisable, where slight indications are obtained, to allow the sulphuretted hydrogen saturated solution to stand over night.

If much color is obtained the lead sulphide can be filtered off, washed, dissolved in nitric acid, and precipitated, and weighed as lead chromate.

It is very important that all re-agents used, including the distilled water, should be tested for lead. Traces of lead are much more common than is generally supposed. I have even found it in new Berlin porcelainware, which had been previously washed with soap and water. Washing once with lead, free acetic acid gave traces of lead. Further washing, however, showed no more lead. All porcelain and glassware is therefore treated with acid before being used in this work, and "blanks" run regularly, to make certain of the absence of adventitious lead.

J. Brownlie Henderson, Queensland Government Analyst.

THE CLOSURE BY SUTURING OF SCLERAL AND CORNEAL WOUNDS, AND OF THOSE MADE BY THE SURGEON IN THE EXTRACTION OF CATARACT, WHEN THERE IS MUCH LOSS OF VITREOUS, GAPING, OR DELAYED UNION.

By James P. Ryan.

In the minds of ophthalmic surgeons a certain amount of prejudice exists against the employment of stitches in the treatment of perforating wounds of the sclera and cornea. I believe that such a procedure was proposed many years ago by Williams, of Boston, as part of the technique in operations for the extraction of cataract, but I do not know that it was ever practised. The prejudice has no doubt arisen from the fear of providing a channel through which pathogenic micro-organisms might gain an entrance to the interior of the eyeball. There is undoubtedly some ground for this apprehension, but I am inclined to think that the danger has been somewhat exaggerated. At all events I have stitched many scleral, and some corneal, wounds during the last few years, and I have not been able to convince myself that in these cases infection has ever resulted from the practice. Indeed, I go somewhat further, and hold that the immediate closure of such wounds does away with a comparatively large surface which would otherwise be open to the chance of infection from the conjunctival sac. In general surgery perfect coaptation of the wound surfaces is a guiding principle, and this principle, so far as it can be applied in ophthalmic surgery, should be a settled rule of action. I need hardly remind you that, in by far the larger number of instances in which infection occurs, the *materies morbi* is lodged within the eye at the moment of the accident, and that cases of after contamination from the conjunctiva are comparatively rare. It must not be understood that I am an advocate for suturing ordinary small rents in the eyeball, or for closing by this means the incisions made in the usual run of cataract operations. In a considerable percentage of sclero-corneal wounds such a procedure is unnecessary; in a few stitching the conjunctiva may be called for; but, in a still smaller number, suturing the sclera or cornea is advisable, and will be followed by satisfactory results.

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I shall briefly illustrate the treatment I recommend by quoting a few cases:—

In August, 1888, a boy of 9 years was brought to me from the country with a jagged sclero-corneal wound of the right eye, extending from near the centre of the cornea to beyond the sclero-corneal junction. The accident was caused by a piece of tin two days previously. The eye was soft, the iris torn and protruding, and the pupil opaque. The protruding iris was excised, as much lens substance as possible was coaxed out with a curette, and, while this was being done, some vitreous escaped. As the tendency was to further loss, and as the patient was of a highly nervous and excitable temperament, I deemed it prudent to stitch the wound; and, accordingly, under chloroform, a single fine silk suture, passed through the whole thickness of the cornea, effectually closed it. The suture was removed on the fourth day afterwards; the case progressed favorably, and the boy recovered with 6-24, or useful vision.

A man, aged 40, had his left eye cut open by a piece of broken glass. He was seen by me some hours afterwards. The eye was soft, and his vision was reduced to perceiving hand movements. There was an irregular wound ½ inch long in the lower part of the sclera, from which vitreous protruded: this was cut off, and the lips of the wound brought together by a single silk suture. His recovery was uninterrupted, and eventually he obtained a vision of 6-9.

On the 22nd of January of last year a man, aged 48, was admitted under my care to the Victorian Eye and Ear Hospital, Melbourne. His right eye was blind from an injury received some years previously; and, as it was tender and irritable looking, I excised it. The cornea of the left eye was much damaged, and cloudy in some portions, from former keratitis and lead deposits; the pupil was irregular, the lens opaque, and the vision amounted to counting fingers at 8 inches. It did not appear probable that much could be done under such unfavorable conditions, but he was most anxious that something should be attempted, and he was willing to take the risk of failure. Accordingly a preliminary iridectomy, upwards and outwards, behind the clearest section of the cornea, was done early in April; and, on May 18th, the lens was extracted, the incision being in the corneal tissue, but close to the sclero-corneal junction. At the end of five days there was no appearance of union of the flap, and the vitreous began to protrude. This was slight at first, but, in spite of careful compression with pad and bandage and the exercise of the greatest care in keeping the patient quiet, the protrusion increased in size, and the danger of the expulsion of the eye contents became so imminent that on June 6th, nineteen days after the extraction of the lens, the hernia of the vitreous was reduced, and the wound closed by two fine silk sutures, which were allowed to remain *in situ* for a week, when complete union of the flap had taken place. There was no subsequent trouble, and six weeks later the vision with + 12.D. was 6-36, sufficiently good to enable him to do rough work. The operation was done under cocaine, and with the usual antiseptic precautions, and the difficulties were not so formidable as I had anticipated. I was fully prepared for a large escape of vitreous, but none was lost. The ordinary eye speculum, Clark's, was used, but it was held well away from globe by an assistant. A small full-curved needle carrying the silk thread was passed from without inwards through the whole thickness of the edge of the lower flap, the latter being steadied by a fine fixation forceps. The hernia was gently smoothed and forced back with a spatula, disengaging it wherever it may have formed adhesions, and held thus back by an assistant. The needle was passed from within outwards through the upper margin of the incision, and the ligature tied. The second suture was similarly passed, and the wound was effectually closed. In this case no other course of procedure was left open to one; and I have no hesitation in saying that no other line of treatment could have saved the eye. This case was published at greater length in the *Lancet* of November 5th, 1904.

S. B., a woman of 67 years, in poor general health and exceedingly shaky, was admitted to the hospital with double cataract. That of the left eye was

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hyper-mature, and was extracted under cocaine anæsthesia. Some loss of vitreous occurred, and, as she was exceedingly restless and unsteady, I closed the incision with a suture, that was removed four days afterwards. The healing process followed the usual course, and, with correction, she got 6-18 and Jg. 4.

W. H., a man aged 64, double cataract. The right eye was first operated on, with good result. In the left both P.L. and projection were poor. After extraction of the left cataract the lids closed spasmodically, and some vitreous was squeezed out: four days afterwards, and while the dressing was being changed, the same thing occurred. The upper lid was forced between the lips of the wound, and there was another loss of vitreous. A single suture was introduced, and the wound closed. I had small hope of saving the eye. The condition was unsatisfactory for a fortnight, then things began to mend, and when he left the hospital, some weeks later, he was able to count fingers with this eye.

P. L., a man aged 45, with prominent eyes, and hyper-mature cataract of the right one. While operating on this eye there was some escape of vitreous and gaping of the wound, which was closed with a single suture. The subsequent healing was slow; but eventually, with correction, his vision amounted to 6-24.

L. G., a woman of 70, was admitted to the Victorian Eye and Ear Hospital for painful sub-acute glaucoma of the left eye. The vision was nil, but an iridectomy was done with the object of relieving tension and pain. There was some loss of vitreous and gaping of the sclero-corneal incision, which was closed with a suture. The case did well.

A man, aged 78, was admitted to the hospital with recent traumatic cataract of the left eye. Vision was P L. Sedative measures were carried out for ten days, and then, as the tension was increasing, I proceeded to remove the lens. On withdrawing a Graafe knife, after completing the incision, some vitreous escaped; the eye had a shrunken appearance, and the wound gaped. In fear of further loss, this was closed with a single suture, and the healing process was complete when the suture was removed five days later. The eye quieted down in three weeks. He has only P.L., but as the case is recent, and as there is still some blod clot and lens matter in the pupil, some improvement of vision may take place.

Now a few words about the technique and difficulties of the operation. It depends on circumstances, and the fancy of the surgeon, whether a speculum or retractors are used to keep the lids apart. In either case no pressure should be made on the globe. The fixation forceps for holding the edge of the wound should be long-bladed and narrow at the points, the needle or needles small and full-curved, the silk not the finest, and the needle-holder—like the fixation forceps—should be long and fine. It might be better to avoid carrying the needle through the whole thickness of the sclera or cornea, and this may be accomplished by entering the needle from without inwards in the first instance; but, when passing it from within outwards, through the second wound margin, the difficulty of only partially transfixing the flap is much greater, and should, perhaps, not be attempted. Or the surgeon may use two needles on the same thread, and pass both from within outwards. While the suturing is being done an assistant should be on guard, with a broad spatula, to oppose or control any loss of vitreous. To avoid contamination of the suture by its touching the head or clothes of the patient, these ought to be covered with antiseptic gauze and a sterilised towel, or the end of the silk may be suspended and prevented from touching anything by an assistant supporting it with forceps. The judgment of the operator will have to determine how long the stitches should remain *in situ:* the time will vary from twenty-four hours to several days, preference being given to the shorter period. The main difficulty arises in preventing an escape of vitreous while passing the needle through the wound margins. Needless to say, antisepsis, so far as it can be carried out in eye operations, should be strictly enforced; and, during the subsequent treatment, the

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eye should be flushed two or three times a day with a solution of cyanide of mercury of the strength of 1 in 1000. I also recommend that, as part of the preparation of the patient, the eyebrows should be shaved and the lashes cut as close as possible to the lid margin, and the latter be thoroughly squeezed, cleansed, and disinfected, as is my custom in iridectomies and cataract operations.

The few cases cited do not, I am aware, afford sufficient evidence to prove that the sclera and cornea can always be stitched with impunity, but at all events they afford some ground for believing that the practice is hardly so dangerous as it has been represented to be.

With regard to the employment of this procedure in cataract cases, I believe, as I stated above, that it has been proposed, but I do not know that it was ever carried out in practice.

CONGENITAL (?) DISPLACEMENT OF LENSES—ERYTHROPSIA.

By Andrew B. Orr, M.D.

J. H., aged 17, son of a German farmer, was sent by Dr. May, of Bundaberg, to the Brisbane General Hospital, where I saw him first and subsequently in my rooms. J. H. stated when first seen that his sight was very bad, and that all objects looked at with the left eye—the right being covered—appeared pinkish red; such coloration was only noticed since the doctor had put a drop in his left eye. The left pupil was semi-dilated. On examination, both lenses, clear where visible, were found to be displaced outwards and slightly upwards; their inner free edge did not quite extend to the centre of the pupil. There was no antero-posterior tilt, and they seemed fixed, movements of the head causing no apparent change in their position. Differences in depth of the anterior chamber and partial iridodonesis corresponded with the description given in similar cases. Corneæ clear, fundi n. Tn. Sight of mother, brother aged 14, sister aged 12, stated to be good; that of father defective, but no glasses worn. Patient went to school when 6 years old, his sight bad then, but got worse since. No history of injury.

Erythropsia is by no means an uncommon complaint of patients whose eyes are aphakic, whether after extraction of cataract with or without iridectomy or otherwise; less frequently is it noticed by those whose eyes are normal. Dilation of the pupil, though probably helpful in inducing it, is not necessary. It is stated to occur more frequently when the eyes are suddenly exposed to bright light, but occurs also under various other conditions. (*Vide* Graeffe's Archiv. f., Ophthalmologie Bd. 42, s. 207; Ueber Erythropsie, von Prof. E. Fuchs & Bd. 43, s. 19; and Erythropsie von Prof. Snellen.)

J. H. complained of objects looking pinkish red when seen by either eye, the pupil of one being dilated and the other eye covered. On dilating both pupils with homatropine he stated that, when out of doors, everything looked pink; in a badly lighted room this was hardly noticeable. I ordered him spectacles O.U., 15 sph., to be worn constantly. This increased the vision of each eye or both together from Fgs. at four metres to 6–12, and enabled him to read Jaeg. 8 easily. Corneal astigmatism, 45, 1.75: 0 1. Correcting such did not improve his vision. O.U., 18 sph., enabled him to read Jaeg. 2.

Wearing O.U., 15 sph., with pupils dilated, the position of his lenses caused him no annoyance; when of normal size, however, he stated that brightly illuminated objects, *e.g.*, trees, appeared green, yellow, pink. The flame of a lamp, looked at from a distance in a darkened room through a 40 D lens, looked

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white, yellow, and pink, and the same occurred when looking through Bennett's pin points. This was, I suppose, due to the edges of his lenses acting as prisms. Monocular diplopia in either eye was easily produced when a red glass was placed vertically over half the pupil. Occlusion of such portion of the glass of his spectacles as would prevent his seeing through his own lenses restricted his field of vision to a greater extent than seemed advisable; and it is to be hoped that the prismatic effect will, in time, be noticed less by him.

I decided against tattooing corneæ.

Spectacles for reading were not ordered, for obvious reasons.

CATARACT, FROM A PATIENT'S POINT OF VIEW.

By Dr. Andrew B. Orr, M.D.

Darier *(vide La Semaine Médicale*, June 5th, 1895) is credited with describing a new method which enables the patient himself to determine the smallest opacities in his lens and notice the progress of his cataract. It is only necessary for him to look through a strong concave lens, *e.g.*, – 40 D, at a distant light in a darkened room. Opacities, spots, striæ, &c., cast shadows on his retina and are seen by him.

Further particulars of this method are no doubt given in his article "de la possibilité de voir son propre cristallin. Utilité pratique de la 'phakoscopie' pour la diagnostic des fine opacités cristalliniennes et pour l'étude du developpement de la cataracte" ("Annales d'Oculistique," tome 114, page 198): but this I have not read. Those of you who care to take the trouble of observing their own lenses can easily do so by this method, or that given below. I hope they will discover the formige Linsenstern only, and no opacities.

The second method, a simpler and better one, I learnt from Mr. E. J. B., October, 1871, who consulted me in November, 1899, stating that he had cataract in both eyes and wanted spectacles for reading.

Corneal astigmatism—90 + 1 : 90 + 1. Vision—Longe. O S 90 — 1 + 3 V = 6-12 O D 90 — 1 + 3 V = 6-36; prox. O S 90 — 1 + 7 Jaeg 1 90 — 1 + 7 Jaeg 16. On lenticular opacities, more in O D than O S. He then showed me drawings of his cataracts, stating that he had made them himself—not by Darier's method, but by looking through a minute opening in a sheet of metal, copper or tin, towards a cloudless sky. The opacities which I noticed in his lenses corresponded with the drawings when inverted—sufficient proof of his assertion.

Four months later the vision of the right eye was practically unaltered, that of left considerably diminished, and the lenticular opacities increased. I have had the drawings of his cataracts photographed, and lantern slides made therefrom; the date of the first drawing being 1887, and the last 1905. Particulars of the conditions under which they were made and date are shown on the slides themselves, of which there are sixteen, numbered 1 to 16. A circular tin plate with eight perforations—the largest being No. 1, the smallest No. 8—is used by him when making his drawings, and the figures on the slides show the opening used. These slides and tin plate accompany my paper. I need hardly add that Mr. E. J. B.'s drawings are the work of a skilled draughtsman, and such can hardly be expected from the majority of persons suffering from cataract. That of Mr. John Henry, with immature cataracts, slide No. 1B, and J. H., with clear dislocated lenses, No. 1c, is what one usually obtains.

"A method, not generally known, by which obstacles to distinct vision situate within one's own eyes may be observed" is the title of a paper by Mr. E. J. Bennett, communicated by Henry Tryon and read before the Royal Society of Queensland on October 14th, 1887.

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A CASE OF MELANOTIC SARCOMA OF CONJUNCTIVA TREATED BY X-RAYS.

Dr. Pockley read a short note of a case which, though still uncured and under treatment, he thought worth bringing under the notice of the Section, in case other members might think the treatment worth trying.

Mrs. X., aged 40, consulted me three years ago with a growth in the conjunctiva of left eye. It was nearly black, about ¼ inch in diameter, and almost touched the limbus. It was removed, and pronounced a sarcoma by Prof. Welch. Eleven months ago the patient returned with a large growth of same character on the former site encroaching about 3mm. on the cornea. The proposal to enucleate did not meet with the patient's approval, and, purely as an experiment (as the patient was informed), X-ray applications were tried, and have been continued on and off ever since. The first effect noticed (in about two or three weeks) was the splitting up of the pigmented patch into an upper and lower mushroom-like patch, joined by the stems. Soon after the corneal portion disappeared; then the stem of the mushroom went, leaving two separate but smaller areas. The lower of these has now quite disappeared, and the upper has broken up into several smaller areas, each about a pinhead in size, and it looks as if the growth were going to disappear altogether. I was indebted to Dr. Clarence Read, of Chatswood, for the patient, and he has watched with me, with much interest, the changes noted.

A CASE OF MYASTHENIA GRAVIS IN SPECIAL RELATION TO EYE AND THROAT CONDITIONS.

By G. H. Hogg, M.D., Launceston, Tasmania.

Miss ———, aged 27 years, consulted me in December for slight difficulty in swallowing. (For some months she had suffered from repeated attacks of mild tonsillitis, for which I had removed a slightly enlarged tonsil). She stated that she had felt much stronger, and that she no longer suffered from the attacks of tonsillitis, but that for the last week or two she had noticed some difficulty in swallowing fluid.

I found on examination paralysis of the soft palate (fluids, if a large mouthful was taken, occasionally returning through the nose), and also weakness of accommodation, which became more marked in a few days.

The patient was nervous and anæmic, with a quick feeble pulse; the various systems were otherwise normal. She had been subject to business worry and overwork, and had always been of a shy nervous temperament.

With the history of previous sore throat in the past, the possibility of diphtheria as a causal factor was kept in mind, although never accepted; and the case was regarded with suspicion.

Treatment by iron, strychnine, generous diet, &c., was carried out, but in a week the symptoms became more marked; then the patient began to mend, the anæmia and weakness disappeared, and swallowing became almost normal. After a time she went to the seaside, and good accounts of her improvement were forwarded from time to time.

Towards the middle of January unfavorable reports were given; and, on her own responsibility, she stopped the medicine and tried cod liver oil. When I saw her in the beginning of February she stated that she felt weaker,

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had had occasional attacks of faintness and shortness of breath, had once or twice some difficulty in moving the little finger, and had noticed some weakness in the legs.

The following condition was then present:—Paralysis of soft palate, slight ptosis of right eyelid, pulse quick and feeble. Next day no ptosis was to be seen. In three or four days it was again marked, and was accompanied by slight paresis of the corrugator supercilii and frontalis, and in the course of a week the right external rectus, orbicularis palpebrarum, risorius, and zygomaticus were affected, and the laryngoscope showed weakness of tension and adduction of the cords. Fluids no longer regurgitated through the nose, but slight difficulty in swallowing, causing occasional attacks of coughing and choking, indicated that pharyngeal and laryngeal paralysis was threatened. Notwithstanding that active treatment had been resumed, strychnine pushed, electricity, massage, &c., carried out, the patient rapidly went downhill.

She was taken to Melbourne and saw several physicians, some of whom evidently regarded the case as one of simple bulbar paralysis. Dr. Maudsley, however, believed it to be the same as I had come to regard it, viz., myasthenia gravis.

On her return she seemed much worse, and the various paralyses were more marked; attacks of dyspnœa began to supervene, swallowing became impossible; tube-feeding was attempted, but had to be abandoned in favor of rectal; speech became more and more difficult to understand. She again rallied, the power of swallowing returned somewhat, and the attacks of dyspnœa became less frequent and less severe; but she relapsed again, the dysphagic and dyspnœic attacks becoming worse. The patient lost most of the power of moving her tongue for three or four days, and then regained it considerably. For some time she had been having the strychnine administered hypodermically, on Dr. Maudsley's suggestion, but without any apparent benefit. As the dyspnœa became very trying, recourse was had to injections of morphia and inhalations of oxygen, which gave great relief and afforded the patient the euthanasia which was so much desired.

EYE CONDITIONS PRESENT.

1. Paresis of accommodation noticed in the early stage of the disease disappeared in a week or two, returning at a much later stage, and disappearing the last few days before death.

2. Ptosis of right side noticed in February absent next day; in three days' time much more marked, continuing for a week; again almost disappearing, to return later and become so severe that the lid had to be raised by the finger, a few days before death becoming much less; occasional weakness of the levator palpebræ of the left side, never amounting to marked ptosis.

3. Paresis of the right external rectus noticed a little later than the ptosis, disappearing completely in a few days; reappearing in about a fortnight to a more marked degree, subsequently becoming less, and, towards the end of the illness, being very slight and variable.

4. Paresis of corrugator supercilii noticed towards the end of Ferbuary, disappearing, reappearing, finally becoming constant; only noticed slightly on the left side.

5. Pupil reaction, vision for form and color, field of vision, and fundus, normal.

Little attention has apparently been given to myasthenia in works on ophthalmology, although Bielschowsky has shown that in one-third of the cases paresis of the external muscles of the eye is the first symptom, ptosis,

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varying in degree, being especially common. In fact a rapidly appearing and fleeting paresis of the ocular muscles produced on exertion is almost pathognomonic, and is often marked before any of the other symptoms of myasthenia present themselves.

THROAT.

1. Paralysis of soft palate was the first noticed sign of disease, and it continued more constantly than any other symptom, varying, however, very much in degree and apparently less severe towards the end of the illness.

2. Paralysis of pharyngeal muscles, and probably of epiglottis, varying very much in degree; sometimes food swallowed with comparative ease, at other times impossible to do so. Less marked the last few days of life than the preceding three or four days.

3. Paresis of adductors and tensors of vocal cords noticed in February. Dr. Maudsley thought that he noticed weakness of adduction. I had not observed it before her departure to Melbourne, and on her return to Tasmania throat examination gave the patient too much discomfort to continue it; but I think that, with the very variable conditions present, repeated examinations might have confirmed Dr. Maudsley's observation.

4. Tongue was apparently untouched until late in the disease, then it began to feel "clumsy," as the patient put it, and soon got so bad that it could not be protruded from the mouth; then she regained the use of it to a great extent, and retained it in a lesser amount to the end.

The most striking feature in the case was the continual remission and exacerbation of symptoms with the corresponding varying degree of paralysis; in fact, on one or two occasions, as regards the ptosis, one felt almost inclined to doubt one's observations, for a droop which was there one day was gone the next.

The symptoms were all increased at the menstrual period, which was followed by a temporary amelioration. Any fatigue or exertion also seemed to increase the amount of paralysis, which varied not only from day to day but at different times of the day, being usually worse towards evening.

The various reflexes were never impaired, no R.D. was present, no perfect myasthenic reaction was elicited although the reaction to faradism varied on different occasions, and after the use of the faradic current voluntary movement seemed impaired.

Treatment seemed to have no effect on the condition except during the first stage of the disease, and even then it seems doubtful whether the temporary improvement was not rather a stage in the natural history of the disease.

Tubal feeding—which seemed indicated at a later period, and is, I believe, recommended by some—gave rise to the most alarming symptoms, even when carried out with the greatest care; the passage of the tube setting up attacks of dyspnœa and faintness, which became so alarming as to make one desist from further attempts. Drs. Bramwell and Campbell have, I believe, found the use of the feeding tube to be dangerous.

As to the pathology of the disease much doubt exists. The theory of an auto-intoxication has been advanced by some, dynamic changes in the lower motor neurones have been assumed by others. Buzzard has lately described certain conditions which have been found present in five cases of the disease which he examined *post mortem*. These consisted of "lymphorrhages"—the presence of small foci of cells resembling lymphocytes scattered irregularly between the fibres of muscles, or between the cells of a glandular organ. The term "lymphorrhage" was applied to these collections both on account of the character of the cells of which they are composed and because their general appearance was suggestive of a capillary hæmorrhage, with

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the distinction that the cells were not red blood corpuscles. A small empty capillary vessel lined by a single layer of endothelium was sometimes observed in the neighborhood of these lymphorrages. The lymphorrages were found in a smaller or larger number of the muscles of each case, but, in some instances, only after a prolonged search. The ocular muscles were most fruitful in the three cases in which they had been examined. The only other changes in the muscles were presented by the fibres themselves; here and there were seen a few fibres which had become swollen and rounded, some hyaline and granular in appearance, and often showing a tendency to stain a paler blue with eosin or fuchsin. "Lymphorrhages" were also found in the cardiac muscle, thyroid gland, adrenal bodies, and in a posterior spinal ganglion in one case.

The fact that in these five cases similar microscopical changes have been found in the muscles and other organs goes some way towards establishing a definite morbid anatomy for the disease..

NOSE, EAR, AND THROAT DIVISION.

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THE INDICATIONS FOR THE RADICAL MASTOID OPERATION—THE METHODS OF ITS PERFORMANCE AND THE VALUE OR OTHERWISE OF EPITHELIAL GRAFTING.

DR. A. J. BRADY (Sydney), President, in the Chair.

THE PRESIDENT thanked the Executive Committee of the Australasian Medical Congress for the honor which had been conferred on him in electing him President of this Section. He thought the members would agree with him that the short time at their disposal would be better spent in discussing the important practical work which would be brought before them than in listening to any formal address from the chair.

The subject for discussion to day-was "Indications for the Radical Operation on the Mastoid: the Methods of its Performance, and the Value or otherwise of Epithelial Grafting." It was important to be able to judge when the radical operation was necessary. In his opinion, before this operation was undertaken a full trial of the usual treatment for the cure of middle-ear suppuration should be made, and no case should be subjected to operation which could be cured by simpler means. It appeared from the published statistics of operations that some surgeons in London found this procedure more frequently necessary than we do in Australia. Whether this was due to the difference of climate, ear cases doing better in our more genial temperature, or to a difference in surgical opinion and practice, he was not prepared to say. On the other hand, there were some surgeons who had an unreasonable dread of the operation: the bogie which scared them was facial paralysis, following injury to the facial nerve. To a skilled operator this was not a danger sufficient to deter him: those who had not the necessary skill had better let it alone. In his own experience, although he had several times seen a temporary facial paralysis, which had taken from one to two months to fully recover from, he had never had a permanent paralysis follow this operation. The temporary paralysis is easily brought about by even slight contusion of the facial nerve, which may happen if the fallopian canal is destroyed by disease. It is of no importance, as recovery always follows. He asked the

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members present to state their experience on this point. The operation was a valuable one, and was the only means of cure in a certain number of cases. The methods of its performance were fairly well fixed. There was a difference of practice in regard to the formation of flaps, and as to the epithelial grafting, or otherwise; on this latter point there was room for useful debate.

Dr. Webster was called upon to open the discussion, followed by Dr. Lockhart Gibson and Dr. Arthur.

Dr. Percy Webster (Melbourne): In the limited time at our disposal it would not become one to give a lengthy account of the aims of the radical mastoid operation, or to indulge in an extensive analysis of the methods of its performance.

I will content myself with a few remarks having a practical bearing, hoping thereby to limit the scope of the discussion to a few interesting and debatable points, recognising that the members of this Section cannot differ materially on the main features of this important subject.

I would at the outset state that by the radical mastoid operation I mean the removal of so much of the temporal bone as will throw the antrum, the tympanum, the attic, and the meatus into one continuous cavity. The operation is a combination of the two operations described by Schwartze and Stacke; and any modification of this combination is not a new operation, but only a change in some detail, and is at once a tribute to the work of these surgeons and an indorsement of their principles.

The chief modification, and one to which I hope each member will address himself, is that of grafting the post-operative cavity, communicated by Ballance to the Medico-Chirugical Society in 1900.

Towards the end of last year C. Heath, of London, communicated to the Otological Society an account of his method of operating, which appeared to be a new operation; but, on critical examination, turns out to be Stacke or Schwartze-Stacke, at any rate, in principle.

As to the indications for operating, the cases which admit of controversy are those of chronic suppuration of the middle ear, with or without much impairment of hearing, and without acute or a recurrence of acute symptoms. Two men of equal ability and experience may very well, and do often, disagree in such cases. The symptoms are not urgent, and the only circumstances which induce the patient to seek advice are the persistence of the discharge, which may or may not be offensive, the trouble entailed in keeping the ear clean, or the defective hearing. I think in the end the patient himself often turns the scale for or against operation, either being sick of the discharge or anxious at all costs to improve his hearing; or, on the other hand, caring nothing for the discharge, or indifferent about his deafness, and entirely unwilling to take the risks of any operation. Where there are recurring attacks of pain, of vertigo, or general malaise, none of which may be attributed by the patient to his ear, our duty is not merely to advise but to urge an operation. Where, however, without these symptoms, the discharge has existed for twelve months, and the patient has been under our personal supervision and treatment for two months, and we are satisfied that the case is standing still or getting worse, we should point out to the patient the risk of general septic infection and of cerebral abscess, and advise an operation; provided always that, before advising the radical operation, the remains of the drum, the ossicles, and part of the margo tympani have been removed. It must have occurred to all of us to have seen a case cured by the lesser operation when we had fully expected to have had to do the greater. I am aware that many circumstances may occur to induce one to change one's practice in individual cases; but I should like to hear from each member what general rule they lay down for their own guidance in these cases.

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As to the method of operating, my practice is to follow fairly closely that of Ballance, without, however, proceeding to grafting. He makes a large skin flap, the incision following the line of the hair, and thus fully exposes the mastoid. Heath, on the other hand, cuts close to the auricle; this seems to me unsound surgery unless the disease of the mastoid be very limited, and we cannot tell this when making the skin incision. The larger incision does not leave an unsightly scar—the only objection I can find recorded against it.

I am in the habit of using the mallet and chisel, sharp curettes, and, to a limited extent, the gouge. The sets of Schwartze's chisels I have found too small for one to obtain a firm grip of. A chisel should be at least 5 inches in length, and straight to the cutting edge, whether that be flat or grooved. Working only with a burr is very slow, and when worked with a treadle the burr is apt to be unsteady from the alteration in pace: an electrically worked burr has not this disadvantage. Hand burrs are useful for smoothing down the walls of the cavity, a matter which should always be most carefully attended to. In removing the bridge over the iter it can be cracked with a light blow from a high-pitched or blunt chisel, either above or below, and then broken off with forceps. A Stacke's protector is of service, but the same object can be gained by pushing a small roll of gauze under the iter from the antrum.

I have not used any grafts. Very soon after Mr. Ballance's paper appeared I operated on a lady according to his directions, except that I preserved the whole of the posterior wall of the membraneous meatus in the concho-meatal flap. On the tenth day I was intending to open up the wound again for the purpose of grafting, but my patient—who had suffered exceptionally severely from chloroform sickness—begged me not to operate again. On examining the cavity I found healing progressing so satisfactorily that I acceded to her request. Much to my surprise the whole of the surface was healed over at the end of a fortnight. I attributed this successful and rapid result to the fact that I was able to remove all the diseased bone, and to the extensive covering that the inclusion of the membraneous meatus gave to the denuded surface. My further experience has been the same, and I have not seen the necessity for employing a graft.

It has been advised that the cartilagenous portion of the meatus should be removed instead of being folded back, on account of it containing the ceruminous glands, and thereby favoring accumulations of cerumen. I do not think this a serious trouble. In the case referred to I have had to remove cerumen only once in four years.

I believe that delay in healing will not take place if the whole of the diseased bone is removed, and as much as possible of the meatal lining is preserved for covering the cavity. As aids to healing, the application of alcohol, in the form of drops, and of nitrate of silver to granulations, are obvious details of treatment; and the drops may be begun as early as the third or fourth day. The difficulty of manipulating the graft, should one be employed, would appear to be much reduced by the device suggested by Waggett, who says, "I have varnished the prepared area of the limb with new skin, a proprietary article of the nature of collodion, a fairly good imitation of which can, I believe, be made by dissolving celluloid in acetone."

As soon as the antiseptic dressing is removed from he prepared thigh, a few drops of "new skin" are poured over the part and quickly spread with the finger. In one minute this has dried to a firm varnish. A method of dressing which I believe will come into more general use is that of using a layer of thin perforated rubber, which is made to line the wound cavity and packed with little balls of gauze.

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With regard to the opening remarks of our President, it may be that we do not have to perform the radical operation here as often as they do in the old country, on account of our milder climate not favoring such severe cases of aural suppuration ; but one reason leading to the more frequent performance of the radical operation there is the advantage they have over us, especially in hospital work, of a large supply of skilled assistants—a very important circumstance in this operation.

Dr. Lockhart Gibson (Brisbane): The object of such a discussion is, I take it, to bring forward our own beliefs and conclusions, with some of the premises upon which these have been founded ; to indicate the signs and symptoms which would suggest radical interference to us ; to outline why we prefer one method of operation to the others ; and to formulate what we have reason to hope for, as the result of such radical interference.

At the present time aural surgeons appear to be divided into three groups in their attitude to the radical mastoid operation. Members of one group operate only very seldom, members of another group operate on almost every case of otorrhœa which has continued for many weeks, perhaps even without their taking the case thoroughly in hand themselves ; and, between these two, is a group composed of those who advise operation after making a serious and unsuccessful endeavor, prolonged if necessary, to stop the discharge by treatment of and from the nose and naso-pharynx and through the meatus. Members of the first group must often withhold their hands from conferring great benefit ; members of the second group report an enormous number of successful mastoid cases, and suggest to one the thought that a considerable minority of these would have recovered as well without such radical interference. Still, its members belong to the class of practitioner who, by frequent practice, develops a natural into a special expert in operative manipulations, whose patients in consequence run few operative risks, and who may be more easily forgiven for his, at times, unnecessary interference. It is well to remember, however, that all unnecessary operations are bad practice. We shall, I fancy, all agree that every case of persistent otorrhœa which has failed to yield to treatment should be subjected to a radical mastoid operation. Each, probably, will have his own views regarding the length of time during which efforts should be made to stop the discharge, and the methods to be employed. I am a very strong advocate, where radical interference is not urgently needed, for putting the nose and naso-pharynx—the last including the fossæ of Rosenmüller—into as natural a state as possible prior to operation. I favor this in spite of the fact that interference with the nose and naso-pharynx is often so belated as to partake of locking the stable after the horse is out. If, in spite of a healthy nose and naso-pharynx, and in spite of careful attention through the meatus and eustachian tube, we fail to stop the discharge, even although there be no evidence of necrosed bone, a complete mastoid operation should be done. It can hardly be that every case in which suppuration has extended to the mastoid antrum fails to recover with ordinary treatment, but a stubborn otorrhœa without evidence of necrosis most probably owes its continuance to the state of the mastoid antrum and to the attic. It is in the highest degree unlikely that a case with necrosed ossicles, and consequent long standing otorrhœa, would have an antral cavity likely to yield to treatment through the meatus. Still in some cases, after removal of necrosed ossicles, it may be right to persevere with treatment in the hope of avoiding a radical operation. Necrosis of the tympanic walls calls for a radical operation. The existence of headache and of giddiness in a case of chronic otorrhœa calls for operation. I look upon curetting of the middle ear and attic, through the ordinary meatus, as

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likely to be in a majority of cases unsatisfactory in its results, because it cannot be complete. It is attended sometimes by serious risks, because with a curette in the attic we are groping in the dark. If the tegmen tympani be perforated and the membranes be pouting through it, we add little fresh danger and remove great danger by a complete mastoid operation; whereas by curetting such pouting membranes through the meatus, and leaving, as we then must do, an imperfectly disinfected middle ear and antrum, we add fresh dangers while removing none. If, then, we are satisfied that the danger of a complete mastoid operation is less or even not greater than the operation for removing remains of membrane and unnecrosed or necrosed ossicles in a case of persistent otorrhœa, we have next to inquire what we may expect as regards hearing from the one or the other. In my opinion the more complete the operation the better is the hearing result. The explanation of this appears easy. Removal of the membrane through the meatus can only be imperfect; traces are left, cicatrices and some re-growth occur, and the sound waves have to that extent an obstructed passage to the fenestræ. With the drum membrane and ossicles away, useful hearing can occur only if the round and oval windows are free to vibrate, and if the sound waves are unimpeded in their access to them. If either window has been fixed, the other must be very restricted in its vibrations, and consequently the perilymph of the vestibule and cochlea cannot be made to oscillate between the two windows. If with persistent otorrhœa we have indications from the surface of the mastoid of implication of its cavity, or of its cells, a radical operation is of course indicated. If an otorrhœa, instead of being persistent, is only occasional, with intervals of apparently complete health, we would not as a rule be justified in recommending an operation, unless the attacks were frequent and incapacitating. Often attention to the nose and naso-pharynx, accompanied by more fresh air, night and day, and more exercise, will lead to a cessation of such attacks. Still, if in such cases hearing be very defective, operation, I believe, would be good treatment—provided other treatment had received a good trial.

Sometimes attacks of acute otorrhœa, with the pus discharging through a small perforation, have acute mastoid periostitis with suppuration. To do a radical mastoid operation in all such cases would be to often operate quite unnecessarily, as better drainage to the middle ear and a Wilde's incision over the mastoid will generally meet the requirements of the case. Cases suffering from recurrent attacks of otorrhœa and earache whose drum membrane has largely disappeared, whose unnecrosed ossicles are retracted and bunched up with remains of membrane in the posterior segment of the middle ear—and whose hearing in each ear is considerably below that of a successful radical mastoid case—should have the advisability of operation explained to them.

Cases of cholesteatoma of the middle ear and of the antrum should be subjected to a very thorough radical operation.

Another class of case upon which I have been anxious to try the effect of a complete mastoid operation is the class containing cases of extreme deafness with good bone conduction, complete but retracted and thickened membranes, and evidence of exudative catarrh: I speak of cases which are to all intents and purposes deaf mutes. It is, of course, impossible to be sure prior to operation that the stapes is not fixed. But there are some cases that have appeared to me to deserve the chance, which such an operation would give. The necessity one feels for giving an unhopeful outlook has so far deterred the parents of cases from having them operated on. It does not appear to me far-fetched to assume that, in some of these cases, the drum membrane and ossicles are themselves sufficient obstacles to the conduction of sound waves, without there being anchylosis of the foot-plate of the stapes.

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Most aural surgeons appear to be now agreed that where it is necessary to open the mastoid antrum it is right and advisable, except, of course, in some acute cases to make antrum middle ear and enlarged meatus one cavity. Only by this means can we hope to eradicate the disease and give the patient permanent relief, and only by this means can we have the parts permanently open for inspection. The names of the three pioneers in this branch of surgery, Schwartze, Stacke, and Zaufal, have been attached to their operations. More recently Ballance's operation has combined their advantages.

I should like to again pay a tribute to Macewen's work on pyogenic infective diseases of the brain and spinal cord. It is a book which an aural surgeon can scarcely afford to be without. Ballance has modified various steps in his procedure, but the essential steps, it is pretty generally agreed, bring the operation up to date. Charles Heath has lately led a crusade against skin-grafting, and against the considerable exposure of the mastoid which Ballance's operation requires. He has reported a very unusual number of operations. Heath's assumption that aural surgeons have not been sufficiently careful to protect the fenestræ during, and to secure good hearing after, operation, and that they have largely failed to do so, is unwarranted; and it has very naturally been resented. There appears to be little really new in his method, but his skill as an operator appears to be acknowledged and undoubted. I have been extremely satisfied with Ballance's skin incision in the line of the hair, with the large field for operation and with the displacement forward of the auricle and cartilaginous meatus.

Since the publication of Macewen's book on pyogenic infective diseases of the brain and spinal cord I have used burrs driven by a strong dental engine (a reliable motor would be better) for the whole operation, and would only use a chisel and hammer on the head if burrs were not available. A gentler and more accurate instrument to work with than a burr it would be difficult to imagine. Its use reduces the danger of damage to a misplaced sigmoid sinus, or misplaced middle fossa, to a minimum. They are, even when misplaced, exposed so gently that no harm to the non-bony wall of sinus or to the dura mater results. I have had personal proof of this. The use of a burr throughout makes the enlarged bony cavity beautifully smooth, leaves none but the natural irregularities, and helps, I think, in preserving the skin lining of the meatus, whose preservation very materially helps the grafts—if we use these—to quickly line the enlarged bony cavity with skin. I have found the supra-meatal triangle an accurate guide to the antrum. All operators dwell upon the importance of avoiding injury to the facial nerve, and upon the necessity for gentle curetting of the middle ear. Charles Heath is inclined to think that injury is often done to the fenestræ by the curette. It appears to me that the situation of these in their niches makes injury to them with a curette in the highest degree unlikely. By the use of burrs, and of a Stacke's protector or a bent probe in the aditus, injury to the facial nerve may be prevented.

Ballance's treatment of the meatus has given me good results. I am careful, however, to preserve as much as possible of the skin of the bony meatus with the cartilaginous meatus. Ballance incises the floor of the meatus in its long axis well into the concha, and then curves the incision upwards and backwards in the concha to a level with the anterior extremity of the helix. The stitching of the raw surface of the conchal flap to the raw surface of the mastoid flap has prevented any alteration in the appearance of the auricle from that of the other side. Nothing but good appears to result from packing with either strips or balls of gauze. Thiersch grafting of the granulating bony surfaces a week or more after the original operation has given me excellent results, and I should be sorry to discontinue it. The

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difficulty in applying the graft may possibly be much diminished by Waggett's modification of Deansley's suggestion to apply sticking plaster to the skin of the thigh before cutting the graft. Waggett uses a proprietory article called "new skin." It is like collodion, and a good imitation of it can, he believes, be obtained by dissolving celluloid in acetone. "As soon as the antiseptic dressing is removed from the prepared thigh a few drops of 'new skin' are poured over the part and quickly spread with the finger. In one minute this has dried to a fine varnish." The graft can then be easily and quickly cut, and has no tendency to curl. Waggett has found such a graft easy to apply and very satisfactory. The only doubt that would occur to me is whether it would be as likely as an unvarnished graft to apply itself closely to the irregular surfaces of the bony cavity. Its use does away with any necessity for gold leaf, and I am inclined to give it a trial, although pleased with the use of gold leaf. Packing after applying the graft should be very accurate, and probably this would specially apply if "new skin" be used. The tendency is to use little balls of gauze instead of strips of gauze. Waggett attaches a silk thread 3 inches long to each aristol ball, to diminish trouble in removing them. The shed portions of the graft are said to be easily removed on the eighth day, in one or two large pieces, when "new skin" is used. I have found the grafts to shed layers of epithelium sometimes for months after operation. Surgeons differ regarding the advisability of not closing the tympanic orifice of the eustachian tube. Some endeavor, by curetting the orifice carefully and by care in applying the graft, to obtain closure. It can hardly influence the hearing, but on the whole appears to be an advantage. The patency of the tube may, of course, lead to catarrhal attacks spreading upwards from the naso-pharynx, and I have noticed during such a catarrhal attack without noticeable discharge that hearing was distinctly less good than at other times. Where packing has to be changed, either prior to or subsequent to grafting, I have found weak peroxide solution useful for mopping out the cavity, and where granulations are troublesome spirit drops are useful.

The objection that Thiersch grafting is likely to diminish hearing by interference with the fenestræ is, I believe, theoretical. The grafts could only apply a thin layer of epithelium to the membranes in the two windows, and no epithelial graft would live over a fenestra unless it applied itself closely and became part of the membrane. This will not occur unless the membrane of the fenestra has lost its own epithelium.

My own experience is that the graft does not increase the thickness of the membranes in the round and oval windows, and that it does not interfere with a good hearing result. After a radical mastoid operation we have an enlarged meatus—we have, I fancy, a resonance chamber in the large antro-tympanic cavity; and if the fenestrae be free to move, the perilymph is put in motion by the sound waves, though of course less well than if the drum membrane and ossicles are in a healthy state: much better, though, than is possible if with an apparently healthy drum membrane we have the ossicles clogged by exudative catarrh, and acting as a bar to the transmission of sound.

It is quite true that in the great majority of cases the object of operation is to remove a danger to life: but if, in addition to this, we can obtain useful hearing, it makes operation more worth while, even if the other ear possesses normal hearing.

The hearing which, I believe, we have reason to expect in cases whose internal ears are healthy, and where there is no bony occlusion of either window, is distinct speech at 8 yards. I have obtained this in childhood, young manhood, and middle life.

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Dr. G. H. Hogg (Launceston) stated, in response to the President's invitation, that he had had one case of permanent facial paralysis in a patient suffering from tubercular disease of the ear, in whom he removed the whole mastoid process for extensive disease; he had had one case of temporary palsy, developing on the third day after the operation, but making a good recovery. As regards the after results, both in his own and other surgeons' cases, he had very frequently found months or years afterwards that in cases which had been regarded as complete cures there was present a slight fœtid secretion or discharge.

Three years ago, in Hobart, he had condemned operations on the attic through the external meatus as absolutely unscientific, and had been rather snubbed for his views. He was very pleased to hear that Dr. Lockhart Gibson was of the same opinion, and that others had seen fit to change their former views

Dr. S. A. Ewing (Melbourne) said—It is important, in treating chronic suppurative disease of the middle ear, to eliminate nasal and naso-pharyngeal factors.

The minor operation of ossiculectomy is often successful and worthy of a trial. If the radical operation be decided upon, it should be thoroughly done; for example, in long standing cases the dura mater is not infrequently exposed, and a careful search often reveals areas of necrosis not evident unless a free opening be made. The more thoroughly the operation was performed the less necessity there existed for skin-grafting operations. Lake's method of placing small pieces of sponge beneath the bridge of the aditus was preferable to the use of Stacke's protector, which, unless skilfully employed, might injure the facial nerve.

In performing the radical operation, good assistance and light were essential.

In reference to the hearing after the operation, he was of the opinion that though this was often unaltered and sometimes improved, yet it was impossible to definitely predict the result.

The President thanked the openers of the discussion for the capable manner in which they had dealt with the subject. He was glad to know that the experience of those present showed that the danger of permanent facial paralysis following the operation was very remote. The solitary case mentioned by Dr. Hogg, where he had to deal with a destructive tubercular disease of the mastoid, was rather the result of the disease than of the operation.

With regard to the relative merits of burrs or gouges, if an efficient surgical engine such as the S. S. White were employed the bone could be removed rapidly. The drawback was the weight of the machine, which, however, was a very useful instrument, if kept in the private hospital of the operator. Heath's incision gave sufficient room in ordinary cases; but it did not suit so well where there was much œdema of the soft parts. One feature of the incision was good, namely, the manner in which the upper part of the same was carried out. Here, after making the skin incision, the pinna was dissected downwards till the external auditory meatus was exposed—the temporal facia was not opened, and the risk of pus infection of the muscular planes was lessened. This modification could be made in any of the usual incisions, instead of carrying them down directly to the bone through the facia, as was generally done.

Dr. Webster, in reply, said—I am gratified at the way this operation has been discussed, and I think most of us have benefited to some extent from the discussion. Some matters have been introduced which I purposely excluded from my paper; but these have, perhaps, added interest to the subject. Dr. Ewing's remarks were very much to the point, and the com-

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parison he was able to make of the work at two London hospitals in which methods differed has special interest and value. I am glad to find him agreeing with me as to grafting being generally unnecessary, and he properly emphasized the importance of leaving a smooth surface on the bone, a point to which McEwen devotes much pains. His comparison of the operation to that of sinking a shaft is most apt; it brings out most clearly the necessity of a good assistant to keep the field of operation clean and dry.

With regard to Wilde's incision, I do not think it should be mentioned in connection with the radical mastoid operation. I look upon it as an entirely useless proceeding. It was introduced at a time when the anatomy and pathology of the temporal bone was little understood, and when surgeons were afraid of operating on it, and it ought to be given up.

I listened with pleasure to Dr. McCallum, who has had the advantage of working with McEwen. The latter had a different object in view to that which we strive for. He endeavored to obtain a barrier of dense tissue behind the ear to form a protection against further infection when the whole of the disease could not be removed; and his dressing was done behind the auricle. Our object is to leave a large cavity which can be dressed and inspected, and remain open for inspection through an enlarged meatal orifice. McEwen's cases were, of course, almost all those with intra-cranial complications.

Dr. Arthur mentioned pain around the ear as sometimes seen after a radical mastoid operation. I have seen two such cases. In one there is still a little diseased bone in the floor of the middle ear, which might account for the pain; in the other I can find nothing to account for it.

THE DIAGNOSIS AND TREATMENT OF SUPPURATION OF THE ACCESSORY SINUSES OF THE NOSE.

Dr. A. J. Brady, President, in the Chair.

The President said the discussion to-day was on "The Diagnosis and Treatment of Suppuration of the Accessory Sinuses of the Nose." In the treatment of these affections the methods of operation were not yet so fixed as in the case of the mastoid. This particularly applied to operations on the frontal sinuses and ethmoidal cells, which gave room for a good deal of discussion as to the best procedure. In suppuration of the antrum of Highmore, the radical operation in chronic cases was undoubtedly the best. If, in addition to a counter-opening in the floor of the nose, a portion of the anterior end of the inferior turbinal were removed, the patient could wash the antrum with a ball syringe from the nose without the use of any special tube, and the opening was not so likely to close. The wearing of drainage tubes was done away with; and uncomplicated cases, as a rule, ended in speedy recovery.

Dr. Kirkland and Dr. Hogg opened the discussion.

Dr. Kirkland (Sydney): Mr. President and Gentlemen—To be asked to open a discussion on diseases of the accessory sinuses of the nose was an unexpected honor. I feel that the task is one requiring rich experience and frequent opportunity of observing the pathological consequences as well as the clinical signs.

At the very beginning I am encountered with the difficulty of deciding what should come within the scope of a discussion intended to bring forth

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what is best fitted to help us in our daily practice. We are always striving after progress, and if we succeed in adding even one new fact to this department of specialism our discussion will not have been unfruitful. One could traverse the whole domain, beginning with the historical review, the clinical, anatomical, and pathological phases, and end with the surgical; but such, I deem, would be too comprehensive for one paper. I therefore shall confine myself largely to my own experience, touching upon points in correlation thereto.

The etiology concerns both acute and chronic cases; but as the first is the precursor of the latter, they can be considered as having a common cause. There are four hypotheses for the occurrence of antral suppuration. First, that the disease begins as a primary affection of the sinus; second, that the disease begins in structures in direct continuity with the sinus, *e.g.*, in the nasal mucous membrane; third, in the root of a tooth which is in direct contiguity; fourth, that the secretion of the nose, nasopharynx, or some part of the respiratory tract, through sneezing or coughing, is forced into one or more of the spaces, just as secretion sometimes finds its way from the nasopharynx into the middle ear and sets up an acute otilis media. It has been proved that there is no specific bacillus. The discharge may contain staphylococci aurus, albus pneumococci, and even streptothrix. I suspect that the pneumococci and influenzal bacillus are perhaps the most active factors in the production of suppuration of these cavities. Luc mentions the case of a student attending the *post mortem* of a patient dead of pneumonia, and who suffered afterwards with suppuration of the frontal sinus from which a pure culture of the pneumococcus was obtained. At the last Congress I showed that, in a series of *post-mortem* examinations of fifty cases dying of pneumonia, twenty-five of them had suppuration in one or more accessory sinuses.

The shape of the nose and the character of the constitution are mostly responsible for acute cases becoming chronic; the strumous diathesis is the one which favors pan suppuration. I take as granted that many cases, acute in origin, recover within a period varying from one week to three. Those becoming chronic have usually some anatomical or pathological stenosis of the ostium.

The theory of dental origin of maxillary empyemata is one which requires our earnest consideration. Tilley is a strong advocate of this theory, and dentists, acting upon it, have ventured in a good many cases to open and drain the cavity. In my experience only a very small number are due to this cause, and these I would distinguish as pure empyemata, having very slight pathological degeneration of the lining membrane, and capable of being treated through the maxillary process. I would here enter my protest against dentists undertaking work quite outside their sphere, and which is, in the nature of things, beyond their therapeutic capabilities. I am sure those who do usually forfeit the confidence of their patients, as the patient drifts into the hands of the specialist finally.

The pathological contents found in maxillary antra may be enumerated as serous fluid with a cyst wall, thick creamy pus, inspissated pus, polypi, new growths, a loose tooth, and sequestrum. In many cases the pus in the antrum is associated with nasal polypi and hypertrophy of the middle turbinate. We meet with cases which have all the characters of antral suppuration—pus in the nose, nasopharynx, absence of the light spots under the eye, &c., and which, in being opened, show no obvious suppuration. In some of these cases I have curetted a large amount of polypoid tissue, representing a degeneration of the lining membrane. One is forced to ask the question, in the absence of microscopical examination, are these cases of the same nature as those found with unmistakable evidence of suppuration?

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There are also some cases which, on being opened, leave one in doubt as to the correctness of the diagnosis—no pus, no polypoid tissue; yet, a few days later, secrete pus. Are those cases of drained empyemata, or has the antrum become infected through an operation conducted on perfect aseptic lines? I believe they are empyemata found empty at the time of operation. With reference to the diagnosis, the symptoms are so clearly understood that I need only refer to cases which cannot be diagnosed if we rely on the ordinary classical symptoms. There is a class of cases to which Dr. Brady drew attention in an article in the *Journal of Laryngology*, viz., those in which no secretion is seen in the anterior part of the nose, but in which, on a rhinoscopic examination, a muco-purulent secretion is visible extending from the lower aspect of the middle turbinate and running over the soft palate. We explain those cases by the natinal ostium having a situation more posterior than usual. My experience is that these cases are less purulent in their contents—possibly due to a larger opening.

Having decided in a given case that there is suppuration of a chronic nature going on, we next ask the question, what is the cause? If it is a tooth, remove it, and make a wide opening for drainage through the socket and carry out antiseptic irrigation for a time. If this fails, the conclusion is arrived at that there is extensive degeneration of the lining membrane, for which all operations singularly fail except a radical one. The patient is more or less annoyed that a second operation is necessary; and this has forced me to examine the symptoms carefully from a differential point of view, and in many cases perform at the outset a radical or Caldwell-Luc operation. When this is done the results are excellent, and in 90 per cent. of the cases a complete cure is obtained within a short time. I assume that all pathological conditions in the nose which would retard a cure have been in the first instance removed. The disadvantages of this operation may be summarised in the greater loss of bone in the anterior wall and the ensuing numbness, which generally disappears in a short time. There is a point in this connection to which hitherto no attention has been drawn, viz., the interference with the nerve supply of the teeth. Patients have pointed out that the teeth after this operation feel dead in the immediate neighborhood, and I am informed by a capable dentist that the teeth really decay later on; personally I have not observed such to take place, and if this really happens it would be a dreaded drawback to the performance of this particular operation.

The Caldwell-Luc operation is the most satisfactory operation, both for the doctor and the patient. It involves a greater degree of destruction of the bony wall of the antrum, it inflicts a greater shock to the patient; but, on the other hand, it secures to the patient, in most cases, a lasting and permanent cure. In this operation an opening is made with a hammer and gouge, a trephine, or a bar worked by an electric motor, large enough to admit the forefinger into the cavity, which can be used to feel every nook and corner. The maxillary antrum is irregularly pear-shaped, with the apex above. As this point has not yet received a name, I would suggest that it be called the "orbital recess"; the extension of the cavity towards the malar bone might be called the "malar recess"; and the space above the maxillary process might be called the "maxillary groove." The next step in the operation is the curettage of the lining membrane. This should be thorough, leaving no space with a thickened degeneration of the membrane untouched. Good light and frequent cleansing are necessary for a thorough inspection. The next step is a counter-opening into the nose, which is intended to remain more or less permanent. Small openings soon close up through the continuous effort on the part of the tissues to return to their former position. Whether this opening should be situtated in the inferior meatus or represent an enlarged natural ostium is

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much a matter of individual preference; no doubt nature placed the opening high up for a good reason. It certainly is not in the best position for drainage, which is the chief consideration for the surgeon. Placed in the inferior meatus it would be more open to infection, and would be covered by the inferior turbinate; nature had therefore no choice in her selection. Situated as it is, high up in the cavity of the nose, it affords an easy egress for the secretion from the tubular glands on the nasal wall, propelled as it is by the ciliated epithelium of the lining membrane. The lining membrane, as Lennox Brown points out, is not a true mucous membrane: it is largely composed of connective tissues. The opening in the nasal wall can be made by trephine or punch forceps. I introduced a punch forceps two years ago for this purpose; it can also be used at the same time after puncturing a portion of the inferior turbinate. At first I had an almost sacred regard for the inferior turbinate, but I am disposed to now think that its partial removal in this operation outweighs in advantage its functional loss. I have abandoned packing after the operation as unnecessary and somewhat harmful to the patient. The subsequent treatment consists in irrigating the cavity through the nasal opening with the aid of an eustachian catheter. As a rule the cure is established within a few weeks. In some cases I have found this even unnecessary, as no pus was present after the operation.

To sum up the different operations recommended for antral suppuration: First, maxillary process drainage; second, canine fossæ drainage; third, Caldwell-Luc operation, or radical operation.

The frontal sinus, by virtue of its communication with the nose being a small opening, is very difficult to cure. Coupled with this anatomical difficulty the sinus is frequently large in size, extending outward towards the outer canthus of the eye, and not infrequently downward towards the orbit; now and again it is divided up into compartments, the division being incomplete. The median division between the two sinuses is occasionally perforated, leading to bilateral infection. In addition to this we have, in most cases, involvement of some or all the ethmoidal cells. It is easy to see that, however much we enlarge the infundibulum opening, drainage can only be very imperfect. It is frequently recommended that a large opening be made through the infundibulum; and no doubt, if this could be done and maintained, our efforts would be in most cases successful in producing a cure. Nature asserts itself in trying to re-establish an opening of the former size: granulations spring up, and, unless we are constantly watching the progress of the cure, our efforts are frustrated. I take it for granted that most men recognise that some cases are best left alone; and, unless we can promise a cure, are we justified in recommending an operation? In most cases a cure could be obtained with the exercise of great care on the part of the surgeon in the after-treatment, combined with patience on the part of the patient. In the case of men, time is of great consequence; and in the case of women, deformity or scarring should be avoided as much as possible. What are the conditions which determine our decision in a case of frontal sinus suppuration? I am guided by the condition of the infundibulum; if it is large, and can be kept open and the whole of the ethmoidal cells can be removed, there is a reasonable prospect of cure. If the case is very chronic, and the patient suffers intermittent pain, it is probable that polypoid degeneration of the lining membrane has taken place, leading to temporary retention of the discharge. Here we are confronted with the possibility of extension of the disease towards the brain. An operation in this case would be clearly indicated, as it would provide for drainage, give the patient a chance of a cure, and lead to nothing worse than a small infra-orbital scar. If we are consulted in a case, say, of a patient about middle-age with bilateral frontal

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sinus suppuration, with no hindrance to drainage, and no inconvenience beyond the discharge, what should the surgeon advise? My advice in a case of this kind is to tell the patient that an operation would be necessary only in the case of the supervention of pain, another symptom indicating a possible extension beyond the sinus. It is surprising how many cases go on for years without danger arising. I know of no data telling us what percentage of cases terminate fatally through extension to the brain, or by the generation of a new disease of the chest or blood. Constant antiseptic cleansing of the nose may keep some cases quite comfortable for years. I am disposed to think, however, that in the end these conditions must shorten life. The consequences of a chronic suppuration of this kind are many, both locally and remotely. In the nose it produces hypertrophy of the structures over which the pus flows, polypi alteration of the ethmoid in the direction of rarefaction or condensation through a chronic osteitis: remotely it produces anæmia, leucocytosis, gastric derangement, bronchiectasis, and pneumonia. Conservative methods of treating sinus suppuration nearly always fail—such as politzerisation, syringing through the infundibulum, and removal of nasal obstructions. The operations in vogue are Ogston Luc, Kuhnt's, Jansen's, and Killian's; but I think most men confine themselves to the Ogston Luc, the Kuhnt's, or Killian's. The latter I have not yet performed. The Ogston Luc operation consists in making a curvilinear incision under the eyebrow extending from the naso-frontal suture to the supra-orbital notch, the knife cutting to the bone. The periosteum is raised and a large opening is made in the frontal wall of the sinus by means of a gouge trephine, or burr (personally I always use the gouge). The cavity is carefully curetted, the infundibulum enlarged, all granulation tissue removed by a piece of gauze passed into the nose to which a sawing movement is imparted. The sinus is most carefully antisepticised with pure carbolic acid and packed with gauze passed down into the nose. Preparatory to the operation all polypi in the nose are removed and the ethmoid cells cut away by Grunwald's forceps; stitches are inserted, leaving, however, a free opening through which a piece of gauze is passed. The gauze is removed on the second or third day, the sinus is irrigated with HgCl. lotion, formalin, or a weak solution of Ag. NO_3, and thereafter packed daily. This operation has undergone many changes in its solution. Some years ago the textbooks advised the insertion of a drainage tube; this was found more or less unsatisfactory, producing a copious discharge, growth of granulation in the neighborhood of the infundibulum, and a free purulent discharge in the nose. The next change was to do away with the drainage tube and close the wound at the time of the operation. My colleague, Dr. Brady, performed this operation on many occasions: he tells me that it was frequently successful in curing the discharge. It, however, has many disadvantages. Firstly, it may fail, and this subjects the patient to an operation without any good results; secondly, it favors the occurrence of a septic osteomyelitis, which is almost invariably fatal; and, thirdly, should it fail, the sinus refills with pus, which now and again causes the weak cicatrix to yield and the pus to trickle over the face. The operation which Tilley recommends is the open one, with the insertion of a drainage tube or silver wire which is removed about the third week. The soft tissues are pressed against the posterior wall and the sinus closed. The essential conditions for the cure of chronic suppuration of the frontal sinus are, firstly, complete removal of all pathological conditions of the ethmoid cells undertaken anteriorly to the external operation; secondly, free drainage into the nose after the operation, and thorough removal of all diseased membrane within the sinus itself.

In what way is the removal of the ethmoidal cells together with polypi accomplished? Tilley and Lask recommend the use of Meyer's ring knife.

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This will succeed in certain cases where the higher region of the nose is fairly wide; in some cases even a small knife is too large, and in these cases I find Hajek's oblong curette more useful. We are, however, I think, exposing our patient in the use of either of these instruments to an unnecessary risk of septic phlebitis. It is clear that curettage in the presence of pus incurs a risk of some septic organisms finding their way into one of the veins. For this reason I more frequently use Grunwald's forceps, in the use of which I think there is less chance of accident. From Tilley's account of suppuration of the frontal sinus one is led to the belief that a cure is easily accomplished provided attention is given to all the details. A statement later on in the same chapter tells us that he has seven cases under his care for which he does not recommend an external operation. This, I think, shows that he is not quite as confident of producing a cure as the earlier part of the book would make us believe.

Grunwald's method of opening the sinus through Ogston's incision, followed by irrigation, takes from six weeks to six months to produce a cure. The great disadvantage of this method is the growth of granulation tissue in the infundibulum, and the constant tendency of the wound to close. Whatever method of an open kind is undertaken, it is absolutely necessary that the after-treatment should be carried out by the operator himself. The dressing of these cases requires special skill, and to leave the subsequent dressing to an assistant is to court failure. I have endeavored to produce a cure in these cases by filling the cavity with paraffin and iodoform after an interval of two or three weeks of aseptic irrigation. In only two cases have I succeeded. Lately I filled the sinus at the time of operation, but the whole mass was extruded within ten days. I think something in this direction is possible, and I hope to experiment with it a little further. Success achieved by a method of this kind would be a great boon to the surgeon. We sometimes obtain a speedy cure quite unexpectedly. Not long ago I operated upon a woman for frontal and maxillary sinus suppuration. The gauze was removed on the third day and the wound closed. The patient thereafter disappeared. Two months later she returned with a slight affection of the ear. I then found that a complete cure had taken place in both cavities, there was no pus visible in any part of the nose or naso-pharynx; and, as I have seen her several times since with an aseptic nose, I regard the case as a complete cure.

The most satisfactory operation is the radical operation, in which complete obliteration of the sinus can be obtained. This is the operation of my choice in any case suitable for its performance. We hesitate to do this on women more especially, and in men to whom a conspicuous scar or deformity is a disadvantage. The cases in which it is really the operation *par excellence* are those in which the sinus is small. The after-treatment of these cases differs in no way from the other cases—a gauze drain inserted down the infundibulum until complete coaptation of the surfaces of the soft parts and the posterior wall has taken place. In this operation, in my first case, I erred in not removing sufficient bone in the region of the nasal opening, so that I found the upper part healed quite well, and left a small canal below which continued to secrete longer than if it had been partially obliterated.

English writers content themselves by dividing the ethmoidal cells into two sets—an anterior and a posterior. On the Continent a much more elaborate division is attempted, especially by Killian. For practical purposes the English classification is all that is desired, but perhaps, from a scientific view, the continental is to be recommended. In the higher mammals the nose is more highly developed than in man, and the olfactory sense is sharper; so that the nasal organs of man have undergone retrograde changes. Killian

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distinguishes at least four turbinates, together with accessory ones. For surgical purposes we can content ourselves with the division given above if we remember a third set, viz., those in connection with the frontal sinus or fronto-nasal cells. Disease may involve these cells in suppuration, or give rise to a mucocele. I have also found these cells much extended without any abnormal contents, representing a form of rarefying osteitis. The pathology of these cases is more or less unknown. The patient presents in some cases an extended nasal bridge, complains of continuous pain—which is probably due to pressure on the nerves—and yet removal of these cells, which opens up a wide expanded attic, fails to give relief. Dr. Brady will remember a case of this kind in the Sydney Hospital, which was under my care in the earlier stages, and later on under his treatment. We meet with cases in which this state of rarefaction is limited to one cell. I have on many occasions, when removing the middle turbinate, found a large empty cell. We meet with cases in which no abnormal state is present in the ethmoid, and in which pain is the only symptom complained of. After excluding all reflex conditions likely to produce pain in this region, what is our diagnosis? There is no pressure of the middle turbinate against the septum, and nothing really within the range of sight to explain the pain. I have sometimes wondered if there is some hidden cell producing pressure; we do meet with unexpected conditions if we are patient enough to search into the hidden recesses. Killian introduced his long specula for this purpose. The most important and interesting condition is that of suppuration of these cavities; suppuration of these cells brings with it polypi, and more or less osteitis. The removal of these conditions is a necessity for the patient's comfort, and to prevent further extension; continuous suppuration in this region more than any other produces morbid conditions of the pharynx and larynx. The pharynx is not infrequently glazed over with a thick layer of secretion, which, in time, tends to produce a pharyngitis sicca. As it trickles down the larynx it begets a chronic laryngitis with extensive changes in the vocal chords; its remote effects go further in some cases by producing bronchitis, and even bronchiectasis. Shall we remove these cells piecemeal by Grunwald forceps, or adopt Lack's heroic method, which he says is unattended with danger? Personally I cannot believe an operation performed in the dark, as Lack's is, with very free hæmorrhage and only the sense of touch to guide you, as one free from great risks. It is true that I have performed the operation many times with no untoward result; but, rationally, I believe the operation by Grunwald's forceps conducted within the range of vision a safer and quite as satisfactory operation. A curetting operation is more likely to open up veins in a lacerated state, into which septic organisms can pass, than a cutting operation. In the cutting operation no pressure is made in an upward direction. I have a case at present under my care in the hospital in which access to an ethmoidal cell could only be obtained by an external operation. The ethmoid cells were all removed, pus continued to flow, and the patient complained of pain near the inner canthus. I suspected the frontal sinus, which, on exploration, was found absent. I then embarked on a voyage of discovery lower down, and found a small cell full of pus, which I regarded as an ethmoid cell; when this was opened the pain disappeared.

Dr. G. H. Hogg: Mr. President and Gentlemen—First of all permit me to express to you my sense of appreciation of the honor you have done me by inviting me to open this discussion; together with the diffidence I feel in venturing to do so in the presence of yourself, Sir, a rhinologist of more than Australian reputation, and of the other distinguished representatives of this section of surgery.

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Clinically, as well as anatomically, the accessory sinuses of the nose may be divided into two groups:

1. The anterior group, consisting of the maxillary and frontal sinuses and anterior ethmoidal cells, all of which have openings leading into the middle meatus of the nose—*i.e.*, below the level of the middle turbinal:

2. The posterior group, consisting of the posterior ethmoidal cells and sphenoidal sinuses which open into the superior meatus—*i.e.*, above the level of the middle turbinal; and this classification is, as will be seen later, valuable from the point of view of diagnosis, for according to the position which the pus occupies in the nose the rhinologist forms his conclusions. Clinically, too, suppuration has been classified as acute and chronic—as suppuration with retention of the inflammatory products, and suppuration with free drainage.

In the present discussion, which deals with diagnosis and treatment, it seems to me advisable to adopt the acute and chronic classification. Acute suppuration is met with most frequently as a complication of the exanthemata; as a sequel to influenza it is not uncommon, and demands a moment's notice.

In the maxillary antrum its presence may be indicated by general malaise, rise of temperature, perhaps rigors, pain in the cheek and forehead, perhaps tenderness over the cheek and gum, stiffness in the nose, with muco-purulent discharge. A free discharge of pus from the nostril of the affected side may occur with relief of all the symptoms; sometimes eye symptoms, such as pain on the eyeball, photophobia, &c., may be present.

When the frontal sinus is affected, the symptoms may be much the same; the pain may be felt more in the forehead, and may be accompanied by a feeling of oppression and fulness over the eye, tenderness on the floor of the sinus, with sometimes redness and œdema of the upper eyelid. The pain or discomfort may abate or disappear with the escape of pus into the nose. I have met with several such cases following influenza; and know of one case which passed from the acute into the chronic stage, and which subsequently had a fatal termination from perforation of the posterior wall of the sinus and abscess of the frontal lobe.

The ethmoidal cells and sphenoidal sinus may also be affected; such cases, though much less frequent, are accompanied by similar general symptoms. In addition, eye complications and cerebral symptoms may develop. In the sphenoidal cases recorded by Schäffer, the headache was intense over the vertex and occiput, and giddiness was marked. Acute suppuration of the ethmoidal cells has been met with as a sequel to gonorrhœa, exophthalmos and orbital cellulitis developing. In these acute forms the methods of examination by rhinoscopy, trans-illumination, &c., are to be applied as described in a later section.

Treatment will be that of the causal disease and the concomitant nasal inflammation. Should the suppuration not subside, or threatening symptoms develop, operative measures will be required.

Chronic Suppuration.—The general symptoms are:—(1) Purulent discharge, with offensive taste or smell; (2) nasal obstruction; (3) pain and tenderness; (4) eye symptoms; (5) cerebral symptoms; (6) throat and chest symptoms; (7) gastric symptoms; (8) neurasthenic symptoms.

Purulent discharge may have lasted weeks, months, or years; may be continuous or intermittent, coming once or twice a day, and is often affected by the patient's posture. It is usually unilateral, but may be bilateral, even when only one sinus is affected, as the pus may pass from one side of the nose into the other. The discharge may be muco-purulent or purulent, of a yellow or greenish color (it is said to be greenish when it comes from the frontal sinus, but this is not diagnostic). It is often fœtid, and the patient complains of the sweetish fœtid taste and smell (contrast it with ozæna); it may come from the

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nose or run into the nasopharynx, and be expectorated. Sometimes there may be little or no discharge; I have seen suppuration of the antrum where there was neither discharge nor accumulation of purulent fluid, the pus apparently drying and forming a fœtid deposit on the antral walls, the patient complaining of the offensive smell and taste. Possibly some such cases correspond to what is called "ozæna of the antrum" by some writers.

Nasal obstruction may be due to the presence of enlarged turbinates and congestion of the nasal mucous membrane, or, more frequently, to the presence of polypi.

Pain and tenderness may or may not exist; if present, it varies very much in character and degree, and may be continuous or intermittent, being relieved by the free escape of pus. In maxillary trouble it is described variously as like toothache, neuralgia of the face, or frontal headache. Possibly the presence of more or less severe and continuous neuralgic pain in some of these cases may be due to irritation of the infra-orbital nerve, which may become exposed by disease of the bony canal or by natural defect thereof. In frontal cases supra-orbital headache may occur, or the headache may be diffuse or even occipital; in ethmoidal disease the pain may be frontal, or deep-seated, and referred to the back of the orbit; while in the sphenoidal cases vertical headache has been described as characteristic; but occipital and diffuse headache, as well as pain behind the eyeball, may occur. The seat of pain is seen, therefore, to be of no constant value in fixing the site of the sinus disease, although, combined with other symptoms, it may be helpful in diagnosis.

Eye symptoms and complications are rare in maxillary disease *per se*, and I have never met with them; but upward displacement of the eyeball, œdema of the lower lid, lachrymal obstruction, &c., are said to occur. In frontal disease the upper eyelid may become œdematous; there may be swelling at the inner and upper angle of the orbit, and orbital abscess may develop, perforation taking place about the middle of the upper eyelid. In the posterior ethmoidal and sphenoidal cases, eye complications are more frequent; orbital abscess, sudden loss of vision from compression of the optic nerve, optic neuritis, thrombosis of the central vein of the retina, cavernous sinus thrombosis, exophthalmos, may occur; the third, fourth, and sixth cranial nerves may be pressed on at the sphenoidal fissure, and various paralyses result.

Brain symptoms and complications are very rare in maxillary disease, although they have occasionally been recorded. When they occur in the other forms of sinus suppuration, they may be caused by direct infection through perforation of the bone or through the vessels of the diploe.

In frontal disease abscess of the frontal lobe is the most frequent, infection usually taking place through a perforation of the posterior wall of the sinus. In this, as well as in ethmoidal and sphenoidal disease, orbital abscess may also cause brain trouble.

In ethmoidal disease abscess of the frontal lobe, extra-dural, sub-dural abscess, meningitis may occur; while in sphenoidal disease these, as well as thrombosis of the cavernous, petrosal, and longitudinal sinuses, may follow; in some cases giddiness, tinnitus, and nausea have been prominent symptoms.

Throat and lung symptoms—cough, huskiness, and irritation of the throat—may be caused by the escape of pus into the naso-pharynx, while the cough and profuse purulent expectoration have in some cases given rise to a diagnosis of phthisis, bronchiectasis, &c. Asthma has occasionally been associated with sinus disease.

Gastric symptoms, sickness, indigestion, foul tongue and taste, loss of appetite, &c., may be complained of.

Neurasthenic symptoms have been most marked in several cases which I have met with—lassitude, mental depression, inability to do mental work, &c.

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—and have persisted even after the local trouble has been practically cured. These symptoms have been attributed to the septic absorption from the purulent products of the sinuses; but in one or two cases of my own the quantity of pus has been so small as to be almost a negligible quantity.

Objective Phenomena.—Rhinoscopic examination may reveal redness and swelling of the mucous membrane of the nose; and it is often useful to reduce this swelling by the application of cocaine and adrenalin, so as to allow of a better examination. Polypi may be present, and may have to be removed to allow of a proper view; and, for the same reason, it is sometimes advisable to remove the anterior part of the middle turbinate. Such measures assist the examination, and serve often to aid the preliminary treatment.

Maxillary Antrum.—Pus can be seen in the middle meatus anteriorly; on wiping away the pus, and making the patient hold his head over so that the suspected side is uppermost, the pus will be seen to reappear. What is known as Fränkel's test may also be tried: this consists in cleaning out the nose, leaning the head forward so that the forehead is down for a few minutes, and re-examining. The reappearance of pus is in favor of the antral source. This test is not conclusive, however, Greville MacDonald stating that this posture test does not exclude frontal sinus suppuration. Sometimes, where accessory ostia exist, little or no pus may lie in the middle meatus, but may drain back into the naso-pharynx.

Trans-illumination, the details of which are too well known to require mention, is of value when considered in combination with other symptoms and physical signs. Taken by itself it is not an absolutely reliable diagnostic phenomenon. Turner has pointed out how non-illumination of the antrum is due more to infiltration and thickening of the mucous membrane of the antrum than to the pus contained therein, and likewise how the amount and consistency of the pus bear no relation to the density of the shadow. (I was much impressed once by a case where, with typical non-illumination, there was only a suspicion of pus present.)

Pressure and percussion may reveal tenderness over the cheek or gum, or diseased teeth may be found in the antral region of the mouth.

Exploratory puncture may be made through the socket of a carious tooth, the canine fossa or the inferior meatus of the nose, and is really the only single positive means of settling the problem. It will be seen, therefore, that a careful consideration of symptoms and physical signs will usually allow of a reasonably certain diagnosis being made. Briefly stated, a unilateral purulent nasal discharge, with the pus lying in the middle meatus of the nose, and answering to the posture tests, is sufficient reason for diagnosing antral disease and for positively proving it by an exploratory puncture.

Frontal Sinus.—Rhinoscopy may reveal much the same condition as in the antral cases; and, as has been seen, posture tests are not absolutely diagnostic. Pressure may reveal tenderness over the floor of the sinus. Trans-illumination is of little or no practical value; as Turner has clearly shown, it is the exception rather than the rule to find opacity of the diseased sinus, and even where there is absence of illumination it may be due to absence of the sinus, to a thick-walled sinus, as well as to disease of the cavity. In doubtful cases, therefore, it is advisable to open the antrum. If there is no pus in it we can eliminate the antrum; if, however, it contains pus, after syringing and carefully cleansing the antrum, we examine the nose in order to see whether pus is running into the middle meatus. If this is the case the frontal sinus is probably involved. Frontal sinus suppuration, however, is complicated often, not only with antral but also with anterior ethmoidal suppuration. Suppuration of the anterior ethmoidal cells is frequently associated with frontal and

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antral disease. The removal of the polypi, which are often present, and of part of the middle turbinates, with careful use of the probe and curette to remove any carious bone, will enable the source of the pus to be detected.

Posterior Ethmoidal Suppuration.—In every unequivocal case, according to Greville MacDonald, the pus does not flow over the posterior naso-pharyngeal wall—which, indeed, can only happen if the patient lies on his back; it may be seen in the superior meatus, between the middle turbinate and septum. According to other authorities it often may be seen in the post-nasal space; and it must be remembered that the pus may find its way into the middle meatus.

In sphenoidal disease the pus obviously flows over the posterior naso-pharyngeal wall (G. MacDonald), while the only position in which it gains access to the nasal fossæ is with the patient lying prone or bending the head forward.

Other rhinologists refuse to accept these views, and it is, I think, quite possible for pus to find its way into the superior meatus. The discharge is sometimes liable to dry and form crusts, and cases may be confused with naso-pharyngeal catarrh, complicated by crust formation.

An important point to keep in mind in the diagnosis of sinus suppuration is the frequency with which more than one sinus is affected, as it tends to further complicate an already complex subject. It is only by a careful consideration of symptoms and physical signs (it is often advisable to submit the patient to repeated examinations), and, if need be, by a process of exclusion of different sinuses by exploration, that an accurate opinion can be arrived at.

Treatment.—In the treatment of chronic suppuration of the maxillary antrum, two methods may be followed:—(1) Treatment by lavage; (2) the radical operation.

Treatment by lavage consists in the opening of the sinus through the socket of an extracted tooth, followed by a continued course of irrigation of the antrum by various antiseptic and astringent lotions; any nasal or dental complications, such as polypi or caries, being attended to. This lavage may have to be continued for weeks or months, and in the simpler cases may give good results; but where complications are present—such as nasal polypi, antral polypi, diseased bone, &c.—or where the disease is of long standing, the radical operation is to be preferred. Into the details of this and other operations I do not propose to enter, as they are too well known to you to require mention; suffice it to say that a very free opening should be made sufficient to admit of exploration by the finger, and that all diseased bone, membrane, and polypoid growths should be carefully removed by the curette.

The variation in the size and shape of the antrum, due to the varying degree of approximation of facial and nasal walls and of absorption of cancellous bone during the period of growth, must be kept in mind in these operations, as well as the position of the infra-orbital canal with its contents, and the presence of the recess above the canal, where disease may escape the operator's attention. The radical operation gives good results in many cases, but occasionally fails to cure the disease. Amongst others I may mention one case where four, if not five, operations had been performed by different surgeons, with years of intermittent applications and dressings; and when the patient died, ten years afterwards, of another disease, the antral trouble still existed.

The operation through the mouth with a counter-opening through the inferior meatus of the nose (Luc), with or without the subsequent suture of buccal opening, seems to be of value in some cases. The possibility of disease of other sinuses must be kept in mind, as failure may result through the implication of these sinuses, and the reinfection of the antrum.

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Frontal Sinus Suppuration.—All nasal disease must be removed, and, in addition, it is advisable to cut away the anterior portion of the middle turbinate, to allow of freer inspection, access, and drainage. Some surgeons recommend that, after this has been done, the diseased ethmoidal cells be broken up by the curette or forceps, and the infundibulum so enlarged. A frontal sinus cannula can be subsequently passed, and lavage carried out. The passage of the cannula is often easier in theory than in practice; even if practicable, treatment by lavage has to be carried on for a prolonged period.

In chronic cases this treatment may fail, and the question of external operation arises. It must be remembered, moreover, that suppuration of the frontal sinus is not altogether free from risk—septic meningitis, inter-cranial abscess, orbital cellulitis, &c., being possibilities; although what percentage of cases develop dangerous complications is, as far as I am aware, unknown. The fact that the external operation may leave a more or less marked scar must also be kept in mind, and must be explained to the patient—more especially if that patient is a woman.

The supra-orbital incision is perhaps the best. If, on opening the sinus, the lining membrane seems healthy, it may be sufficient to wash out the sinus and effect free drainage; should, however, the membrane be diseased, more of the anterior wall must be removed, and the sinus wall carefully curetted. A probe is to be passed from the sinus into the nose as a guide, and the anterior ethmoidal cells removed by the curette; by this means the fronto-nasal passage can be enlarged, and made to allow of free drainage. The external incision is not to be closed completely, but room left for the end of a gauze packing placed in the sinus.

Numerous modifications of the operation exist—thus, a vertical incision is proposed by some, a median by others. Luc completely closes the external incision, and brings the end of his gauze packing into the nose: Jansen opens the floor of the sinus by an incision below the supra-orbital margin, and claims better access to the ethmoidal cells as the advantage of his method. Another advantage of Jansen's method is the absence of risk of opening into a healthy sinus of the opposite side, where the septal obliquity is marked. Kuhnt advocates complete removal of the anterior wall; which may be possible in small sinuses, but would cause great deformity if the sinus was large. Milligan suggests that, in uncomplicated cases, the infundibular passage be closed by means of ivory nails, thus cutting off all communication with the nose. The frontal sinus thus becomes a bony cavity communicating with the air by the supra-orbital incision only, and may be allowed to granulate up.

Skin-grafting, after the methods employed in mastoid cases, seems to me to be possibly a valuable adjunct to some of these operations on the frontal sinus. Whether they have been tried, I am not aware; if not, they are worth trying.

It is important to bear in mind in all operative work in the frontal sinuses their very variable extent and shape, the septal obliquity which may result in the wrong sinus being opened, the imperfect partitions which may subdivide the sinuses and produce irregular recesses and pockets, where disease may escape notice.

Into the details of treatment of ethmoidal and sphenoidal disease I shall not enter, as Dr. Kirkland deals with them in his paper. Suffice it to say that it consists in removing any polypi which may exist, together with the middle turbinate, then carefully curetting and punching out the diseased bone, so as to allow of free drainage; and that any operative measures in the ethmoidal or sphenoidal cells should be undertaken with the greatest care.

It will be seen from my preceding remarks that the treatment of suppuration of the necessary sinuses is hedged round by not a few difficulties. Patients,

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therefore, should not be buoyed up with too confident assurances of speedy cure, for, in the words of Milligan, the intricate arrangement of the sinuses, the difficulty encountered in securing at any one operation a complete eradication of disease, and of maintaining free drainage, together with the risks of reinfection, are factors which militate in no small measure against a successful issue.

Dr. Barrett said that the discussion on the treatment of empyema of the maxillary antrum had interested him extremely, as, for some reason or another, he had had a very large number of these cases pass through his hands. What had most impressed him was the fact that empyema of the maxillary antrum, discharging foul pus into the nose, might exist for months, or even years, without causing any very grave symptoms. Poor health it certainly caused, digestive troubles, loss of weight; but he had not been able to discover one case in which a fatal result could be directly attributed to it. It had been brought home to him in this way, that he had urged patients to have an operation done, when they declined to have anything whatever done—even washing out through the nose. They had continued to follow their business, and, so far as he was aware, none of them had lost their lives. The operation he usually practised in the first instance was to drill a hole through the alveolus—preferably of the second or first molar, with a cylindrical saw, driven by an electrical motor or a dental engine. The operation did not usually require any anæsthesia, and, if the appliances were in good order, took but a few seconds to complete. A cylinder of bone was removed, and the antrum could be readily washed out. Treated in this way, the bulk of the cases got perfectly well, though the recovery was sometimes very protracted; but the gain in weight and general health immediately following the washing out was very striking. The position then reached was this:—If the antrum is being regularly washed out, and the discharge does not disappear, undoubtedly the right course is to explore the antrum through the canine fossa, and to perform one of the various so-called radical operations. But many patients, realising that they were running no risk whatever, and were suffering nothing but the inconvenience of washing out, declined to have any operation performed, and one could not in fairness indicate to them that they were taking any serious risk in doing so.

He had pushed the radical operation as far as he could, usually removing the whole of the anterior wall of the antrum, down to the floor and to its anterior edge, then making an opening through the inner wall through the thick bone at the bottom of the partition of such a size that a finger passed into the nostril and a finger passed through the opening met, the corresponding portion of the middle turbinate being removed. And yet he had found that after such a radical operation as this, with complete curetting of the antrum, some amount of discharge frequently continued, exaggerated by any catarrh, nearly disappearing in between. He had further noticed that, immediately after a radical operation was performed, in which the whole of the lining membrane of the antrum had been carefully curetted, trans-illumination still gave dulness on the affected side. All this seemed to indicate that by the time an antral empyema was fairly set up the changes in the mucous membrane and the underlying bone were tolerably profound, and he doubted very much whether in a marked case a complete restoration to normal condition was ever attained by any surgical method. Such physiological restoration, however, was not of very great importance. As long as it was drained, no risk apparently existed. The practice he had indicated seemed to him to be the sound one: first an opening through the alveolus, and, in obstinate cases, the radical operation.

Dr. S. A. Ewing (Melbourne)—The many difficulties of successful treatment of chronic suppurative diseases of the accessory cavities of the nose

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emphasize the importance of early treatment of acute suppurative diseases of these cavities. Such conditions are not infrequently found as sequelæ of influenza and infectious diseases in which the mucous membranes of the nose are involved. Sprays of weak solutions of adrenalin, in order to widen the openings of the cavities, followed by appropriate posture for drainage and use of a saline nasal douche, are often sufficient to effect a cure in acute cases, and are of great service in chronic cases where a radical operation is inadvisable. In chronic suppurative disease of the sinuses removal of obstacles to drainage, such as polypi and anterior part of the middle turbinal, often yields good results.

In radical operations on the maxillary antrum, the opening into the nose should preferably be situated on a level with the floor of the nose; this favors drainage in the erect position.

Lambert Lack's operation for ethmoidal sinus disease was not devoid of risk; he had known of one death, from fracture of the cribriform plate and subsequent meningitis. As acting House Surgeon at Golden Square Throat Hospital he had assisted at these operations, and had known of no such accidents under Dr. Lack.

Sphenoidal sinus disease can generally be effectively treated by removal of the middle turbinal where the opening of the sinus cannot be found, and washing it out by means of a long channeled tube. Diagnosis of suppurative conditions in the nose occasionally presents difficulties. He recently, in a case where an operation had been performed on the frontal sinus, found polypi and pus in the ethmoidal region; sections of the polypi showed that they were innocent. On performing Lack's operation on the ethmoidal sinuses, large masses of growth were removed which proved on examination to be carcinomatous.

Dr. Herbert Marks (Sydney) said I do not altogether agree with the views expressed by Dr. Barrett and Dr. Arthur with regard to non-interference in the case of chronic suppuration of the maxillary antrum—that is, by substituting a drain only through the alveolus without removal of the diseased lining membrane of the antrum, the cause of the trouble. It is true that we cannot promise a complete cure in every individual case; but still many cases are completely cured by the radical operation, and we are perfectly justified in performing the complete operation and doing our best for our patients, in the hope that the cure will be permanent. These cases must, or should, be attacked on general surgical principles; and I advocate in every case the radical operation with drainage through the inferior meatus of the nose, removing a portion of anterior end of inferior turbine if necessary.

With regard to ethmoidal disease, I agree with other gentlemen who have spoken, that Lack's method of operation is to be condemned. His operation is unnecessarily heroic and dangerous. Whatever is necessary can be done by cutting forceps, such as Grunwald's, and the careful use of a small curette. If the nostril is too narrow, the condition may be attacked from the outside, as in the common operation for frontal sinus disease.

The ideal operation in chronic frontal sinus suppuration is to clean out the sinus and establish as complete drainage as possible into the nose, and to close up the external wound; but drainage, as a rule, is so uncertain—and having seen Dr. Herbert Tilley's case of septic osteomyelitis, which proved fatal, I always feel it safer to pack the sinus with gauze through the lower angle of the wound, and gradually remove it as healthy granulations appear. This procedure gives very satisfactory results in the majority of cases.

The President, commenting on the debate, said the members were much indebted to the gentlemen who had opened the discussion. With the majority of Dr. Kirkland's conclusions he was in agreement, but he could

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not indorse the opinion of the dentist, as quoted by Dr. Kirkland, that "as a rule the teeth died and decayed after opening the maxillary antrum in the canine fossa." This did not accord with his experience. Such a result would practically condemn the operation. As had been pointed out, the dead teeth would be liable to re-infect the antrum. To avoid injury to the nerve supply of the teeth, the opening should be made in the thin bone of the canine fossa, and the alveolus and the roots of the teeth should not be encroached upon. He could not agree that we should not operate in cases of suppuration of the frontal sinuses except we were sure of a cure. It was in the worst cases—where we could not make sure of a cure—that an operation was most required, and we ought to give the patient a chance, if not of a cure, of alleviation of his symptoms. In his earlier operations he had frequently succeeded in curing frontal sinus suppuration by a very simple operation; but increased experience had led him to treat this disease with more respect. He had come across cases where, on account of anatomical peculiarities in the sinus, the cure was very difficult. The rule which he now followed in chronic cases was, after turning back soft parts and periostium, to completely remove the anterior wall of the sinus, and to carefully remove all traces of polypoid lining membrane. In a few cases, after separating the pulley of the tendon of the superior oblique muscle from the bone, he had removed enough of the inferior wall of the sinus to allow the orbital fat to rise into the same. It is claimed that this tends to a more rapid filling up of the cavity and consequent healing. In the few cases where he had used this method there was no permanent disturbance of vision. The after-treatment consisted in packing with gauze—two or three weeks—till the cavity was lined with healthy granulations: it was then allowed to close, and a pad and bandage assisted in bringing the two walls together. The deformity following removal of the whole of the anterior wall of the frontal sinuses was not so great as might be anticipated. In the case of small sinuses the sinking was hardly noticeable; where the sinuses were very large the patients had prominent brows, and the operation produced a flattening which did not greatly exceed the normal condition. The incision which he used where both sinuses were involved was carried under the line of the eyebrows, and across the root of the nose in the natural crease which is there. This allowed a flap to be turned up, giving plenty of room, and, when healed, was not disfiguring.

Regarding Dr. Lack's method of dealing with the ethmoidal cells in cases of suppuration and polypi, they would remember, when this method was introduced, it was looked upon by rhinologists with a good deal of alarm. He thought the operation was not so dangerous as it appeared. In one very bad case of nasal polypi, and profuse discharge of pus, the procedure was followed by a septic osteomyelitis; this was the only case in his experience where a bad result had followed the operation. In some cases the removal of the diseased ethmoidal cells was the only means of cure. We had to choose between Lack's method and the slower one of working with Grunwald's forceps. In Lack's method the attic of the nose was to be avoided.

Dr. Barrett's case, where the radical operation on the antrum failed to cure, as there was a mucopurulent discharge after every cold, could hardly be used as an argument against the operation, as people with healthy noses were so affected after a cold. He mentioned a case which he had treated, where the radical operation failed to bring about a cure; subsequently he found the prong of a tooth imbedded in a mass of granulation tissue on the posterior part of the floor of the antrum. The removal of this was followed by a permanent cure. The failure of antral operation was generally due to some complication—as ethmoidal or frontal sinus suppuration.

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NASAL AND NASO-PHARYNGEAL FACTORS IN CHRONIC MIDDLE EAR CATARRH.

By S. A. Ewing, M.R.C.S., Eng., D.P.H., Cantab.,

In the first place, in treating this subject on which so many conflicting opinions are and have been held, it will be useful to make some preliminary reference to the term chronic middle ear catarrh. The term catarrh is not a happy one, since, as a general rule, excess of secretion—generally accepted as the chief criterion of catarrhal diseases—is not evident in the cases classed under this category. From this point of view it would seem preferable to describe chronic catarrhal disease of the middle ear as a group of dry non-suppurative diseases of the middle ear; but, though in reference to the condition of the middle ear, deductions have to be drawn from the state of the tympanic membrane, an examination of the eustachian tube—which is practically an extension of the middle ear—usually reveals evidences of catarrhal changes. This, I think, is sufficient justification for adherence to the older descriptive title.

From the deficiences in our knowledge of chronic catarrhal diseases of the middle ear, a satisfactory definition is difficult, and must be more or less conjectural. That given by Lake (1) is generally applicable. Lake states that chronic catarrhal otitis media is a progressive, insidious, and sub-inflammatory affection of the middle ear, consequent upon an extension of so-called catarrhal inflammation from the mucosæ of the naso-pharyngeal space, which involves, firstly, the eustachian tube, and eventually the middle ear. Exceptionally, however, cases occur in which the tympanic evidences of chronic middle ear catarrh alone are present.

I am leaving out of consideration many forms of middle ear deafness which are often erroneously classed as chronic catarrh; typical of these is the deafness, generally intractable, resulting from adhesive processes supervening on neglected acute and sub-acute exudative catarrhal attacks. Also another class of cases, which Politzer has named "otosclerosis," in which we now know —largely owing to the pathological labors of that distinguished otologist—that bony changes affect the stapes and labyrinth; though, owing to the difficulties in diognosing this condition, especially when co-incident with evidences of catarrhal processes in the ear or naso-pharnyx, differentiation is not always possible.

In studying cases of chronic middle ear catarrh, the larger number can be naturally grouped into two classes:—Firstly, those in which the catarrhal inflammation of the nasal and naso-pharyngeal mucosæ, inclusive of the eustachian tube and middle ear—and, probably, in some cases, of the middle ear alone—is independent in the beginning of any other local abnormality or disease. Secondly, those in which the chief factor is local, and the catarrhal condition of the mucosæ secondary to this. Such local conditions as excess of pharyngeal lymphoid tissue, hypertrophies, tumors, irritants, disease of accessory sinuses of the nose, septal deformities, and generally any cause leading to nasal or naso-pharyngeal obstruction or irritation.

This classification is of importance from the point of view of prognosis, which, generally, is better in the latter division, as the rectification of a local condition is more within our power than a dyscrasia, which may be the inheritance of generations. The age incidence is also noticeable; in the first division the onset of chronic middle ear catarrh is chiefly in early adult and middle age, in the second at any period from childhood to old age. Statements of patients are rarely to be relied upon in reference to onset of deafness.

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Ostmann, as the result of a series of examinations with his objective audiometer, found that a diminution of from one-third to one-half of the hearing is regarded as of slight consequence; and frequently it is only on the supervention of tinnitus in the early stages, or marked deafness in the later, when irreparable damage has been done, that the patient is seized of his condition.

In connection with secondary catarrhal disease, striking differences are manifested in different individuals. In one, for example, there may be marked reaction to the presence of a septal deflection; in another, the resultant irritation is at a minimum. The influence of local abnormalities is most marked when there is lowering of bodily tone, and tends to establish chronicity in acute rhinitic attacks, such as are associated with colds and certain infectious diseases.

It is not always possible to draw a hard-and-fast line between primary and secondary catarrhs. For example, as the result of the continuance of a primary catarrhal condition of the nasal mucosa, hyperplasias result; these, by increasing the nasal obstruction, tend to increase the catarrh. In this way a vicious circle is established, which, unless broken, often leads to grave local and constitutional disease.

In the consideration of the onset and cause of catarrhal disease certain pathological and physiological influences require attention. Gouty tendencies and plethora are often associated with congestive mucosæ, and the influence of diet—more especially in the above states—is often very definite. Perverted sexual states are not infrequently reflected in congestion of the nasal mucosa. Puberty and pregnancy occasionally cause a similar condition. I have seen several cases of partial nasal obstruction developing in the third and fourth month of pregnancy, progressing till childbirth, and then subsiding afterwards. The tendency in youth to growth, in age to atrophy, requires to be remembered.

Mayo Collier (2) has recently drawn attention to what he has named latent or intermittent nasal obstruction, in which the nasal passages are partly occluded during sleep. As Dr. Collier points out, statements of patients are rarely to be relied upon when they are questioned as to mouth-breathing. He relies upon the presence of a groove upon the septum opposite the inferior turbinals. Though this is sometimes present, a better guide is to ask the patient to test each nostril on waking, before rising, and at short intervals on assuming the erect position. Each nostril should be alternately closed with light pressure, and the air gently inspired through the free nostril: this should be contrasted with the breathing before lying down. Where nocturnal obstruction is well marked, a hypertrophic condition of the posterior ends of the inferior turbinals and adjoining part of septum is almost invariably present.

Dr. G. Oliver (3), in a paper entitled "Recent Studies on Tissue Lymph-circulation," affirms that, as a result of many investigations, the venous pressure rises to a high point during sleep; whereas, during the waking states, the venous pressure is as a rule only about one-fifth of the arterial pressure, in sleep it rises to a little over half. This venous rise of pressure may play some part in causing nocturnal obstruction.

Gravitation is also a factor, for patients frequently answer on inquiry that the nose is only blocked on the side they lie on in sleeping; and, in some, rest in a horizontal posture for a short period will cause partial obstruction. The obstruction sometimes persists for some time after rising, disappearing some hours later; this may be due to transudation of lymph into the tissues of the mucosæ; possibly the diseased state of the vessels in hypertrophic conditions of the nasal mucosæ may alter their permeability. Nocturnal obstruction is more marked where there is ill health, or want of tone; it is not uncommon in overworked city men, and, in such, frequently disappears on sending them away for a rest.

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How do nasal and naso-pharyngeal conditions lead to catarrhal changes in the ear ?

It is still a matter for discussion whether there is any direct connection between the nose and the ear apart from extension by way of the eustachian tube. Politzer (4) states that " after the cauterisation of the inferior turbinal a decrease, rarely a complete cessation, of the subjective noises in the ear follows ; from this fact it may be concluded that there is a connection between the turbinated bones and the ear. Up to the present such a connection has not been discovered." Dench (5), relying on Zuckerkandl's observation of the communication of the venous sinuses in the eustachian tube with those of the posterior ends of the turbinals, states that in cases where eustachian tube remains patent we must conclude that the turgesence of the turbinated bodies interferes with the venous return current from the labyrinth ; but it should be remembered that patency of the eustachian tube to instrumental methods is not necessarily a patency that secures ventilation of the ear under normal conditions.

Support would seem to be given to Dench's contention by the fact noted by some observers that decrease in hearing, during a cold, is present in some patients upon whom the radical mastoid operation has been performed. I have only noticed this decrease where the eustachian opening into the middle ear has remained patent, and some extension of the catarrhal condition present in the tympanic wall. Whatever doubt there exists in reference to direct connection between the nose and ear, there is none in regard to the dependence of the middle ear upon the integrity of the eustachian tube. In considering this portion of the auditory apparatus, it is important to remember the variations in the position of its opening, length of the isthmus, and patency. In children, and adults in whom the naso-pharynx is small, adenoid growths play a more important part in setting up catarrhal changes in the eustachian tube than in adults with normal-sized naso-pharyngeal spaces. The roomy naso-pharyngeal space, and the adhesion of crusts to the vault of pharynx of ozœna patients, may explain their comparative immunity from ear involment. In adults the eustachian tubal opening is continually immersed during the action of swallowing in the bacteria-sodden secretions that collect in the back part of the inferior nasal strait, and on the roof of the soft palate in catarrhal diseases of the nose.

Charles J. Bond (6), in a recent contribution on ascending currents in mucous canals and gland ducts, and their influence on infection, states that, after insufflation of indigo particles into the naso-pharynx in a case of suppurative otitis, these were found a few days afterwards in the discharge from the ear. Though this has not been proved where the tympanic membrane is intact, sufficient evidence is brought forward by Bond, in his researches on other mucous passages, to establish the probability of the transference of catarrhal infection in this way from the naso-pharynx to the eustachian tube and middle ear. A similar transference probably results from continuity of structure, and from forcible expiratory efforts. That the presence of irritant secretions in proximity to the ostia of the eustachian tube is a powerful factor in the causation of secondary catarrh of the middle ear is shown by the temporary improvement resulting from washing these away, by the use of the nasal douche, and the permanent relief resulting from successfully removing the cause of these secretions. The influence of nasal obstruction requires consideration. This may be complete without any apparent change in the ear.

I examined last year, at the Victorian Eye and Ear Hospital, a patient, age 24, in whom both nares as far back as the choanæ were completely occluded by large polypi, the naso-pharynx was slightly hyperæmic, but both ears were

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normal, and the hearing above the average. Such obstruction generally leads, sooner or later, to aural disease. Cases of unilateral obstruction, more or less complete, are not uncommon. In such, chronic catarrh is frequently met with, and usually the ear that is affected is on the same side as the free nostril; where both ears are involved, the ear on the obstructed side is generally affected last. Tested by the mercurial manometer, the negative pressure behind the obstruction is the same in inspiration as in the free nostril. Examination of the anterior nares shows that the inferior turbinal on the freer side of nose is invariably hypertrophic: examination of the posterior nares generally discloses excessive posterior turbinal hypertrophy on the free side, with excess of mucus secreted by it in the immediate vicinity of the eustachian tube of the same side. During the examination, if the patient gags or swallows, the mucus will be seen adherent to the eustachian opening. Where the mucus is abundant, the opposite eustachian orifice is similarly affected. Removal of the hypertrophy and the catarrh in these cases checks the progressive loss of hearing.

Nasal obstruction, when partial, causes a great increase in the negative pressure in the nose when forced nasal inspiratory efforts are made. This increase in negative pressure, owing to the hyperæmia produced, is one of the disadvantages of the system advocated by Arbuthnot Lane, of nasal breathing exercises in cases of nasal obstruction associated with adenoids. Nasal obstruction causes a marked increase in the negative pressure in the naso-pharynx during deglutition; during this act the eustachian tube is open, and after a slight rise a marked fall of pressure occurs in the middle ear. This explains the retraction of the tympanic membrane in some cases of deafness, and also the sudden increase of deafness during eating where there is nasal obstruction.

Reference has already been made to the conveyance of infection to the middle ear by the eustachian tube. The effect of obstruction of the eustachian tube is to set up negative pressure in the middle ear; this leads to impairment of function in two ways—firstly, by its effect upon the vessels of the tympanic mucous membrane, causing hyperæmia; and, secondly, by its effect upon the tympanic membrane, causing retraction.

It has been estimated that 20–40mm. of mercury is the average pressure necessary in normal conditions in Valsalva's experiment to open the eustachian tubes, while, with a slight swelling of the mucous membrane, a rise of 100-120mm. of mercury is required.

Normally the establishment of ærial equilibrium between the middle ear and the naso-pharynx is involuntary, and is independent of great pressure, and any interference with the maintenance of this ærial interchange is apt to disturb the integrity of the ear. Forcible methods of inflation of the middle ear, by Valsalva's method, or by Politzerisation, are often relied upon in order to determine the functional patency of the eustachian tube. Politzer states the amount of pressure used in his method of inflation varies from 0.1 to 0.4 atmospheres and over. Such tests are fallacious, as these high pressures are only present in the naso-pharynx during strong expiratory efforts.

The mode of treatment by inflation of the middle ear in chronic middle ear catarrh, which is dependent upon catarrhal conditions that are remediable, has many disadvantages. Temporary relief is sometimes gained, but the rapid re-absorption of the air in the tympanic cavity requires repeated renewal of the inflation. The catarrhal disease of the ear meanwhile goes on unchecked, and the continual forcible distension of the tympanic membrane leads to permanent stretching of its upper portion, rendering it less capable of transmitting vibrations.

In some cases of chronic middle ear catarrh the temporary improvement in hearing caused by successful inflation is rendered permanent on removing

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the causes of the catarrh. In other cases the improvement resulting from removal of the catarrhal causes is greater than the temporary improvement got by inflation. In a third class, where inflation of the middle ear gives no improvement in hearing, yet improvement—sometimes marked—may result upon removing the catarrhal conditions affecting the ear. This I have only found where the catarrhal causes are excessive.

I have endeavored in this brief sketch to indicate some of the ways in which catarrhal processes are set up in the nose and naso-pharynx, and the manner in which they affect the ear, and also the importance of the nasal passages in reference to the production not only of naso-pharyngeal catarrh, but also of chronic catarrh of the middle ear.

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SECTION OF PATHOLOGY, ANATOMY, PHYSIOLOGY, AND THERAPEUTICS.

SOME RECENT OBSERVATIONS ON CANCER AND TUMOR GROWTH.

By D. A. Welsh, M.A., B.Sc., M.D.

Of all the great scourges of mankind, only one remains untempered by that scientific study of disease which we term Pathology. Smallpox, except in isolated outbreaks, is a thing of the past. The pyæmia that was wont to render perilous every surgical operation yielded to antiseptic and aseptic measures. Diphtheria and hydrophobia have been robbed of half their terrors. Bubonic plague, despite the colossal failure of India, may be successfully combated in an intelligent community. Outbreaks of enteric fever and of cholera are in every sense of the word preventable. The prevalence of malaria, with its long tale of disablement and death, is conditioned by the facilities provided for the breeding of mosquitoes. And that tuberculosis should continue to be of all diseases the most wasteful of human life is a fact that is not creditable to us as a profession, nor to us as a people, for it is due wholly to the disregard of what pathology with no uncertain voice has taught. But cancer and tumor growth are as rife to-day as ever. If there be any change, it is probably not in the direction of abatement; and, even if there be abatement, pathology has contributed nothing to it. No means of prevention is known, nor certain means of cure. The only relief—that afforded by careful, free, and prompt excision—is at the best uncertain,

No problem in pathology, therefore, is more urgent, or more elusive, than that of the nature and origin of tumor growth, and within recent years there has been awakened interest and renewed activity in its investigation. Under the gracious patronage of His Majesty the King, the Imperial Cancer Research Fund has been inaugurated on a scale unprecedented in the history of medicine, while the able workers who are more directly responsible for its scientific application have made excellent use of their unique opportunity. Elsewhere in Europe and in America many similar institutions have been started, and under their auspices, as well as independently, much good work is being done. The result is that since the last meeting of this Congress, when papers on cancer, of great interest and value, were read by Professor Allen and other members, there have been published observations and hypotheses of a most noteworthy and suggestive character. That as yet their full significance is uncertain, and their practical bearing unknown, does not detract from their present interest as illuminating conceptions, nor from their permanent value as fresh scientific truths. It is with the object of inviting your attention to some of this recent work that I have framed my address, although the time at my disposal will necessitate a very fragmentary review.

It is unnecessary to introduce subtleties of definition or to discuss the relation of tumor growth to those conditions known as inflammatory. I have throughout used the words tumor, new growth, and neoplasm as interchangeable, and the word cancer as connoting all the more malignant forms, irrespective of structure. I have occasionally used a general term instead

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of the more restricted term cancer or malignant growth, because, in my judgment, any explanation of the nature and origin of the most malignant forms (cancer and sarcoma) must be ultimately applicable to the least malignant (benign) forms also. The varying degrees of malignancy and the various factors that condition malignancy are mainly dependent upon attributes that are common to all neoplasms, though not always present in the same degree. The distinction between a malignant and a benign tumor, all-important in clinical practice, depends not on any essential difference in kind, since all neoplasms alike are independent of the needs of the parent organism, and independent of the laws that regulate the growth of the parent tissue, and exhibit, moreover, a certain mutual independence and intrinsic vitality in their constituent cells. The distinction depends rather on the degree to which are developed these fundamental attributes, and also depends upon such accidental conditons as the ease with which the individual cells may be mechanically detached from the tumor mass.

ARTIFICIAL PRODUCTION AND TRANSPLANTATION OF NEW GROWTHS (NEOPLASMS).

Half a century ago the scientific genius of Virchow laid the foundation of modern experimental pathology in two general biological principles—(1) that every cell in the body is descended from a pre-existing cell, and (2) that the reactions of the living cells of human tissues are essentially similar to the reactions of other animal cells—so that it is legitimate to argue from what occurs in animals to what occurs in man. Both of these principles became of fundamental importance in relation to cancer research, since it became known that animals, too, suffer from new growths of which the structural details are often identical with those of tumors occurring in man. In view of these principles and facts it has long been recognised that the artificial production and transplantation of tumors in animals was essential to the proper study of neoplasia, or tumor growth.

To this end innumerable experiments have been made, and, when investigation flagged under the deterrent influence of consistently negative results, a fresh stimulus was given by the development of bacteriology. The resemblance between the invasion of a cancer and other infective processes which had been compelled to yield the secret of their origin fostered the expectation that the cause of cancer also would be found in some form of microbe. Many different parasites were described within cancer cells, several were obtained in pure culture, and a few inoculated into animals gave rise to cell-proliferations somewhat resembling tumor growth. But such resemblances were only superficial and misleading. Notwithstanding the great amount and the occasionally brilliant character of such work, it must be conceded that no one has yet been able to originate an unequivocal new growth by the agency of any parasite.

When healthy tissue is transferred from one animal to another, the graft may "take" or may fail to "take." The probability of success is greater when the graft is made into another animal of the same species. Such an artificial graft may form a hypertrophic overgrowth when transplanted from a less vascular to a more vascular site, but it never acquires the independent and unregulated growth characteristic of a tumor in the sense in which we are using the term. The oft-quoted experiment of Lambert Lack, who in 1900 disseminated the ovaries of a rabbit throughout its peritoneal sac, and more than a year afterwards found an extensive cancerous infiltration of the abdominal and thoracic viscera, is open to other interpretations. This particular experiment, moreover, has been many times repeated with invariably negative results. In July, 1903, I instituted a series of trans-lantations of

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epithelial cells from the chorion, ovary, thyroid, adrenal, and epidermis in such animals as guinea-pigs, rabbits, cats, and dasyures, the transplantations being made into other parts of the same animal, into other animals of the same species (of the same and of opposite sex, and at different ages), and into animals of different species; but, with one doubtful exception, I succeeded only in adding another to the long list of failures to produce tumor growth by this means. The exceptional case was that of a guinea-pig into whose peritoneal sac chorionic epithelium from another guinea-pig had been introduced, and which later died, having in its pelvis a large mass of recently extravasated blood in which proliferating chorionic epithelium was found.

When introduced into animals of different species, the transferred cells are usually, though not invariably, destroyed by cytolysins, the living tissues reacting towards them as towards other alien albuminous molecules. Occasionally, however, the alien cells are tolerated, and may even be adopted as integral portions of the new organism. Blood corpuscles, for example, do not invariably undergo hæmolysis when transfused into animals of different species, and grafts of epidermis from the rabbit and the puppy may thrive on denuded surfaces in man. It is important to recognise these facts, since they indicate no reason why cancers should not occasionally be transmissible to alien species. It is known that epitheliomata tend to arise in the cicatricial tissue of old burns and in other scars, and it would be interesting to ascertain whether the liability is greater when no grafts have been made, or when grafts have been taken from another person of earlier or later age, or from animals whose term of life is shorter than that of man—whether, in fact, the presence of an epithelium of a different tenure of life has any relation to the incidence of epithelioma.

The secondary dissemination (metastasis) of certain malignant new growths may be regarded as a process of natural transplantation, and illustrates the possibility of transferring such tumors to other parts of the same individual. That this may be accidentally effected in the excision of a malignant growth has been repeatedly emphasized. If a cancer be incised during removal, cancer cells may escape from the cut surfaces, or be carried by the knife into adjacent tissues, and, becoming there implanted, give rise to a local recrudescence. Moreover, a growth of restricted malignancy may accidentally be given an opportunity of extensive infiltration, as when a cystic adenoma of the ovary is ruptured and the liberated epithelial cells infect the peritoneum.

Since, therefore, a natural or artificial dissemination of certain tumors is so readily effected in the individual in which they originally grow, it might be expected that such growths would bear transplantation at least to other similar individuals. Such an expectation is far from being realised. Human new growths have never to my knowledge been transmitted to other animals, notwithstanding many attempts, of which a most extensive series was undertaken by Shattock and Ballance in 1890. Nor am I aware of any authentic instance of the successful transference of a new growth to an animal of different species. Even when inoculations are made into animals of the same species, except in the case of certain infective granulomata which are not true neoplasms, the usual result is failure, and at the date of the last meeting of this Congress, in February, 1902, only a few isolated successes had been recorded. Obviously such a record of failure has constituted a serious bar to the investigation of tumor growth.

The first to achieve systematic success in the transplantation of tumors was Jensen, of Copenhagen, whose results were not made generally known until 1903. In the same year Borrel, of Paris, published another series, and in 1904 there appeared the first scientific report of the Imperial Cancer Re-

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search Fund, in which further examples were recorded. All these successes were obtained by transplanting tumors of mice to other mice. In no other animal, with the single exception of the rat, which is of the same genus as the mouse, has there been effected a successful transplantation of a neoplasm, even from another individual of the same species. Attempts to transfer malignant new growths from the horse, dog, cat, rat, and carp to other animals of the same species have at the hands of the Cancer Research Fund resulted in failure, although in the case of the dog, cat, and rat over 900 inoculations were made. In December, 1904, I made an endeavor to transfer a squamous epithelioma from a dasyure (*Dasyurus Viverrinus*) by inoculation into fifteen other dasyures and into ten cats, but nearly all the animals died within a week of a virulent septicæmia, and of the few survivors the dasyures prematurely fell victims to the misplaced zeal of another worker in the University, while the cats (three in number) show no sign of new growth to this day.

Earlier in the same year I had, through the kindness of Mr. A. S. Le Soef, of the Zoological Gardens, Sydney, obtained specimens of cancer from a lioness and from a tigress, both of which were old animals, and, by a remarkable coincidence, had died within a few days of each other. I have now under investigation a growth from a frog (*Hyla Aurea*) which I received a few weeks ago through the kindness of Dr. Chapman, and with it I have been able to infect other frogs, but I have not yet been able to determine whether it is true neoplasm or merely some form of infective granuloma. I have not yet been able to obtain a new growth in a mouse, but that is not surprising, since out of 30,000 tame mice examined by the Cancer Research Fund only twelve were found to have malignant new growths.

Jensen's original tumor was an epithelioma growing in a white mouse. Introduced into other mice, it yielded at first 30 to 60 per cent. of successful transplantations, and it has proved to be one of the most readily transmissible of all tumors hitherto tested. It is still growing, like a pure culture of bacteria, in various laboratories on the Continent of Europe, in England, and in America, the medium on which it thrives being successive generations of mice. In its duration of life (three and a half years) it has already exceeded the natural term of life of a mouse, and in bulk it has already exceeded that of many thousands of mice. This great power and persistence of growth is paralleled only by the natural propagation of successive generations of mice, and indicates an enormous potential vitality in the cells of the new growth, a vitality comparable to that which secures the continuance of the race. Nor is this an isolated phenomenon. The production of tissue by transplantation of other cancers of mice may proceed no less rapidly.

Jensen made so careful and accurate a study of the conditions of successful transplantation and of the nature of the process that his results have been abundantly confirmed, notably by the Cancer Research Fund. He found that the cells of the new growth could be isolated from the body for a considerable time without losing the power of originating a fresh tumor. When kept in a sterile condition on ice the cancer cells could still give rise to a new growth even after eighteen days, whereas an exposure of less than forty-eight hours to the body temperature rendered all inoculations abortive. "The tumor received by the Cancer Research Fund from Professor Jensen was forwarded by post from Copenhagen, under sterile precautions, and used for inoculation on the fifth day after it left Copenhagen." (First Scientific Report, p. 14.) These facts illustrate from another point of view the remarkable vitality with which the cells of the new growth are endowed.

In their general characters such transplanted growths resemble very closely the secondary (metastatic) disseminations naturally occurring in the course of many malignant tumors. The constituent cells of each are the direct

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descendants of the cells of the primary growth. In the case of epithelial growths which alone have been transmitted the stroma is variable in amount, and is produced by the reaction of the invaded tissue. These facts are demonstrable in conditions of natural metastases also, as in the development of embolic foci in the liver. Throughout the longest series of sub-inoculations yet attained each successive tumor has been composed of cells structurally identical with those of the original growth.

The transmissibility of new growths from mice is conditioned to a remarkable extent by racial distinctions among the mice inoculated. Thus a cancer originally growing in a German mouse meets a subtle antagonism when introduced into an English or into a French mouse, and gives a very low percentage of successes; but, as it becomes accustomed to the new race, the percentage of successes rises, and may ultimately equal that attained by direct inoculation of the original growth into German mice. Such specific distinctions between different races of the same species reveal the possibility of a reaction even more subtle than that elicited by the specific precipitins which can distinguish the corresponding albuminous molecules of different races within the same species.

When introduced into other mice of the same race, the new growth appears to be independent of such factors as the age or sex of the mice inoculated, and the frequently high percentage of successes contrasts very strikingly with the relative infrequency of primary new growths in the same animals. The conditions required for the successful transplantation of a new growth cannot therefore be identical with those necessary to its primary origin, a fact which indicates that in becoming a new growth the cells have acquired potentialities which enable them to continue growing in circumstances inimical to their original development.

PHENOMENA OF CELL-DIVISION EXHIBITED BY PATHOLOGICAL NEW GROWTHS AND THEIR SIGNIFICANCE.

Some of the most significant of recent observations on tumor growth relate to the phenomena of cell-division. In order that I may be better able to make myself intelligible, allow me to recall to you the main features of the process as it occurs in healthy tissues. As a rare and exceptional occurrence a cell may divide by a process of amitosis, or direct division, in which the nucleus becomes simply constricted, and separates into two parts, the cell likewise becoming constricted and later separating into two cells, each containing a portion of the original nucleus. This simple mode of bi-partition is limited to such highly specialised or degenerating cells as are found in glandular epithelium or temporary embryonic envelopes. (Wilson.)

The cell-divisons most characteristic of the development of animal and vegetable tissues are exceedingly complicated and constitute the process of indirect divison, or mitosis, of which only the briefest outline can be given. Here the nucleus undergoes elaborate preliminary changes, both of a physical and of a chemical nature, the prophases of the mitosis. The chromatin reticulum becomes gradually transformed into a convoluted filament, the spireme, of more intense staining reactions. At first fine and closely interwoven, the filament forms the close spireme; later it shortens and thickens to form the open spireme; ultimately it breaks across at intervals, giving rise to separate masses of chromatin, usually in the form of short bent rods, known as the chromosomes. These chromosomes become arranged equatorially upon the threads of a spindle-like structure which has in the meantime made its appearance within the cell. Each chromosome splits lengthwise (metaphase) into two exactly similar fragments, which gradually separate (anaphase) along the spindle-threads, so that precisely one-half of each chromo-

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some diverges towards each pole of the spindle. The terminal phase, or telophase, consists of the division of the cell itself in the equatorial plane of the spindle, so that each daughter-cell contains one of the groups of chromosomes, from which the corresponding daughter-nucleus is reconstructed. The fundamental event in mitosis is the rigid distribution of one-half of each chromosome to each pole of the spindle, since it ensures that each daughter-nucleus shall contain exactly one-half of the chromatin originally present in the mother-nucleus.

From the time that the fertilised ovum first divides, and throughout all the cell divisions that go to make up the body of the embyro, this type of mitosis is maintained, and may be termed the somatic mitosis. Moreover, the number of chromosomes formed by each nucleus preparatory to division does not alter. It remains constant not only throughout the development of the individual but throughout all the individuals of the same species, and it is invariably an even number. Thus in man, the ox, the guinea-pig, and the onion the number is said to be sixteen; in the mouse, the salamander, the trout, and the lily it is twenty-four; in some nematodes it is as low as two or four, and in some crustaceans as high as 168. (Wilson.)

But not all the cells resulting from repeated segmentation of the fertilised ovum go to make up the body of the new individual. After a certain number of divisions the newly formed cells are called upon to play different parts. Some go to form the embryo, and by combined somatic divisions build up the body of the adult organism. Others go to form the embryonic envelopes and appendages, and yet others are early set apart as the precursors of the reproductive cells, and become incorporated within the embryo. It is to the cell-divisions met within the third group that I now invite your attention.

At first the reproductive cells multiply in the same way as do the body cells, but, as they approach maturity, they exhibit types of nuclear division characteristically different from that of the body cells. The critical mitoses that precede the maturation of the sexual elements are known as the reduction divisions, since one of the more obvious of the points of divergence consists of a reduction of the number of chromosomes to exactly one-half of the number characteristic of the somatic mitosis. Of these divisions there are at least two, and in the higher animals only two, the first being distinguished as the heterotype, the second as the homotype mitosis. (Fleming.) In the heterotypical division the reduced chromosomes assume peculiar forms, often appearing as loops, or rings, or tetrads, and their division on the spindle is not longitudinal but transverse. It is needless to enter into further detail. It is sufficient to emphasize the facts that reduction divisions are distinguishable from ordinary somatic divisions; that, in natural circumstances, they appear only in one tissue—the reproductive tissue—and there only at one phase of its development that preceding the ripening of the sexual cells.

That this phenomenon is in some way not understood, an essential prelimininary to the propagation of new individuals is indicated by its almost universal distribution. It has been found in all the higher organisms examined, animals and plants alike, marking the maturation, or a definite stage in the maturation, of the cells set apart for reproduction. In animals at all events, when all the critical mitoses have supervened, the cell is fully prepared for union with a matured cell of opposite sex, and until this union is accomplished no further development can, save in abnormal and exceptional conditions, occur. A new generation is originated by the union of two mature sexual cells, or gametes—the spermatozoon and the mature ovum. Each gamete contains within its nucleus half the number of chromosomes that are characteristic of the somatic mitoses of the organism arising from their

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fusion, and the union of the two gametes restores the full number of chromosomes to the resultant cell—the fertilised ovum—which is the first of the new generation. And throughout all the cell-generations of the new organism the full number of chromosomes will be retained, except in the maturation divisions of its reproductive cells, when they in turn make their final preparations for that union which will introduce the next succeeding generation.

We are now in a position to consider the phenomena of cell-division as they appear in cancer and other pathological new growths. Cytologists have for many years been familiar with the fact that derangements of the mechanism of mitosis are common in cancer cells, and that in tumors of rapid growth the processes of cell-division may exhibit extreme disorder. (Klebs, Hansemann, Galeoti, quoted by Wilson.) Such derangements include asymmetrical mitoses, the chromosomes being unequally distributed to the daughter-nuclei, and multipolar mitoses, in which more than one spindle is formed. Even amitosis, or direct division, is not infrequently met. Similar abnormal mitoses may be artificially induced by the action of irritants or poisons, *e.g.*, in the epidermal cells of salamander by antipyrin, potassium iodide, &c. (Galeotti, quoted by Wilson.)

But it was left to Farmer, Moore, and Walker to discern a certain orderliness in this disorder, and their discovery, communicated to the Royal Society in December, 1903, was as simple as it was significant. They found that at the primary site of origin, and within a zone behind the advancing margin of malignant growths, the cells of the tumor were dividing after the manner of the heterotypical mitoses of reproductive tissues. Not only were the chromosomes of the dividing nuclei reduced to a number approximately half that of the somatic divisions, but the resemblance extended to other and often to minute points of detail, including the disposition of the chromosomes in the form of loops, rings, and tetrads, and their transverse division on the spindle. In other words, they demonstrated in the newly formed cells of primary cancerous growths processes of cell-division which all biological research has shown to be characteristic of the maturation, or of a definite step in the maturation, of the reproductive cells preparatory to conjugation.

Indorsed by the high authority of Professor Farmer and his colleagues, the essential similarity of cancerous tissue, at a certain stage of the primary new growth, to reproductive tissue is a fact of the utmost moment in relation to tumor growth. The attainment of the critical mitoses indicates that the cell has entered upon the terminal phase of its existence as a cell of the old generation, and that it is now ready to initiate a fresh life cycle, a new growth of cells, an independent organism. It does not indicate that a new generation of cells has yet arisen.

A further significant resemblance between the cells of cancerous and those of reproductive (gametogenic) tissues has recently been described by the same observers. In cancer cells there are frequently encountered clear vesicular bodies containing small deeply-staining granules, but their nature has long been an enigma. Occasionally they have been described as some form of protozoon parasite, whose presence stimulates the cell to undue proliferation and is the excitory factor in the ætiology of tumor growth. The presence of such cell-inclusions has formed the basis of many hypotheses regarding the parasitic origin of cancer. The parallel raised by Professor Farmer and his colleagues suggests a wholly novel solution. They point out that, in the final metamorphoses of the spermatozoon in the vertebrate class of animals, a vesicular body, the archoplasmic vesicle, containing one or more darkly-staining granules, is developed alongside the nucleus, and may

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attain such a size as to indent and deform the nucleus. The vesicle with its contents ultimately persists as the so-called "cephalic cap" of the spermatozoon. This phenomenon is a conspicuous, and, so far as is known, a constant characteristic of spermatogenesis in the vertebrata, and is known to have an even wider distribution. In its general appearance the archoplasmic vesicle strikingly resembles the vesicular inclusions of cancer cells, and, as these authors conclude, "it is not, perhaps, accidental that, just as in the case of nuclear divisions, so also in the cellular inclusions, a parallelism between the cells of reproductive tissues and those of cancerous growths should be found to exist." But they do not on this acocunt regard cancer cells as identical with sexual cells.

Of the many fresh observations for which Drs. Bashford and Murray, of the Imperial Cancer Research Fund, are responsible, the majority deal with the nature and distribution of the mitoses in cancer cells. The work of Jensen and of Borrel on the transmissibility of cancer in mice, and that of Farmer, Moore, and Walker on the resemblances between certain cells of cancer and those of reproductive tissues, provided an opening of which they were not slow to avail themselves. While emphasizing the difficulties that attend the interpretation of the numerous forms of abnormal mitoses in cancer cells, they showed that heterotypical forms occurred not only in cancers of human origin but also in those of lower animals, including such widely divergent types as the horse, cow, dog, cat, mouse, and trout; that, in fact, the resemblance of cancer cells at a certain stage in their life history to reproductive cells approaching maturation is a phenomenon inferentially present in all cancers of vertebrate animals.

While thus confirming and extending the observations of Professor Farmer and his colleagues, they indicate other significant phenomena. I understand that, in their experience, somatic types of mitoses in which the nuclear divisions exhibit the full unreduced number of chromosomes are in fully developed tumors more common than reduced divisions, and that, whatever be the age and extent of the cancer, ordinary somatic mitoses are almost invariably present at certain stages and in certain conditions of growth. It is thus that the cells multiply at the advancing margins of malignant growths, whether primary or secondary, and thus that they multiply when, detached from the tumor mass, they undergo the initial proliferation that starts a natural metastasis or an artificial transplantation. Not only, therefore, does the heterotype and post-heterotype tissue form only a small proportion of any extensive new growth, but the cells which are youngest in time and presumably oldest in descent almost invariably exhibit somatic mitoses. Once the reducing divisions have appeared in reproductive tissues, the cells that are youngest in time and oldest in descent are in animals represented only by the mature and maturing sexual cells, and in animals and in plants alike are known to multiply only by mitoses in which the number of chromosomes is reduced by one-half. Not all the cells of cancerous growths can, therefore, be regarded as comparable to reproductive cells. After a time there must intervene some circumstance which restores the somatic mitoses, and, apart from parthenogenetic development, in which the ovum is fertilised by one of the polar bodies, there is only one event which is known to have this effect.

It is, as we have already seen, the conjugation of two cells, already prepared for union by a sequence of reducing divisions, that restores the full number of chromosomes to the resultant cell, and originates a new generation. This phenomenon, too, has been described (Bashford and Murray). In transplanted cancers of mice nuclei are found staining more darkly than usual, and drawn out into blunt processes as if amœboid. Frequently one of these

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processes becomes closely applied to an adjacent cell wall. Occasionally the nucleus of the cell may be seen in contact with that of an adjacent cell, and apparently continuous with it. So far as I am aware, this conjugation of resting nuclei has been observed only at certain stages in the growth and development of transplanted tumors in mice. Though it has not yet been observed at the primary site of cancer, the reports of the Cancer Research Fund insist on the view that some circumstance equivalent to conjugation is both requisite and adequate to explain the salient phenomena of the growth, as distinct from the origin, of cancer. It is argued that only a new generation of cells, constituting in all essential respects a new organism, could be endowed with that independent and autonomous growth characteristic of a neoplasm.

Both Professor Farmer and Dr. Bashford draw a comparison between the intercurrent relations of certain cell-generations in plants and the relation of neoplasia to an animal organism. I propose rather to emphasize a relation between recurrent cell-generations in animals which constitutes a still closer analogy. In marsupials, and to a greater extent in the higher mammals, the new generation originating in the fertilised ovum is for a time definitely parasitic upon the old generation—the maternal organism—and the relation of the fœtal chorionic epithelium to the maternal decidua is essentially that of a neoplasm of restricted malignancy so far as local infiltration and independence of growth is concerned. The chorionic epithelium is already a new generation and already parasitic in relation to the maternal organism, but its growth is limited and its natural term of life restricted to the gestation period of the species. When it becomes cancerous, it is peculiar among cancers in being one generation further removed from the organism in which it grows—that is, altogether, two generations removed if the cancer itself represents a new generation, and, though embryonic in relation to other tissues, it is intrinsically senescent or decadent.

The new generation—neoplastic and embryonic alike—grows independently of the requirements of the parent organism, but is at the same time wholly dependent on the parent for its nutrition. In this relation of mutual dependence and independence no distinction whatever can be drawn between the new growth of a tumor and the new growth of a mammalian embryo. Where these two types of new growth fundamentally differ is in the regulated and orderly growth of the embryo and its envelopes, as opposed to the unregulated and disorderly growth of the tumor. Of this difference no explanation has been advanced. The origin of a cancer from the conjugation of cells would be sufficient to explain its independence in the sense above defined, but inadequate to explain its autonomy. A new organism, with laws of growth of its own, is an entity unknown in the whole range of normal biological development. Every new generation has most rigidly impressed upon it the laws of growth to which its parents have conformed, only minor variations being permitted. But in the development of a cancer we find the unprecedented phenomenon of a new generation of cells growing independently of, and often at variance with, the laws that regulate the growth of the parent tissue.

Recent observations in biological science have indicated a unit of living matter beyond the cell, beyond the chromosome—a unit consisting of the chromatin granule (chromomere). Boveri and Brauer (quoted by E. B. Wilson) regard the splitting of the chromosomes as an independent reproductive act of the chromatin. "In my opinion," adds Brauer, "the chromosomes are not independent individuals, but only groups of numberless minute chromatin granules which alone have the value of individuals"; and Wilson indorses this opinion in the words, "These observations certainly lend strong support to the view that the chromatin is to be regarded as a

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morphological aggregate—as a congeries or colony of self-propagating elementary organisms capable of assimilation, growth, and division." (P. 114.) The chromatin forms the hereditary basis by which the processes of growth and differentiation are controlled, and from which they receive the specific stamp of the race (E. B. Wilson), and the accurate subdivision of the chromatin in mitosis is commonly regarded as the mechanism by which genetic continuity and consistency is maintained. In the normal development of the embryo an abnormal mitosis is rare; whereas in neoplasia, and particularly in malignant forms of rapid growth, pathological mitoses are abundant, and may indeed be so frequent as altogether to obscure the nature of the nuclear divisions. While I am fully aware that the significance of fertilisation and mitosis is one of the vexed questions in biology, it may not be unreasonable to correlate the regulated growth of embryonic tissue with the accurate subdivision of chromosomes, and the unregulated growth of cancerous tissue with the frequent irregularities in chromatin distribution.

So far as they have been observed, the mitoses in transplanted growths exhibit no abnormality such as would cause an unequal subdivision of chromatin even at the periods of gametogenic activity; and, further, the successive new broods of cancer cells show no deviation from the structure of the original brood throughout the longest period of observation yet attained, and appear to be subject to precisely similar laws of growth. Deviation in stucture to a slight degree, and liberation from the original restrictions of growth to a great degree, probably therefore depend on the extent of the uneven distributions of chromatin attendant on the birth of the primary generations.

THE RELATION OF NEOPLASIA TO SENESCENCE OF TISSUE.

The well-known fact that the liability to malignant new growth increases with the lapse of years, the so-called " age-incidence " of cancer, has acquired a fresh significance in the light of recent work. It is commonly stated that sarcomata are more frequent in the early years of life, while cancers are more frequent in the later. It is interesting to note that the recent statistical report of the Imperial Cancer Research Fund indicates that sarcomata, like cancers, increase in frequency as life advances. In my own experience I have been impressed with the fact that cancers form a much larger proportion of the malignant new growths occurring in young persons, and even in children, than is commonly supposed. It appears, therefore, that while either of the two main types of malignant new growth may occur in the young, yet the frequency of both increases with advancing years.

If this phenomenon be further analysed, it is found that the increase does not uniformly progress. Cancers of the stomach, for example, appear and attain a maximum frequency earlier than cancers of the skin; cancers of the uterus and mamma are more frequent towards the decline of reproductive activity than in the later years of life, and cancers of chorionic epithelium closely follow the appearance of that epithelium. It is not so much, therefore, the age of the individual in whom it occurs as the age of the tissue in which it arises that determines the maximum incidence of cancer. Even as regards the tissue, it is not, strictly speaking, its age, but its senescence that is the important predisposing factor. Tissues appear to be most liable to neoplasia when, having attained maturity, they enter upon the phase of decadence, or involution, and this phase is reached by different tissues at different periods in the life of the organism. Moreover, the same tissue (*e.g.*, mamma, uterus) may become effete at different periods in the life histories of different animals, and the age-incidence of cancer correspondingly varies. This is well illustrated by Professor Woods Hutchinson, who, in his " Studies in Human and Comparative Pathology," correlates the relative infrequency

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of mammary and uterine cancers in the lower animals with the fact that these organs remain actively functional nearly to the end of life; in other words, that the lower animals do not outlive these organs to the same relative extent as does the human subject. The reports of the Imperial Cancer Research Fund also insist on the importance of the "age-incidence" in the ætiology of cancer, and emphasize the fact that the same relation of cancer to decadence tissue obtains in man and in other animals, though in the latter, with their shorter tenure of life, the period of relative immunity is correspondingly shortened. The widely recognised influence of irritation and of injury in predisposing to tumor growth is conceivably and probably operative only in so far as it accelerates senescence of tissue.

The relative infrequency of new growths in animals other than man is probably due to various causes. If their brief tenure of life shortens the period of comparative immunity, still more does it abbreviate the period of maximum liability, and I am not aware that an average growth of cancer becomes more rapid in short-lived animals. Further reasons for this difference may be found in the facts that the habits of animals do not predispose so frequently as in man to premature senescence of tissue, and that organs which, in the human subject undergo involution many years before the natural term of life, in animals may remain functional to the end. And particularly in the case of wild animals in natural conditions is it true that those approaching decrepitude are either killed off or die of starvation. The important point is, not that new growths are less frequent in animals than in man, but that a consideration of the causes of this relative infrequency serves only to emphasize the significance of tissue senescence as a factor in the origin of new growths.

Following out this relation of the incidence of neoplasia to the age limit of tissue activity, Dr. Bashford and his colleagues were led to a remarkable observation. The animal in which transplantation of tumors from one individual to another has been most successfully effected is the mouse, whose natural term of life is about three years. Within this brief compass there are condensed the vital activities which in man may occupy more than half a century, and the processes are correspondingly shortened. Thus gestation in the mouse is completed in three weeks, while in man it occupies nine months. But new growths from mice had been propagated through generations of other mice over a period far exceeding the average duration of life in mice. It was natural, then, to inquire how the new growth came to be endowed with this apparently limitless power of growth without deviation from its original structure. A systematic examination of such transplanted cancers revealed the discovery that the growth was not continuous but interrupted; that at periodic intervals there appeared a number of associated phenomena, including heterotypical mitoses and nuclear conjugations. The onset of these phenomena is related, I understand, not to the age of the animal or to the duration of the tumor growth within it, but solely to the number of cell generations of new growth that have intervened between the last preceding heterotypical mitoses, that is, in the strictest sense, to the age of the new growth. Just as mice after a few years would become extinct unless new individuals were generated by the union of reproductive cells, so apparently a maturation of gametes and their conjugation becomes at recurrent and definite intervals of time essential to the propagation of the cancer. This, if it can be substantiated, is the strongest argument yet adduced in support of the hypothesis that a new growth is essentially a new generation of cells arising after the manner of a new individual of the species. Nevertheless, it raises fresh difficulties.

That the senescence of the cells of the new growth should be the only apparent factor in determining the whole sequence of these phenomena in

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transplanted growths is altogether remarkable. Consider for one moment what it means. There is abundant evidence that reducing divisions in non-reproductive tissues are not necessarily indicative of malignant new growth. They have been encountered not only in benign tumors, but also in proliferations of tissue of an inflammatory or irritative nature. Moreover, they may not be observed in some new growths of an undeniably malignant type. So far from their presence being a criterion of malignancy, it is not even a criterion of a new growth, nor does the new growth inevitably follow upon their appearance in a tissue. All that they signify is that the tissue is so far prepared for the initiation of a new growth that certain of its cells have in some way been induced to acquire the characters of reproductive cells undergoing maturation. Granted, then, that the senscence of the tissue, naturally or artificially produced, is adequate to determine the onset of the reducing divisions, nevertheless in the primary growth of a cancer it cannot be the only ætiological factor, since not every obsolete tissue gives rise to a new growth—since, even when maturation divisions have supervened, the tissue does not necessarily undergo neoplasia. Presumably the additional factor is some form of cell conjugation, and this in turn requires some stimulus not invariably present even when the conditions as to senescence of tissue and maturation of gametes has been fulfilled.

Not all cancers can be suocessfully transplanted; but, if I interpret the observations aright, a successful transplantation does not depend upon the immediate initiation of a new generation of cancer cells in the new animal. I understand that the initial proliferation of a transplanted growth consists of cell generations continuous with the preceding cell generations, and that once the cancer cells have started to grow in a new animal they have never failed to continue to grow in that animal. Presumably, therefore, a transplanted cancer has never failed to propagate new generations of cancer cells when it approaches the age limit of its growth. There may be a failure to infect a new animal, but apparently never a failure to propagate a new brood of cancer cells, whatever be the age or sex or other conditions of the animal in which the cancer is growing. Why the origination of the secondary generation in transplanted cancer should be independent of that infrequent conjunction of conditions required to start the primary generation is a problem that awaits solution.

On the hypothesis that a new growth consists of a new generation of cells, it is not easy to understand why it should so infrequently follow upon the primary appearance of reducing divisions in somatic tissues, and so inevitably follow the recurrence of the reducing divisions in transplanted growths; whereas, on the view that a new growth is analogous to a mass of reproductive (gametoid) tissue, the difficulty disappears. Once the primary differentiation from somatic to reproductive (gametoid) tissue has been effected, then subsequent maturation of the gametoid tissue might be expected to recur at periodic intervals in transplanted masses, whether in the same or in other individuals, and the infrequency of a spreading new growth as a result of the primary differentiation presents no difficulty in view of the well-known and often extreme difference in cell-vitality.

Professor Farmer and his colleagues had noted the occurrence of somatic mitoses in the proliferating cells of the advancing margin, but regarded them, as doubtless they would regard similarly dividing cancer cells elsewhere, not as the descendants but as the precursors of the cells undergoing reducing divisions. Until it has been shown that the conjugation of resting nuclei is a constant phenomenon at certain stages of tumor growth, it is not possible to prove that the somatically dividing cells are the products of the union of gametes. The data accumulated by the Imperial Research Fund on the recurrence of appearances resembling conjugation in transplanted tumors of

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mice affords a certain measure of presumptive evidence in favor of both views, though the hypothesis of the origin of cancer as a new generation is certainly more consistent with other known facts of tumor growth. It must be admitted, however, that the phenomena described by Bashford and Murray as effective nuclear conjugations are capable of bearing other interpretations, notably that of imperfect and abnormal forms of division, or merely that of abortive conjugations. It is undoubtedly a further objection that these reputedly effective nuclear conjugations occur between gametes matured not only in the same organism, but side by side in the same tissue—a circumstance which demands the assumption of a phylogenetic reversion to an extent unparalleled in biology.

The gametogenic tissue of animals does resemble a new growth in so far as its cells ultimately form highly independent individuals—independent of the needs of the organism and independent of each other. Moreover, although their growth is not unregulated and aberrant, as in cancer, it is, during the period of reproductive activity, unceasing and practically unlimited. If a similar twofold independence and unlimited growth could even for a time be acquired by a group of somatic cells, the product would not greatly differ from cancer with a limited tenure of life. And, if it be objected that the life of a cancer may, so far as we know, be unlimited, it must be borne in mind that a patient may outlive a new growth, and not rarely does outlive the less malignant forms presumably endowed with a smaller measure of vitality.

In this relation I should like to quote the views of that eminent biologist, Professor E. B. Wilson, who says: "As far as we can see from an *a priori* point of view, there is no reason why, barring accident, cell-division should not follow cell-division in endless succession in the stream of life. It is possible, indeed probable, that such may be the fact in some of the lower and simpler forms of life where no form of sexual reproduction is known to occur. In the vast majority of living forms, however, the series of cell-divisions tend to run in cycles, in each of which the energy of division comes to an end and is restored only by an admixture of living matter derived from another cell" (fertilisation); and he adduces evidence that even in unicellular animals, infusorians, the processes of growth and division sooner or later come to an end, undergoing a natural senescence, which can be counteracted only by conjugation. He concludes: "That fertilisation in higher plants and animals does, in fact, incite division and growth is a matter of undisputed observation. We know, however, that in parthenogenesis the egg may develop without fertilisation, and we do not know whether the tendency to senescence and the need for fertilisation are primary attributes of living matter" (pp. 178, 179). It is possible to find in these quotations some measure of support for each of the hypotheses under discussion. It is only fair to point out, however, that neither is regarded by its authors as more than a tentative working hypothesis, to be discarded when fresh discoveries have revealed its insufficiency, but meantime to be put to its legitimate use as a means of co-ordinating known facts and as a guide to future research.

In this relation also I would again emphasize the fact that the cells which ultimately form the reproductive tissue are of the same generation as those which go to form the embryonic envelopes (trophoblast) and those which go to form the body of the mammalian organism. They are all descendants of the fertilised ovum, derived from it by repeated mitotic division, and for a time indistinguishable one from another. It is perhaps not too much to assume that cells so intimately related in origin, however much they may ultimately differ in structure and in function, should retain certain tendencies in common, and in particular that, just as reproductive cells approaching maturity make preparations of a very special kind for conjugation with other

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specially prepared cells, so something of the same tendency has descended to the non-reproductive cells also. It is not inconceivable that such an inherited trait should be evidenced only when the other vital activities of the cells begin to cease, when the onset of senescence reveals the need for a further renewal of energy. This is not inconsistent with the fact that the higher the differentiation of the tissue of origin, the more restricted is the differentiation of its progeny in the form of a new growth or otherwise.

The occasional occurrence of heterotypical mitoses in senescent tissues, with or without the subsequent conjugation of matured gametes, might thus be regarded as the fulfilment of a natural law, as the ultimate realisation of a potentiality latent throughout the greater part of the life history of the cell, being obscured by other, and for the time being more urgent, demands on its vital energy. It is not impossible that tumor growth will prove to be a natural phenomenon determined by a definite concatenation of natural circumstances, of which one is the senescence of the tissue, and that the nature and origin of cancer will be essentially a problem of biology, and only accidentally a problem of pathology.

THE RELATION OF CHEMICAL CONDITIONS TO TUMOR GROWTH.

The intimate relation of chemical to biological processes has been demonstrated most strikingly by Loeb, whose results have been abundantly confirmed by subsequent research. It may be accepted as proven that the development and growth of living cells may be profoundly modified by slight chemical alteration in their environment. Perhaps the most remarkable of all these observations relate to the development of the unfertilised ova of certain echinoderms, notably sea urchins and starfish. In these animals the sexes are separate, and development of a new organism naturally occurs only by the union of two mature sexual cells, suspended in sea water. Loeb, however, by minor, though definite, alterations in the saline constituents of the water, was successful in causing the unfertilised ova to segment and develop just as if they had been fertilised by the spermatozoon, and in suitable circumstances the segmenting ova could be induced to form actively-swimming normal larvæ. Not only in the echinoderms, but also in annelids (segmented worms), in which also the sexes are separate, an artificial parthenogenesis could be similarly induced, so that free-swimming larvæ were developed from unfertilised eggs. In this artificial parthenogenesis only one polar body is given off, so that there is no reduction in the number of chromosomes; where there is a reduction the normal number soon becomes restored by a process of auto-regulation. (Delage.) I am indebted to Professor Haswell for this reference. That it should be possible, by a comparatively slight variation of chemical conditions, to induce in the ova of sexually developed animals a reversion to a non-sexual form of development (parthenogenesis) is not without possible significance in relation to neoplasia.

These phenomena are not merely scientific freaks. They acquire a new importance in view of the work of Professor Moore and his colleagues on the reaction of the gastric secretions in cases of malignant disease. As these authors indicate, it was one of the few definite facts known about cancer that, when the stomach was the seat of the new growth, the secretion of free hydrochloric acid was nearly always in abeyance, and it was also known that this might occur even when the growth was of small dimensions and had not produced any other noticeable alteration in the stomach. I do not propose to discuss the various suggestions that have been advanced in explanation of this phenomenon, nor the observations made on the effect of new growths in different positions of the stomach, nor the occurrence of diminished acidity in other morbid conditions.

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It occurred to Professor Moore and his colleagues to investigate whether this phenomenon was independent of the site of the cancer, and they found—I give the findings in their own words—that " the absence of free hydrochloric acid in cancer of the stomach is not due to any local action in that organ, for hydrochloric acid is absent or greatly reduced in amount whatever may be the situation in the body of the malignant growth. The amount of free hydrochloric acid present in the stomach contents was determined in seventeen cases of malignant disease situated in different regions, such as the uterus, mamma, prostate, rectum, tongue, cheek, and mouth, and in about two-thirds of the cases the free hydrochloric acid was found to be entirely absent, while in the remaining cases the amount found was much below the normal, being reduced to a mere trace in all except one case, where it reached about one-fifth of the average amount obtained in control determinations carried out upon ourselves." Since the phenomenon is independent of any local action in the stomach, and since, moreover, it is so pronounced and constant, so far, at least, as the limited number of observations recorded can justify such a statement, it certainly suggests a profound chemical alteration in the body fluids, and, as the authors point out, this change may be a common cause of the new growth and of the absence of the acid, or it may be a result of the growth and the cause of the absence of the acid. The cause of the absence of the acid, however, is not yet determined. It may be that the oxyntic cells are atrophied, or inhibited by some toxic substance in the blood, and the presence of a ferment (malignin) within cancer cells, and liberated by their disintegration, has been described. (Petry and Blumenthal.) Nevertheless, it is more probable that the explanation must be sought in the chemical constitution of the blood itself, and that the elimination or arrest of free hydrochloric acid is an indication " through the mechanism of the oxyntic cells " that the essential acidity of the blood is decreased and its essential alkalinity increased. Dr. G. E. Rennie has suggested that an increased alkalinity of the blood plasma might be determined by some disturbance in the nervous control of metabolism such as is indicated in the contrasted condition of acidosis and diabetes. These facts and considerations further illustrate the extended scope of modern pathology and its relation to, and foundations in, biology, chemistry, and physics. I much regret that time does not permit me to discuss other recent contributions, notably those of Ribbert, of Beard, and of the many cancer research institutes.

INDICATIONS FOR THE TREATMENT OF NEW GROWTHS.

When the cells of one animal are repeatedly introduced within a second animal of a different species, it frequently happens that substances antagonistic to these cells are developed in the body of the inoculated animal. If, now, some of the blood serum from the second animal be injected into an animal of the same species as that from which the cells were originally taken, the serum may exert a destructive action on these cells. It is upon this general biological law, governing the ultimate reactions of living cells to alien albuminous molecules, that the principles of immunisation against bacterial infection are based.

Whether we regard a tumor as a new generation of cells or not, the fact remains that it constitutes an invasion of the organism by a brood of independent and hostile cells, and, in the sense in which we have defined the process, it is equivalent to an infection. It seems not unreasonable to hope that substances antagonistic to the cells of a new growth could be developed in the blood of an otherwise suitable alien animal by injections of the tumor cells, as in the process of immunisation, and that the blood serum could then be used with effect either to inhibit or destroy further growths of the same tumor or growths of other similar tumors. The extremely restricted specificity of the action of

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living tissues to cancer cells, so that evidence of a difference is obtained among closely related cancer cells from the same species (Scientific Reports, No. 2, part ii,, p. 33), indicates a possible difficulty in the preparation of a serum antagonistic to other new growths of similar structure to that used in its preparation, though much distinction might not be apparent when injections were made into alien animals. Already considerable attention has been paid to this possible development, but as yet no certainty has been attained.

The remarkable agencies revealed within the last few years by physical science have been impressed into the service of medicine, and their influence on tumor growth is now on its trial. The effect of radium on transplanted new growths of mice has been investigated by the Cancer Research Fund, and, since the life history of these tumors has been under continuous observation for several years, the results obtained may be regarded as independent of the numerous sources of error which discount the value of merely clinical experience. Moreover, as the experimental results corroborate the earlier clinical observations, a considerable body of evidence has now accumulated in support of the fact that radio-active bodies and solutions do in a most surprising and apparently capricious manner cause the disappearance of some tumor growths, though not of all.

Pieces of radio-active lead, solutions of radium-emanation, and sterile radio-active saline solutions were used. The duration of each exposure varied from three to thirty minutes, and was repeated for the same length of time on the following days. Within the second week after the first exposure, four of the tumors began to diminish in size, while others continued to grow. There was no noticeable relation between the length of exposure and the effect on the tumor. The influence of radium in producing absorption appeared to be directed to the connective tissue elements, which underwent marked proliferation, and split up the tumor into smaller islets of cells. Even at this stage the tumor cells appeared healthy, but within a few days they degenerated, then died, then disappeared, probably owing to cicatricial contraction of the fibrous overgrowth. These observations confirm the previous work of Exner, and controvert the more common assumption that radium exercises a selective action on the tumor cells.

The Röntgen rays (X-rays) have also been employed in the treatment of cancer, and some measure of success has been recorded in superficial forms. I am not aware, however, that their influence has been submitted to any such rigorous test as that undertaken for radium. It is known, through the extensive researches of Heinecke (quoted in the Second Scientific Report, part ii., p. 61), that exposure to the Röntgen rays or to radium produces alterations in the spleen and lymphatic glands, and several accounts have recently appeared of the successful treatment by X-rays of leucocythæmia—a condition recognised to be closely related to, if not identical with, neoplasia. Only last month Dr. Reissmann, of Adelaide, published an illustrative case.

I have grievously failed if I have raised any false hopes that the treatment of cancer by any other means than actual removal is yet practicable. It is obvious that to be effective the removal must be early, free, and careful; and the lamentable disregard of one or other of these precautions is responsible for many disasters. Patients should be encouraged to seek advice in the early stages of suspicious tumors, and the surgeon should remember that these are not cases for expectant treatment nor for conservative surgery. I do not advocate wholesale butchery, but I do advocate the common-sense application of known facts and scientific methods to the surgical treatment of tumor growth. In these days there is no reason why every doubtful tumor should not be submitted to a microscopic examination. If you are still in doubt whether the condition is inflammatory or malignant, give the patient the

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benefit of the doubt and remove the suspicious mass. Remember that prolonged irritation induces senescence of tissue, and predisposes to neoplasia. If you are in doubt whether the growth is malignant or benign, remember that all are potentially malignant, that the difference is one of degree, not of kind, and do not hesitate to remove it. If you are satisfied that a malignant growth has really started, bear in mind the ease and rapidity with which secondary infections may occur, and do not risk losing a life for the sake of saving a limb. The knife should never be within an inch of the new growth, and, where possible, many inches should intervene. I do not need to warn this audience against those secret nostrums which, born of cupidity and nurtured in credulity, if they do not aggravate by irritation, serve only to delude the patient into waiting until perchance the time for operative interference has passed.

ADDENDUM.

Since this address was delivered, I have been repeatedly asked whether there is any foundation for the not uncommon belief or suspicion that cancer is contagious or infectious. While I am fully aware of certain recorded instances of the communication of cancer from one person to another (*cancer a deux*), and of the existence of so-called cancer houses and cancer districts, I cannot too strongly emphasize the fact that the difficulties attendant on the artificial transference of new growths sufficiently demonstrate that cancer is neither contagious nor infectious. The hypothesis that cancer is due to the invasion of parasitic microbes is far from being substantiated, and, even if it should later be proved that the phenomena of cancer are initiated by some form of parasite, it does not necessarily follow that cancer is contagious or infectious. On the contrary, it must be admitted that the distribution of the presumptive parasite and its mode of action are wholly dissimilar to those of any known bacterium or protozoon, and that in particular it is not capable of reproducing the disease in other persons after the manner of other morbific agents known or assumed to exist.

NOTE ON A LARVAL TAPEWORM FROM THE HUMAN SUBJECT (BOTHRIOCEPHALUS MANSONI, OR LIGULOIDES).

By Drs. MacCormick and Hill.

About three and a half years ago a young man, A. S., aged 27, married, a plumber and gasfitter, residing at 27, John Street, Tempe, a suburb of Sydney, was treated by me in the Royal Prince Alfred Hospital. This man never lived out of Sydney, with the exception of a fortnight's holiday at Penrith, some forty miles from Sydney, six or seven years ago. He was employed during the previous eight years in a cocoanut oil factory in Balmain. The cocoanuts came from the South Pacific Islands.

Twelve months prior to admission to the hospital what was said to be a fatty tumor was removed from under the skin in the upper part of the right lumbar region, at the costal margin. One month afterwards he noticed a similar swelling 2 inches lower down. This swelling was soft, and fairly circumscribed, and about the size of a hen egg. This tumor was removed by dissection, under chloroform. On making a section through the part removed it appeared to be soft granulation tissue, in the centre of which was a white body, which, on further investigation, was found to be a worm about one and a half inches long, of the color of lard, and possessing feeble movement. On August 22nd, 1905, patient was examined, and no trace of any

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further disease could be detected. During the intervening three and a half years which had elapsed since the operation he enjoyed good health. The worm was sent to Dr. Hill, of the Zoological Department of the University, for investigation.

Manson, in his book on Tropical Diseases, mentioned the fact that this parasite is found in Bathurst, in Australia. The late Dr. Spencer published an account of two cases which had occurred in his practice in Bathurst; and possibly Manson may be referring to these cases. In Spencer's cases the worm apparently gave rise to a diffuse abdominal tumor, containing some fluid; and both of these cases ultimately died.

This parasite, according to Manson, has its habitat in the sub-peritoneal fat, and thence may find its way into the bladder or pleural cavities. It has been frequently passed per urethram. As far as I am aware Spencer's cases and my own are the only instances in which this parasite has been met with in Australia, and the tumor that it causes from its mere rarity must naturally be a matter of difficulty in diagnosis.

NOTES BY DR. J. P. HILL.

The worm proves, on examination, to be an immature sexless tapeworm, or larva of the "plerocercoid" type. So far as we are able to judge, our specimen is apparently identical with *Bothriocephalus Mansoni*, originally found by Manson (1) in a Chinaman at Amoy, and described by Cobbold (2) as *Ligula Mansoni*. Leuckart (3) has also examined a specimen of this parasite, and has given a detailed account of its structure. He showed that it was more closely allied to the genus *Bothriocephalus* than to *Ligula*, and gave it the name of *Bothriocephalus liguloides*: the parasite is, accordingly, now known as *B. Mansoni*, Cobbold. The adult form is still unknown, or, if known, its connection with the larval form has not been established.

On removal from the mass of granulation tissue in which it lay embedded, the worm exhibited distinct, but feeble, movement. On transference to alcohol it underwent strong contraction, and, unfortunately, broke into several fragments. In the living condition its total length is estimated at about 3.5 cm.: in spirit the fragments give a total length of 1.5 cm., and a greatest breadth of 1.5 mm. It is thus very much smaller than Manson's specimens, the length of which is given as about 30–35 cm.

Cobbold's figure gives a fairly accurate view of the general form of the organism. The narrow elongated body is flattened and unsegmented, and, in the preserved fragments, is seen to be crossed by numerous transverse wrinkles. It is broadest just behind the bluntly pointed anterior end, and tapers gradually posteriorly. The head is not marked off from the succeeding portions of the body: it is small, and only partially protruded. On its flat surfaces are recognisable the two longitudinal suctional grooves, or "bothria," characteristic of the family *Bothriocephalidæ* (cf. Leuckart's fig, 404).

Transverse sections through the mid-region of the body of our specimen show a general agreement with Leuckart's figure and description. The cuticle is a relatively thick layer, to the outer surface of which fragments of the enclosing granulation tissue have remained firmly adherent. Immediately within the cuticle the dermal musculature consists of the usual circular and longitudinal fibres, the latter being well marked. The subcuticular cellular layer presents the usual characters. The ground parenchyma tissue consists of a reticular vacuolated matrix, with scattered nuclei; cell outlines are not readily distinguishable in the preparations. It is uniform throughout its extent, there being no distinction into cortical and medullary zones, owing to the absence of distinct bundles of circular muscle. The muscle bundles present in it are small, and show no definite arrangement or

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grouping—many of them run dorso-ventrally. There are no definitely arranged bundles of longitudinal muscles such as are found in the closely allied larval tapeworm (4) occurring amongst the superficial muscles of the thigh of the common frog, *Hyla aurea*.

The vessels of the excretory system are, as noted by Leuckart, extensively developed and of large size. The "lime" cells are readily recognisable in the sections—they are relatively large, and not very numerous. Unfortunately the calcareous corpuscles themselves were not observed, as all the fragments of one single specimen were inadvertently treated with acid after staining.

As regards the life-history of *B. Mansoni*, we are as yet completely in the dark. It seems probable that man is only an accidental, and not the regular, host of this larval form.

(1) Manson, *Lancet*, October 14th, 1882: Tropical Diseases, pp. 601-2, and fig. 98.
(2) Cobbold, *Jour. Linn. Soc. (Zool.)*, vol. xvii., 1884.
(3) Leuckart, *Parasites of Man*, p. 745, *et seq.*, figs. 402-4.
(4) Haswell, *Proc. Linn. Soc. N.S.W.*, vol. v., 2nd series.

TRYPANOSOMIASIS.

By E. Angas Johnson, M.D., M.R.C.S.

Mr. President, Ladies, and Gentlemen—In the course of my peregrinations into pathological laboratories abroad, nearly always have I found that parasites (with a few exceptions) are almost ignored. It may be that investigators do not care for the disgusting surroundings (not so for a zoologist) in which parasites are found, or have not opportunities; or, more frequently the case is they do not care about them.

In Australia only a few continue their study of pathology. the majority of medical men are content with the rudiments they learnt to pass their examinations. Why is this thus? The simple reason is, this fascinating study takes time as well as money; and, as Governments do not look with favor on expenditure of money for research, few are in the position to devote their time and money for investigation.

Parasitology is practically ignored. However, the Liverpool and London schools of tropical medicine are now awakening to the importance of parasitology and devoting much time, skill, and money to its elucidation.

It behoves us in Australia now to energetically direct attention to this subject, because communication with any part of the world is only a question of weeks, and soon, with turbines, it will be days; besides, when the Panama Canal is opened no doubt many diseases of the tropics will find their way through this channel direct to Australia.

We know all over the world that history repeats itself, and "crass ignorance" on the part of so-called experts has frequently resulted in a disease getting a foothold in a country; and then enormous sacrifice of life, with a corresponding necessary lavish expenditure of money to combat the epidemic, takes place. We know that malaria has been taken to virgin lands in the following manner:—A patient suffering from malaria is on board a ship; in the water-casks are anopheles in all stages of development, and these become infected from the patient and then infect other people. The new land is reached, conditions are favorable, and so the disease takes root.

Frequently the source of infection is present in a land, but the intermediate host is wanting: and the contrary holds true.

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Here in South Australia we have the anopheles, and, from Mauritius, we often get malaria subjects through the sugar trade; and unless we take great care this place will be noted for malaria. Tropical dysentery was unknown here before the South African war; last year there were several small epidemics near Port Adelaide. What diseases may the soldiers introduce in their saddlecloths, or in monkeys hidden under their greatcoats? Now the horses are not allowed to return, but the saddlecloths are. This is "crass ignorance" on the part of the Commonwealth experts.

Foreign animals are constantly being imported for domestic pets, circuses, and zoos. Think of this danger! Think of the diseases introduced into America from Africa 200 years ago when the slave trade flourished.

In 1850 there were as many centenarians, in proportion to the population, in Mauritius (in spite of its being a tropical country) as elsewhere in the world; then malaria and trypanosomes were unknown there. Now, fifty-five years afterwards, the place has almost been devastated through these agencies, in spite of its being an excellently ruled British colony; and so the proportion of centenarians has naturally diminished—in fact there are less in Mauritius, in proportion, than elsewhere in the world. What can we expect in Australia, unless drastic preventative measures be adopted at once?

As I remarked previously, parasitology has almost been ignored. When I reported a case of trichina here three or four years ago many medical men doubted it, although shown the specimens—and here they are!

Dengue fever has played havoc lately in Queensland, and possibly will come here, through the train; then the trypanosome will work havoc in our horses unless we protect them. Now, as this whole subject of trypanosomiasis is in its infancy, and still *sub judice*, it is not necessary for me to go over the whole of the ground; but, as I have had experience with it in three different laboratories abroad, and having had time to conduct original research work myself, I will endeavor to give you a few facts from an unbiased point of view, and let you draw your own conclusions. Where an observer has quoted a discovery I have used his own words as far as possible, but most authors in the case of the trypanosome have always referred me to Laveran and Mesnil, who have so exhaustively reviewed this subject in a book of 400 pages, and this work I have freely consulted.

PROTOZOA.

1. Subphylum. Plasmadroma.

Class I.—Rhizopoda orders 1-5.

Amoebina, Radiolaria, Helizoa, Foraminifera, Mycitozoa.

Class II.—Mastigophora. Sub-class Flagellata. Orders 1-5.

1. Protomonadina. Family Cercomonadina. Family Trypanosomidæ.
2. Polymastigna (Trichomonas Vaginalis). Lamblia.
3-5. Englenoidina, Chromonadina. Phytomonadina.

Class III.—Sporozoa. Sub-class 1. *Telosporidia.*

Order I.—Coccidiomorpha.

Sub-orders.—(1) Coccidia. (2) Hæmospoirdia. (3) Piroplasmata.

Order II. Gregarinida. (ii.) Neosporidia. (ii.) Subphylum. Ciliophora

Class IV.—Ciliata.

Class V.—Suctoria.

This is Professor Nuttall's classification, and shows where the trypanosome is placed.

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THE MORE COMMON VARIETIES OF TRYPANOSOMES MET WITH IN MAN AND ANIMALS.

Common Name of Disease.	Species of Parasite.	Discovered by—	Geographical Distribution of the Disease	Propagation of the Disease.	Pathogenicity.
Dourine (mal du coit)	Tr. equiperdum	Rouget, 1894	Nebraska, Wyoming, S. Dakota, S. Europe, Turkey, Asia Minor, N. Africa, Hungary, Spain, Java, and Switzerland	Coitus	Same as surra
Galziekte (gall sickness)	Tr. Theileri	Theileri, 1902	S. Africa, Rhodesia, Togo, Mafia (German E. Africa)	Hippobosca rufipes	Cattle only
Trypanosome fever.....	Tr. Gambiense	Forde & Dutton, 1901; Dutton, 1902	Equatorial and W. Africa	? Glossina palpalis. ? Tabanus dorsovitta. ? Fleas and lice	Monkeys, rabbits, guinea-pigs, rats, dogs, sheep, goats, &c.
Rat trypanosome.....	Tr. Lewisi	Lewis, 1878	World wide	Fleas and lice	Occ. guinea-pigs
Mal de caderas.....	Tr. Equinum	Elmassian, 1901	S. America	? Tabanus ? Stomoxys	Same as surra
Nagana (tsetse fly disease)	Tr. Brucei	Bruce, 1895	S. and W. Africa	Glossina morsitans	Same as surra
Surra	Tr. Evansi	Evans, 1880	India, Burmah, Persia, Mauritius, Philippines	? Tabanus tropicus (horse flies) ? Stomoxys calcitraus (stable flies)	Rats, monkeys, all domesticated animals, and nearly all wild ones

The distribution of glossina palpalis and sleeping sickness in Uganda is identical. Where there are no flies, there is no sickness.

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Classification of the genus Trypanosoma by Salmon and Stiles (Greek Trupanon—borer, soma—body).

Class, Mastigophora. Sub-class, Flagellata. Order, Monadida.

Independently living organisms, but in some cases they form colonies. The number and arrangement of the flagella varies. There is sometimes an undulating membrane. *Family*—Trypanosomidæ includes the genus trypanoplasma, parasitic organisms resembling the trypanosomes, but possessing two flagella, one of which is situated at each end. *Genus*—Trypanosoma. *Species*—Tr. rotatorium (Mayer), Tr. Lewisi, &c. Trypanosomiasis is a group name, and it belongs to the protozoa. (1) The trypanosoma, characterised by having a longitudinal undulating membrane, which arises posteriorly from the centrosome, and it ends as a free flagellum anteriorly. Division takes place by longitudinal division. (2) The trypanoplasma has an anterior flagellum, which forms the thickened border of the undulating membrane, and a posterior flagellum, which curves round the posterior end of the parasite, and is free, being about the same length as the anterior one, and both arise from the same centrosome.

Trypanosomiasis is a term used to denote a specific infection in birds, fishes, reptiles, mammals, and amphibia, and it is universally distributed. I have collected eighty-two terms which designate it. It is endemic as well as epidemic over large tracts of tropical and sub-tropical countries, being worse during the rainy season.

In 1841 Valentine discovered the trypanosome in the trout (*salmo fario*), and in 1842 Grube gave it its definite name. From 1843 to 1850 trypanosomes were found in frogs, rats, moles, and field mice. In 1878, Lewis, whilst working in India, discovered the trypanosomes in rats, and this served as a starting point for investigation. Harmless in rats, and presents, as far as we know, no definite symptoms. In nature we only see the survivors of the fittest; so, if young rats are susceptible, they might die of an acute disease. It is distributed world wide. From 15 to 95 per cent. of rats affected. Rats may harbor the disease for two years and yet show no symptoms. Fleas transmit it in rats, as proved by Rabinowitsch, and she thinks it possibly may be a repeated infection. The rat trypanosome has been kept on ice for forty-five days and retained its virulence; at fifty degrees C. for two hours, it is still infective. It has been observed in fleas and bugs twenty-four hours after their sucking blood; acquired immunity is active, and due to antibodies; it has a specific agglutinating power on the trypanosome. Incubation period is three to five days.

The filtrates of cultures of Tr. Lewisi that have passed through a Berkefeld filter are infective, and Schaudin thinks that, as the trypanosome (male) of Spirochaeta Ziemanni is so small, it could also pass through a Berkefeld filter. (Smedley thesis, M.D., Cantab.) Cultural trypanosome of rats differ considerably from those of the adult parasite found in the blood. They are:—(1) Very motile and also spindle-shaped. (2) Centrosome placed anteriorly. (3) The flagellum is very long and active, and has but a short intra-cellular course, on account of the position of the centrosome. (4) No undulating membrane. (5) These trypanosomes form colonies, which ultimately contain many thousands of individuals.

Surra struck Mauritius in 1902-1903, and killed off 100 per cent. of the horses and 25 per cent. of the cattle attacked. It is limited in Java on account of very severe legislation.

Musgrave and Williamson (Trypanosome of Horses, Department of Interior, No. 3, p. 10) say that in 1901 some twenty-six horses came direct

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from Australian ports to the Philippines, and one was ill at the time of arrival and three others died later from surra If this is correct we might at once start investigations. (Professor Watson denies this statement; he says the horses were Australian, and had been shipped to India and thence to Hong Kong, and from there to Manila, and that the infection took place in India.)

A number of extremely interesting immunising experiments have been made by several competent observers, and these have been most helpful in separating the types :—

Expt. 1.—Laveran and Mesnil immunised a goat successfully to nagana, mal de caderas, and surra, and yet it died with the horse trypanosome of Gambia (*tr dimorphon*).

Expt. 2.—Nocard, Vallee, and Carre infected a cow with surra, which was immune to nagana.

Expt. 3.—Nocard killed two dogs with nagana that were immune to dourine.

Expt. 4.—Lignieres killed two dogs with mal de caderas which were immune to dourine.

So that these immunising experiments have helped to clear up the difficulty in almost definitely distinguishing between surra and nagana, as well as between dourine, surra, and mal de caderas.

But there is this fact to be borne in mind, that there are variations of susceptibility of the same species of animals in different parts of the world that are confusing. Previously one had to depend on minute differences in the morphology between the various trypanosomes causing the various diseases; but there are differences almost as great existing between individual members of the same species.

Dourine (*mal du coit*).—Rouget discovered the trypanosome of this disease in 1894. This disease is only propagated by coitus, and some authorities affirm that the Tr. equiperdum can bore its way through an intact healthy mucous membrane. When an animal is affected the extremities become paralysed, especially in the hind limbs, and the penis and scrotum in a stallion gets very much swollen. If so, castrate all stallions and kill all mares affected.

Nagana (Tsetse fly disease).—First seen in the Zambesi basin, in 1857, by David Livingstone (*British Medical Journal*, May 1st, 1858), and described by him. Laveran and Mesnil say his description is very precise and scientific for the time at which he wrote. He used to treat his horses with arsenic in 2-grain doses daily for a week. This relieved them (he says cured) for a time; but they all died within six months afterwards. This disease is chronic in bovines, sheep, and goats, as well as in many wild animals in Zululand, although often the parasite cannot be found in their blood; but inoculate a tame animal from a wild one and usually one can reproduce the disease.

The Tsetse fly (*glossina morsitans*) (Austen tabulates eight varieties of the *glossina*).—A stinging fly, not much larger than our common house fly, is the actual carrier of the disease. It is a mechanical transference, because there is no sexual process in the fly, since forty-eight hours after sucking the blood of an infected animal they are non-infective. These flies are found in little hollows where wild animals go, and wild animals infect these flies. Sambon disagrees with this, and thinks that perhaps the fly is an intermediate host; otherwise why do not tabanus and stomoxys spread the disease mechanically ?

Gray and Tullock have confirmed Sambon's views, that the parasites undergo a cycle of development in these flies.

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Compare the mosquito (*stegomyia*), which is the actual carrier of yellow fever; it takes twelve days to become infective, and in malaria, where it is the anopheles, it does not become infective for ten days (Bruce).

Mal de caderas.—The centrosome of tr. equinum is very small, and stains very slightly, thus differing from nagana and surra. Fatal to mice, rats, hedgehogs, armadillos, otters, monkeys, field mice, dogs, rabbits, coati, sheep, guinea-pigs, and cats. The goat and ox seem to be immune, but their blood is able to harbor the parasite for months.

In South America there are epidemics of this disease concurrent with epidemics in animals, like guinea-pigs (*carpinchos hydrochoerus capibara*), which are natives there. These small animals live on the banks of the Paraguay; they are hunted for their skins; the dogs eat their carcasses and become infected with trypanosome equinum. The horses become infected, but not known how, as there is no biting fly known. Fleas and lice?

Human Trypanosomes.—Tr. Dutton, Tr. gambiense. In Bombay it is known as epidemic general congestive dropsy. In Africa as splenomegaly. Winterbottom first described it (1803). Clarke noted it in Sierra Leone (1843). In 1890 Nepveu at Algiers found flagellates in the blood of a malarial patient: he called them trypanosomes, but failed to classify the variety. Ross did not know whether Dutton's Trypanosome approached Tr. Lewisi or Tr. Brucei.

Lancet, vol. 1, January 11th, 1902, p. 107.—Dutton wrote from Bathurst (Gambia) to Ross about a European male who was under Dr. Forbes' care, which Dr. Forde thought was filaria, saying that:—"(1) The patient suffered from a relapsing fever although the temperature never was very high. (2) Was general weakness and wasting. (3) Local œdemas, especially around the eyes and legs, and general puffiness of the face. (4) Increased frequency of pulse and respiration. (5) Congested areas of the skin. (6) Enlargement of the spleen. (7) Chronicity of the case and no lesion of the heart or the kidneys detected."

Dutton then examined 150 apparently healthy children, between 1 and 15 years, in Gambia, and in the ninth case found the trypanosomes, although there were no clinical signs or symptoms present. He also found it in a child, age 3, whilst looking for malaria (ring forms), which was also present. This is the smallest of all known mammalian trypanosomes, its average length, including the flagellum, is 22 microns, average breadth 2–2.8 microns, *i.e.*, the breadth is greater in proportion to its length than in other animals. In fresh blood, with a one-sixth objective, one can see actively motile trypanosomes when present, moving between the corpuscles, and when they come to rest one can see the flagellum and the undulating membrane. The flagellum is intimately connected with the micro-nucleus, and runs along the free margin of undulating membrane situated at the anterior end; it stains a light-crimson, and is about one-third of the total length of the organism. Where does the anterior part of the body end, and the free part of the flagellum begin? Because one can always see a narrow streak of the protoplasm staining blue for some distance beneath the free part of the flagellum. The body is usually bent or curved at an angle opposite the nucleus. The anterior end is pointed, the posterior end blunter. The protoplasm does not stain evenly, taking on a basophile reaction; in some specimens one sees fine blue granules, which are usually situated around and in front of the macro-nucleus; several vacuoles may be seen as circular clear spaces. The undulating membrane is a narrow band, wavy, attached along one side, staining a faint pink. The nucleus (macro-nucleus of Plimmer and Bradford) is situated a little anterior to the middle of the body, round or oval, stains a dark-crimson, due, in older specimens, to the aggregations of chromatin

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granules; sometimes it occupies the whole of the animal. The centrosome (micro-nucleus) (Bradford and Phimmer). Usually circular or elongated; about 2.5 inches from the posterior end it appears as a dark-red spot; there is nearly always a vacuole adjacent to it, sometimes close to the nucleus, sometimes anterior to nucleus (Laveran and Mesnil).

British Medical Journal, May 30th, 1903.—Manson had a case, the wife of a missionary from the Upper Congo (the tenth case on record). The blood was examined, negatively, daily for two weeks, until Dr. Daniels found the trypanosome. Manson thinks Argas monbata (a tick) is the carrier.

Of the sixteen cases reported in man, two were in an apparently healthy condition; six associated with malaria; eight had clinical symptoms of trypanosomes, and of these eight five gave similar symptoms to one another.

Castellani found trypanosomes in the cerebro-spinal fluid in twenty out of thirty-four cases. Bruce got it in lumbar puncture in thirty-eight out of thirty-eight, and, of these latter cases, in the blood in twelve out of thirteen. Bruce considers that all the stages of development of the trypanosomes take place in the human host. Leishman has reported the possible occurrence in the blood of trypanosomes in a case of dum-dum fever.

Blood examination showed no marked anemia, no dividing forms met: red blood corpuscles, 3,850,000; white blood corpuscles, 12,000 per c.c.m.; lymphocytes increased at expense of polymorphonuclears in proportion of 50 per cent. of latter to 40 per cent. of former. Cultivation of trypanosomes (Trypanosome Lewisi).

Journal of American Medical Association, pp. 1266 to 1268.—McNeal and Novy used a mixture of defibrinated blood and agar at 45 degrees C., equal parts. Slant the tube to get fluid of expression (trypanosomes only grow in this fluid of expression). Incubated at 37 degrees C. the cultures grow quickly, but rapidly degenerate owing to the alteration of the blood medium. At 20 to 25 degrees C. in the cool incubator, or 18 to 20 degrees C. in a dark room, get numerous colonies at the end of three weeks, and they remain alive for several months; put on caps to the tubes to prevent evaporation. Also have grown try. Brucei, surra in Philippines, during 1904. Tr. Lewisi has been cultivated for twenty-six generations during two years, and still retained its vitality. This is a most remarkable advance in science, to be able to grow protozoa artificially in a test tube, and opens up a very wide field in the future.

British Medical Journal, September 17th, 1904.—Rogers described the development of flagellated organisms from Leishman's bodies, in kala azar. He used sodium citrate; 5 ccm. of blood with 1 ccm. of 5 per cent. sodium citrate soln to prevent coagulation, and kept the mixture at 22 degrees C. He saw a multiplication of the parasite, and their conversion into flagellate-like organisms, without an undulating membrane.

Lancet, January 7th, 1903.—Chatterjee repeated Rogers' experiment successfully. On first day he saw three to four Leishman-Donovan bodies in every field of a stained specimen. On second day 200 to 300 in each field; the centrosome was larger, and the cell contents were more granular. On third day elongated bodies with flagella could be seen, and later fully-developed trypanosome-like bodies. The parasite differed from the ordinary trypanosome in the following respects:—(1) Micro nucleus was at anterior end. (2) Long thick anterior flagellum. (3) Absence of undulating membrane. (4) Rudimentary post flagellum. Compare Smedley's experiments at Cambridge, previously reported in rat trypanosome; practically identical with Rogers' kala-azar experiments.

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Staining sections, Halberstædt—

1. Fixed tissues, twelve to fourteen hours in glacial acetic acid and a concentrated watery solution of corrosive sublimate, *e.g.* :—Mercuric chloride conctd. watery solu., aqua destillata a.a. 1500, acid glacial acetic 40; wash in running water twenty-four hours, embed in paraffin.

Stain thin sections with polychrome methylene blue ten minutes, wash in water. Absolute alcohol—Xyol very quickly. Tissues very deeply stained blue, trypanosomes pinkish.

2. Leishman's stain.—If the film is old, recoat it with human serum, stain with Leishman's stain; can wash off, if not satisfactory, with absolute alcohol, and then restain with Leishman's stain; also can apply it for tissues in section.

3. Hæmatoxylin.—The best for permanent specimens.

4. Weak carbol.—Fuchsin 1 to 10, stain ten minutes for diagnosis.

Prophylaxis.—(1) Kill all infected animals. (2) Kill all rats and mice. (3) Kill all stinging and biting insects. (4) Kill all game and wild animals. (5) Kill all fleas, lice, bugs. (6) Sunlight kills (Laveran and Mesnil).

Blood-sucking insects are the carriers and are not the intermediate hosts. Schaudinn says that the halteridum in the owl is the sexual stage of a trypanosome (Tr. noctuæ), which undergoes a complex form of multiplication both in the gnat and in the blood of the owl, giving rise to the sexual forms of Halteridum.

Treatment (*British Medical Journal*, Thomas, 1905, p. 1140).—Laveran uses sodium arsenite for animals. Boden Fowlers' solution in homo, with very promising results. Ehrlich trypanroth, especially in mal de caderas; but it causes nephritis and necrosis if given in big doses. Wendelstadt malachite green; too dangerous, on account of necrosis. Arsenic kills trypanosomes free in the blood, but it must be repeated if it gets eliminated or fixed in the tissues, else they start multiplying at once.

Atoxyl-aniline C6 H6, No. 2, is very good, because less toxic, and causes no necrosis. A combination of trypanroth and atoxyl in intra-muscular injections is very good, and very little necrosis results. Serum therapy—With ordinary human serum death is retarded. No cure for disease.

SLEEPING SICKNESS.

British Medical Journal, August 20th, 1904.—Dutton, Todd, and Christy classify cases according to symptoms. 1. Cases with no definite symptoms. 2. Cases with a few symptoms (temperature rises in evenings). 3. Fatal cases with marked symptoms, with sleep symptoms, no sleep symptoms. (*a*) Fever. (*b*) Lassitude. (*c*) Weakness. (*d*) Wasting.

The patient may harbor the trypanosome one year without symptoms. When symptoms develop the patient lasts two to four months. Secondary infections are common, thirteen out of thirty-two *post Mortems* showed this, especially purulent meningitis.

The number of parasites found bears no relation to the severity of the disease.

Post Mortem.—Trypanosomes found in pericardial, pleural, and peritoneal fluids; not infective to animals (Stephens).

British Medical Journal, ibid.—Manson reported that the entire population at the southern end of Victoria Nyanza, in German territory, is affected with trypanosomes, yet no sleeping sickness there.

If this is so, then trypanosomes cannot be the only cause of this disease. It is a determining cause, the active cause being bacterial—a streptococcus in Castellani's case.

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Ibid.—Bruce; trypanosomes found in the blood of natives on the west coast of Africa and in Uganda; those found in sleeping sickness are identical, proved by experiments on animals.

Trypanosome fever is the first stage of sleeping sickness; neither native nor European is immune. In fifty-four out of seventy-four cases parasites found in cerebro-spinal fluid.

In the Congo district the symptom of sleep is an inconstant factor of the disease, so the term is a misnomer. The patient's expression is dull and stupid. In very advanced cases the patient dozes over the fire; he can be roused by shouting, and then relapses; eats nil. If trypanosomes get into the cerebro-spinal fluid early, get mania; mortality 100 per cent. in natives and Europeans. Sometimes natives who have trypanosomes in their blood recover, because the trypanosomes seem to get diseased and die.

Manson.—Compare Dr. Bowden's case with trypanosomes for three years and now quite recovered.

Experiments.—Have infected rats, mice, guinea-pigs, rabbits, and monkeys. Used 1-5 ccm. of infected cerebro-spinal fluid; 50 per cent. of cases negative.

Prophylaxis.—Forbid natives from sleeping sickness areas moving into any part of the country where any species of the tsetse fly is found.

In Uganda the distribution of glossina palpalis and sleeping sickness is identical; where no flies, no sickness.

Treatment nil so far, as all cases die.

THE LEISHMAN-DONOVAN BODY.

This disease is widely distributed, particularly throughout India, in Egypt and Northern Africa, and in China. In 1885 Cunningham saw these bodies in the granulation tissue from a tropical ulcer. In 1903 Wright found them in a tropical ulcer.

British Medical Journal, May 30th, 1903.—Major Leishman discovered these bodies in a film of splenic blood from the *post mortem* on a dum dum, or dam dam, fever patient. Also found in Delhi boil, Aleppo button, and Scinde sore; and all of these parasites are identical. Marchand and Ledingham discovered (in 1902) these bodies in sections of spleen, liver, and bone marrow from a patient who had been a resident in the tropics and had an enlarged spleen and irregular fever. One is particularly cautioned to look for them in cases of malaria cachexia, with an enlarged spleen, when the malaria parasite is absent.

The Characters of the Fever:—(1) The spleen is markedly enlarged, descending to the umbilicus, or even to the pelvis. (2) Irregular pyrexia—reaching to 103 to 105 degrees F. (3) Emaciation. (4) Abdominal symptoms are common—(*a*) Diarrhœa is very constant; (*b*) peritonitis from perforation of an ulcer in the intestine, three to seven cases fatal (Christophers). (5) Local phagedænic ulceration fairly common complications, *e.g.*, cancrum oris and noma vulvæ. (6) Skin eruptions. A papular eruption in the region of the thighs and scrotum in advanced cases. Papules on trunk and limbs. Small ulcers occur. (7) Hæmorrhages. Epistaxis, bleeding from the gums, purpuric eruptions. P.M. serous membranes affected.

Post-mortem Lesions.—Spleen enlarged and of firm consistence, may or may not be pigmented. Liver usually not much enlarged; aboresence on section, due to deposit of macrophages containing pigment in centre of lobules. Large intestine.—Extensive multiple ulceration. Cancrum oris, noma vulvæ, purulent peritonitis, broncho-pneumonia, septic infarcts, &c.

British Medical Journal, September 17th, 1904.—Nature of parasite.—Rogers mixed the blood of kala-azar patient with sodium citras to prevent

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coagulation, and kept the mixture at 22 degrees C.; he observed a multiplication of the parasite and their conversion into a flagellate, like a trypanosome without an undulating membrane.

Appearance of the parasite.—Very small, so do not confuse with blood platelets (slightly greenish tinge and more refactile than platelets).

Stain with Leishman's stain.—Round, oval, or oat-shaped bodies, 2 to 3 microns in diameter, with two chromatin masses, a large nucleus and a small centrosome compare with the trypanosome. These two chromatin masses are usually situated opposite to one another in the long axis of the parasite. The small mass is rod or dot shaped, and usually placed laterally in the protoplasm of the parasite (which is displaced by the vacuole which takes up most of the body substance). The protoplasm stains pink or blue, and sometimes see a tail of protoplasm between these two chromatin masses.

British Medical Journal, January 23rd, 1904.—The Zooglea mass of Manson is really fragments of large macrophages. Division forms.—Cases observed where division taken place in the large nuclear mass before it does in the smaller, sometimes get as many as six division forms, the large nuclei arranged peripherally and the small centrally. Distribution in the body.—(1) In peripheral blood, very rare. (2) Splenic blood, many, especially if some of the splenic pulp is aspirated at the same time. (3) In liver puncture, blood. (4) In the portal vein, *post mortem*. Distribution of parasites in the tissues.—Immense numbers in spleen, liver, and bone marrow; to a less extent in the lungs and testes, supra-renals, and lymphatics. In skin and intestinal ulcers, parasites always intra-cellular in—(*a*) Endothelial cells; (*b*) macrophages. Mode of entry (?) through the gut. Make smear preparations, or, better, thin sections. Stain with Leishman's stain or with hæmatin.

A FEW BRIEF NOTES ON THE CHANGE OF GENERATION AND HOST IN TRYPANOSOME AND SPIROCHÆTA (SCHAUDINN).

Schaudinn has done some most interesting as well as remarkable work on the above subject. His experiments were conducted on the (*athene noctuæ*) small owl, near Rovigino (Berlin), and mosquito (*culex pipens*). I will just give an abstract, and those who want to go more fully into this subject I refer to his original work, "*Arbeiten aus dem Kaisterlichen Gesundheitsamte Band XX., Heft* 3, 1904." The developing trypanosomes undergo their fertilisation in the stomach of the mosquito (*culex pipens*), where an ookinet is formed by the fertilisation of the macrogamete by the microgamete. Ookinets develop in three ways—(1) As indecisive hermaphrodite forms; (2) as female forms; (3) or as male forms. The hermaphrodite forms may, under certain conditions, become either males or females. These three forms may multiply asexually, the hermaphrodite forms by dividing into two. Through parthenogenesis, the females can reproduce the three series of forms; but, in the case of the males, their powers of development are very limited: if they do not copulate with the females they perish.

I shall reproduce Schaudinn's pictures, as by their aid at once is the development clearly understood, and I shall let the pictures speak for themselves, as no description I could give—or even giving Schaudinn's text complete—would make it so clear.

Fig. I.—*a* to *h* shows schematically the transformation of an indifferent hermaphrodite ookinet into the trypanosome stage, by a process of heteropolar mitosis. In (*a*) the complete synkaryon lies in the centre; there are also to be seen eight chromosones surrounding the darkly-stained karyosom. In the broad end are seen pigment and four darkly-stained bodies, the residue

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of the process of reduction. In (*b*) the ookinet is seen to be getting rid of the residual body and pigment. In (*c*) the nucleus undergoes division, and a diaster is seen, and so two nuclei are formed. In (*d*) the left daughter nucleus is seen to be reconstructing; the eight chromosomes are now twisted filaments. Each nucleus contains a central granule, which is connected by a fine achromatic filament with the sister nucleus. The smaller nucleus becomes the blepharoplast of the trypanosome. In (*e*) the smaller nucleus re-divides, the three nuclei being connected by a filament. In (*f*) the smallest of the three nuclei at once forms a spindle, and this spindle becomes transformed into the locomotor apparatus of the trypanosome, the undulating membrane developing at the thickened border, the central spindle being excentrically situated. The eight mantle fibres (corresponding to the chromosomes) become eight myonema, four of each of which run along either side of the flattened anterior part of the trypanosome in the ectoplasm, and, by uniting with the central spindle at the anterior end, produce the conical flagellum. The locomotor apparatus (myonema and undulating membrane) is therefore a product of the nucleus. The development of the ookinets into the female form. The female ookinet has its plasma filled with dark staining bodies, *i.e.*, reserve stuffs. The formation of the heteropole spindle takes place in the same manner as the indifferent forms.

Fig. II.—In (*a*) see the division of the nucleus, and the smaller nucleus leaves the larger, and, quickly dividing and re-dividing, forms eight small compact nuclei; these move at once into the posterior section of the cellular body. (*b*) Each of these nuclei now produces a smaller nucleus, which remains connected with it. (*c*) Now the eight groups of nuclei gradually perish, and the large nucleus develops two smaller ones by heteropolar mitosis (*d*). This becomes the blepharoplast by forming the flagellate apparatus, as in the case of the indifferent one. All the locomotor apparatus is less developed then in the indifferent type, hence the movements of the female are sluggish, and during their growth they keep on producing more reserve stuff, thus allowing them to resist external influences, and so, under adverse circumstances, remaining the longest alive. They remain alive in the ovaries of the mosquito during winter, and develop in the eggs deposited in spring time. Another remarkable fact is through parthenogenesis the female can go back to the gregarine (ookinet) stage should the males and hermaphrodite forms perish, and from this ookinet form both male and female forms are developed.

Fig. III.—The origin of the male trypanosome from the ookinets. No reserve stuffs in the ookinets, and their plasma is more hyaline and coarsely vacuolar. As in the case of the female ookinet, a heteropolar spindle is produced, and the smaller nucleus multiplies into eight. Now in the female type these eight nuclei died, and the large nucleus divided into two: in the male type the large nucleus dies, and the eight nuclei re-divide, each forming a blepharoplast, or micro-nucleus, or centrosome (*a* to *d*). The ookinet now becomes globular, and the eight nuclei move to the periphery, the blepharoplasts being directed towards the upper surface (*e*). The blepharoplast of each group then forms its flagellate apparatus through fission of the nucleus, and eight small trypanosomes are set free (*f*). The males have a very well developed flagellate apparatus. The trypanosomes agglomerate by their posterior, and sometimes by their anterior, extremities into rosettes. All forms of the parasite affect the red blood corpuscles, growing at the expense of the hæmoglobin.

Fig. IV.—(*a* to *g*)—showing the indifferent trypanosomes in the blood of the owl. (*a*) Attachment of the youngest stages to the red blood corpuscle by the anterior extremity. (*b*) Stage of rest. (*c*) Forty-eight hours after attachment. (*d*) Migration in the gregarine-like condition; development

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of the flagella-like apparatus. (*e*) The free motile trypanosome attaching itself to a red blood corpuscle. (*f*) Condition of rest five days after the first attachment. (*g*) Fully developed trypanosome.

Fig. V.—Schematic growth of an indifferent ookinet from Spirillum Liemanni, and the formation of the indifferent trypanosomes therefrom.

Fig. VI.—Various stages of an indifferent Spirilla, from the body of a mosquito. (*a*) A single parasite greatly enlarged. (*b*) Stage of fission. (*c*) Two parasites which have remained attached by their posterior extremities after division (the actual spirillum stage). (*d*) Rosette, agglomeration by posterior extremities. (*e*) Much smaller agglomeration. (Schaudinn thinks, owing to this decrease in size of the spirilla by multiplication, that spirilla may be the exciters of yellow fever.) Supporting this close relationship of Schaudinn's between spirochætæ and trypanosomes, Theiler has found both in red water and in Rhodesian coast disease. Another case:—G. F. Petrie discovered spirochætes in the blood of a martin, and trypanosomes in its bone marrow. Schaudinn thinks that the trypanosomes, when very small, could pass through a Berkefeld filter, because it is only when they are agglomerated that they can be seen. A spirillum is a bacterium made up of a large number of conjoined vibrios, each helping to make up a spiral. The spirillum moves with a flagella. A spirochætæ is a single long thread, one cell, with pointed extremities, movement due to the contractility of its protoplasm. Since the above paper was delivered Schaudinn, working further, has shown that the probable cause of syphilis is spirochæta pallida.

In conclusion, I wish to say that I have reduced very largely the volume of my original paper on account of space for the printer, so that it reads more as an abstract: and all medical journals bearing on the subject have been freely consulted, as well as numerous authorities, the chief being—Laveran and Mesnil, Stephens and Christophers, Manson, Daniels, Smedley, Liemann, Stiles, Musgrove, &c.

The following specimens were shown:—*Macroscopically*—Lingulatula rhinaria, male and female. Pentastomum denticulatum. Tænia echinococcus infection of the intestine of a dog (artificial). Tænia medio-canellata, five worms from a patient *post-mortemed* at the Adelaide Hospital, collectively 105 feet long. Simple cyst of a liver. Multiple cysts of a liver. Multiple tubercular strictures of the small intestine. Multiple subumcosal herniæ in small and large intestine. Trichina spiralis. *Microscopically*—Trypanosome Duttoni (Sir Patrick Manson). Tr. Evansi. Tr. Brucei (Dr. Low). Tr. Lewisi. Tr. Danielwyski. Schistosomum Cattoi (Dr. Catto). Leishman Donovan bodies (Dr. Low). Tænia echinococcus. Taæni echinococcus alveolaris. Bilharzia specimens, &c. Guinea worm embryoes. Filaria (five varieties). Filaria nocturna from a case in my out-patients. Trichina spiralis. Trichina spiralis, intestinal form, from Professor Virchow's original case, &c.

AN UNUSUAL FORM OF FIBRO-CELLULAR GROWTH.

By Dr. J. Thomson.

Ramah Ram, *æt.* 24, single, Punjaubee, was admitted to one of my beds in the Brisbane Hospital in June, 1905. He had a very large growth covering the top and right side—practically half—of his head, and it overhung his right eye, obstructing its vision. It was for this interference with sight he

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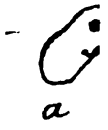

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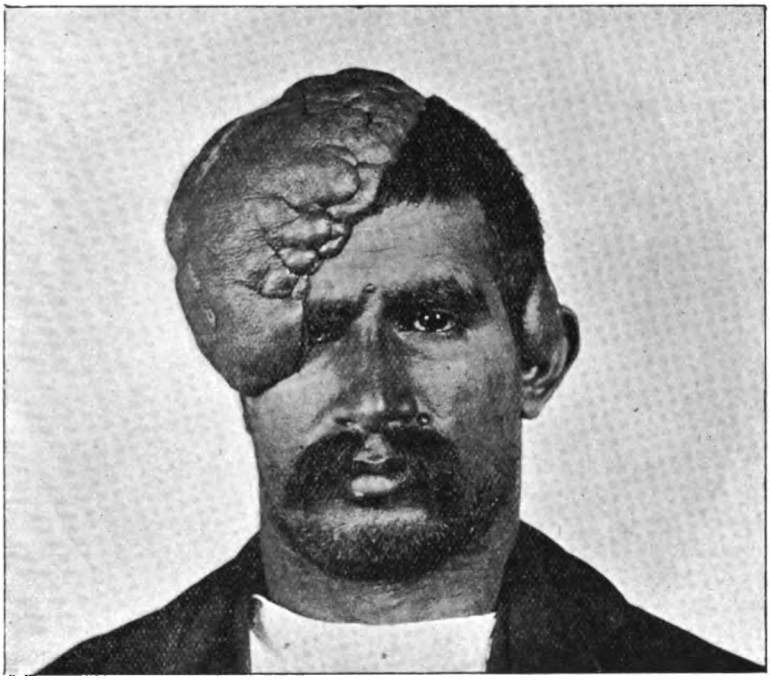
sought relief. There was another growth, about the size of a walnut, over his left ear, and there were numerous small growths on his face, trunk, and limbs—in fact, all over him.

The large growth is dark—a blue-black—smooth, elastic, india-rubbery, freely lobulated, resembling the convolutions of the brain; devoid of hair, and, to the touch, seems warmer than the rest of the body. There is nothing tender or painful about it; and, beyond the visual disturbance, it gives rise to no inconvenience, for—after the manner of his countrymen—he wears a turban, not a hat.

He states that up to the age of 12 there was no growth, only the skin was black, smooth, and hairless. Growth then started and has continued, slowly increasing, since.



In every other respect the patient's condition is normal, and although the blood was examined on several occasions—early in the days and also late at nights—no filariæ were discovered. There have never been any febrile attack nor any conditions suggesting filariasis.

To relieve the right eye, a wedge-shaped piece was removed from the overhanging mass. This piece was about three inches long, corresponding to the line of the eyebrow, and the wedge base about one and a quarter inches broad. On section, the growth was found to be very tough and leathery; in itself it was but sparingly vascular, yet, when cut, the few vessels in it pumped very briskly, for there was no retraction; deeper down, beneath the growth proper, vessels were in plenty, and there was very free bleeding.

There was primary union, and the patient left the hospital, rejoiced that his right eye was permitted to see.

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In about seven weeks he returned; the eye again covered. This was not due to any increase of growth, but to the mass being dragged, by its own weight, over the eye and on to the cheek. A second operation was performed. A larger wedge removed, similar conditions of vascularity met with, and healing by first intention obtained.

Pathological Notes.

Skin.—Stratum corneum, well marked, not desquamative. Rete Malpighii considerably narrower than usual, and not nearly so much broken up in outline by the papillæ of the dermis as is normal. Fibrous tissue more condensed at the surface, and becoming more swollen and œdematous towards the base. No sebaceous, or hair follicles, are visible. Rudimentary, or

atrophied, sweat-glands are seen. Cellular masses lie scattered through the more superficial layers of the fibrous tissue. Pigment exists in the lower layers of rete and in some of the deeper cellular masses. In the deeper layers the cells mostly disappear and the fibro-elastic tissue becomes more swollen. Some pigment is also seen.

Microscopically the above description corresponds with the structure of fibroma molluscum, though different clinically. The structure does not appear to be rich in blood or lymph vessels, though the succulent nature of the deeper fibres suggests Elephantiasis, which is, however, not borne out by the presence of isolated nodules on body.

Conclusion.—A fibro-cellular growth, allied to Fibroma Molluscum in structure, or possibly Elephantiasis of the scalp; the absence of filaria in the blood would tend to negative the latter view.

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The only case that I know at all similar to this is the one reported and figured by Dr. Allardice Macdonald in *The Journal of Tropical Medicine*, April 1st, 1905, and described as "Elephantiasis of the scalp." Pictorially the two cases appear identical, and very probably they are in most other respects. Macdonald's patient suffered from the periodic febrile and other disarrangements peculiar to filariasis, and the nematodes existed in his blood in great numbers.

It is not actually so stated, but it is inferred that the tumor—the elephantiasis—of the scalp had a filarial origin.

In Macdonald's patient the growth is solitary; in mine, there are many growths—they are multiple.

My patient has no filariæ, nor has he, at any time, had any of the constitutional disturbances created by their presence; therefore there is no reason to suspect that his growths are due to blood worms.

For the clinical notes I am indebted to Dr. T. G. Ross, and for the pathological to Dr. Wilton Love.

A NOTE ON THE FREQUENCY OF STREPTOTHRIX INFECTION IN MAN.

By Professor D. A. Welsh, M.A., B.Sc., M.D., and J. E. V. Barling, M.B.

Within the last eighteen months four specimens of pus have been brought to the Pathological Department of the Sydney University, in which the presence of a streptothrix has been demonstrated. In addition, there have been one or two cases at least during the same period in which, though the clinical features clearly pointed to actinomycosis, yet the organism was not found in the pus; probably owing to the fact that in the earlier cases Gram's method of staining was not used. All the cases occurred in Dr. MacCormick's practice, the specimens having been obtained through him.

Three types of the disease were represented in the four cases, in which the presence of a streptothrix was definitely ascertained. In the first place, in two of the cases the infection had evidently taken place through the mouth, the resulting lesions taking the form of hard, brawny, inflammatory swellings. In one of these cases the swelling was relatively small, being situated below the left malar bone, and involved the tissues of the cheek, the bone apparently escaping: the other case was more extensive, the swelling reaching from the upper part of the temporal region down to the middle of the neck on the right side. Scattered throughout the mass were small shallow abscesses. Subsequently, at the operation, portion of the lower jaw was found to be involved in the inflammatory process. In both these cases the pus contained the characteristic sulphur grains.

Secondly.—In one case the infection was a pulmonary one. The trouble started with an attack of pleurisy over the left base, followed later by the development of a cough and the expectoration of a good deal of sputum, with small amounts of blood. Later, several small subcutaneous swellings appeared over the left base, which, on being opened, discharged a thick yellow pus, which contained sulphur grains.

Thirdly.—In one case the infection was an abdominal one. The process started as an inflammatory attack in the region of the appendix, associated with the appearance of a mass along the course of Poupart's ligament, on the right side. At the operation the appendix was found buried in a mass of recent inflammatory material, with a perforation about its centre. There

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was no pus present. Later an inflammatory mass developed in Douglas' pouch, which, however, disappeared in the course of three weeks without suppurating. Subsequently a small pocket of pus pointed in the left nipple line, just below the costal margin, which was found to communicate with a large abscess cavity behind the spleen. Later an abscess developed between the diaphragm and upper surface of the liver, on the right side. It was in films of the pus taken from this situation that the organism was first detected, though no characteristic grains were present. During the course of the illness the appendiceal wound gave occasional issue to small amounts of a thick creamy pus, in which were present typical sulphur grains and in film preparations of which the presence of a streptothrix could be readily demonstrated. Just prior to the fatal termination of the disease the subcutaneous tissues of the abdominal wall above the umbilicus became thickened and infiltrated with inflammatory products.

As to the staining reactions and appearance of the organisms in film preparations:—As regards their staining the most noticeable feature was the difficulty experienced in getting them to take up the ordinary dyes—such as methylene blue, thiouin blue, or weak ziehl. It was owing to this fact that the organisms were overlooked on one or two occasions. They, however, stain well by Gram's method, and all suspected cases should therefore be treated in this way. In specimens stained thus the branching mycelial filaments present a characteristic appearance. The branches are usually short, and disposed laterally along the course of a filament. In some cases the ends of the branches were bulbous. In none of the cases was the "ray fungus," or actinomycotic arrangement, observed in the colonies, branching mycelial filaments being the only elements noticed.

The cultivation of the organisms in artificial media presents some difficulty. In three out of the four cases, however, pure cultures have been obtained, though their full cultural characteristics have not yet been worked out. The following points may, however, be noted:—

First.—Their comparatively slow growth in the ordinary culture media.

Secondly.—As pointed out by Fullerton and Jones, they grow best in media which contain maltose.

Thirdly.—As also noted by Fullerton and Jones, when cultivated in maltose broth they produce granular masses of growth, which, settling down to the bottom of the test tube, produce no turbidity in the medium.

Fourthly.—There are indications that they have a leaning towards anærobic conditions.

As regards the mode of infection, in two of the four cases there was a history of the patients being in the habit of chewing either wheat grains or the straw of wheat previous to the onset of the symptoms.

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SECTION OF PUBLIC HEALTH.

SOME LESSONS FROM THE STATISTICS OF INFANTILE MORTALITY IN SYDNEY.

By W. G. Armstrong, M.B., Ch.M., Sydney; D.P.H., Cambridge.

Gentlemen—Before beginning my address I wish to gratefully acknowledge the distinguished honor which has been conferred upon me of election to the Presidency of this Section—an honor which, I am well aware, is due to the position I occupy in the Public Health Service of the State of New South Wales, and not to any personal merits of mine.

The subject I have chosen for my address is a departure from the custom often observed in presidential addresses, in that it deals with a special subject rather than with a general review of progress in the work and knowledge peculiar to this Section. I trust you will pardon the innovation.

It will not be out of place to begin this paper by drawing a comparison between the infant mortality of England and Wales and that of New South Wales. Table I. gives the materials for such a comparison. This table gives the mean of ten years' statistics, and has been extracted from the annual reports of the English Registrar-General and the New South Wales Government Statistician. Corresponding figures for the city and suburbs of Sydney have been introduced into the table, showing the effects of urban conditions upon infantile rates. The rates in the table, as throughout this paper, are calculated on the number of deaths under one year of age per 1,000 births, which is the most accurate method of stating such deaths.

There is a great contrast between the total infantile mortality of England and that of New South Wales. The former exceeds the latter by 44 per cent. and is 25 per cent. greater than the infant mortality of Sydney. This difference is one of relatively recent growth, as in former years the infantile mortality of New South Wales exceeded that of the mother-country. With so great a difference in the totals, it is not surprising that most of the component rates should be in favor of New South Wales. As a matter of fact, under the thirteen different headings which compose the totals in the table, there are only two under which the English infantile death rates do not exceed those of New South Wales. The two in question are diarrhœal diseases, with an English mortality of 29.1 per 1,000 births, as against 30.7 per 1,000 births in New South Wales; and congenital defects, in which the rate (3.9 per 1,000 births) is exactly the same in both England and New South Wales. Very striking differences are observed in the mortality from pneumonia and bronchitis, which is nearly 150 per cent. greater in England than in New South Wales, and in those from tubercular diseases and convulsions, both of which are in England more than double the New South Wales rates.

That the death rate from respiratory affections should be greater in a country with the climatic conditions prevalent in the British Isles than in New South Wales is not astonishing, but the great excess in deaths from convulsions is more difficult to account for, and, it appears to me, must be due

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to divergences in the practice of certification or in classification. The relative superiority of the position of New South Wales as regards infantile deaths from tubercular diseases cannot, I think, be dissociated from a lesser prevalence of tuberculosis among Australian dairy cattle. But probably the most important factor is that the standard of living among the poorer classes in Australia is higher than among the corresponding population of Britain. Among the causes predisposing to tubercular disease a food supply insufficient in quantity, or defective in quality, ranks high. It is to be noted that thirty years ago the infantile death rate from tubercular diseases was higher in New South Wales than in England—a statement which is equally true of the general infantile mortality. During the past thirty years the English infantile mortality has actually shown a tendency to increase, while that of New South Wales has, in the same period, declined considerably. Table II. shows that in Sydney and suburbs the fall has been continuous and fairly regular since the year 1886, when it stood at 173 per 1,000 births, until 1904, when it reached the minimum of 98 per 1,000 births.

The influences which bring about infantile mortality may be classified generally as (1) hereditary, (2) intra-uterine, and (3) post-natal. To what extent each of these groups of causes is severally responsible for the infantile mortality of cities is extremely difficult to determine. As regards the first and second groups, very little effective control can be hoped for in the present state of our knowledge; and it is noteworthy that deaths classified under two headings which fall within one or other of these groups, namely, prematurity and congenital defects, have latterly shown a slight but undoubted tendency towards increase in Sydney (Table II.).

The most important, numerically, of the causes of death among children under twelve months of age in New South Wales, as in Great Britain, is diarrhœa. Diarrhœal diseases, which include epidemic or summer diarrhœa and enteritis, as well as the more sporadic forms of fatal diarrhœa which occur from week to week throughout the whole of the year, are annually responsible in England for 29.1 deaths in every 1,000 births, and in New South Wales for 30.7 deaths; while in Sydney the number of children who succumb during their first year to this cause is not less than 40.9 out of every 1,000 births.

Apart from rigid mathematical or experimental proof, there are few facts better established than the intimate connection between infantile diarrhœa and improper feeding. Ernest Holt, in his "Diseases of Infancy and Childhood," emphatically expresses the dictum, "This disease prevails to the extent to which other food than breast-milk is given to infants"; and the weight of all authority, and of common experience, points unmistakably in the same direction. But the influence of the food factor is not only shown in the deaths from infantile diarrhœa.

The cause of infantile deaths which is, numerically, of second importance to "diarrhœa" is that which is classed by the statistician under the heading of "atrophy, debility, and inanition," all of which expressions are of course synonymous for the same pathological condition—or, rather, for the same clinical group of symptoms. Looked upon as a fatal disease, they were responsible for 17.6 infantile deaths in every 1,000 births in Sydney during the past ten years.

Atrophy, debility, and inanition are all very loose terms, and but little superior in accuracy to "dropsy," since they ignore the primary cause of the physical condition which they indicate. In so far as they convey any information, it is that of inability to assimilate food, a pathological condition which frequently—probably in the majority of cases—is a sequel to an attack

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of acute diarrhœa. Even in those cases in which the condition has not been ushered in by an attack of acute diarrhœa, clinical evidence is strong on artificial feeding as a cause.

Let me quote Holt again. Speaking of marasmus—another synonym for infantile atrophy—he says, " In the vast majority of cases it depends upon two factors—the food and the surroundings . . . . . The following is the story most frequently told at the hospitals. ' At birth the baby was plump and well-nourished, and continued to thrive for a month or six weeks, while the mother was nursing it; at the end of that period circumstances made weaning necessary. From that time the child ceased to thrive. It began to lose weight and strength, first slowly, then rapidly.' "

Eustace Smith sets forth the four principal causes of infantile atrophy thus :—(1) unsuitable food, (2) chronic vomiting—gastric catarrh, (3) chronic diarrhœa—intestinal catarrh, (4) constitutional diseases. The same opinions are expressed by every author of repute.

Looked upon as causes of death, infantile convulsions and teething may also be regarded as due to artificial feeding. The latter—which, I am glad to point out, is rapidly disappearing from Sydney statistics as a cause of death—ought certainly never to be so assigned in any certificate. That it still is given occasionally on the certificate is a satirical reflection on the scientific medicine of the present day.

The diagram which I have prepared from the statistics published by the New South Wales Registrar-General and Statistician represents the mean monthly deaths from diarrhœal diseases (diarrhœa, cholera, dysentery, and enteritis), atrophy, convulsions, and dentition at all ages for the past thirty years in Sydney and suburbs. Each vertical square on the diagram represents a rise or fall of 10 per cent. above or below the mean. It should be premised that the curves ought to have been constructed on weekly figures, and for infantile deaths only; but the data necessary for these are, I regret, unavailable. The curves are interesting. In the first place, they all closely resemble one another in their salient outlines. Practically, at their minimum level during the months of June, July, August, and September, they begin to rise in October. Diagram number 2, which is compiled for three years on the only weekly data which are available, shows that the rise in diarrhœa does not begin until after the middle of October, and continues to rise during November and December. The diarrhœa and atrophy curves attain their apex in this latter month, and show a beginning of a fall in January; but the curves of dentition and convulsions are at their highest in January. The curve of convulsions shows another departure from the common type in a slight fall in December, which is an intensification of a tendency in the other three curves to fall away slightly in December, after their abrupt November rise. From January to February there is a marked fall in all the curves, and thereafter a more gradual decline to the minimum. Another point of difference between some of the curves is the lesser amplitude of departure from the mean observed in the curves of atrophy and convulsions, when compared with those of diarrhœa and dentition. But the close general similarity of the curves thus demonstrated is strong presumption in favor of identity of etiology of the diseases classed under these several heads, and clinical observation further supports this presumption, so that I think I am justified without further laboring of the subject in treating all these assigned causes of infantile deaths as essentially one. Let us now examine Table II., and see how this is borne out by the vital statistics of the infantile population of Sydney during the past thirty years. In the table causes of death which are intimately connected with the feeding of children have been placed in juxtaposition. Diarrhœa, as a cause of death,

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actually shows an increasing mortality during the past thirty years; whereas the headings of convulsions, dentition, and the various synonyms for atrophy, all show a strongly diminishing tendency. Convulsions, as an assigned cause of death, has decreased from 18.6 deaths per 1,000 births in 1875 to 3.2 in 1904, or 83 per cent; dentition from 8.4 to 0.7, or 92 per cent; and atrophy, debility, and inanition from 43.6 to 11.4, or 74 per cent. These enormous falls would be quite incomprehensible except on the supposition that increased accuracy of diagnosis and certification has gradually transferred most of the deaths, which would formerly have been loosely attributed to them, to some other heading; and a brief examination of the table leads to the conclusion that the only headings to which such deaths could have been transferred are those of diarrhœa or enteritis. And this supposition probably exactly represents the actual facts, except that the sudden diminution in deaths from dentition between the years 1892 and 1894 suggests the introduction of a modification in the system of classification adopted by the Registrar.

We shall therefore get a clearer mental picture of the statistics of infantile mortality due to improper food as exemplified in Sydney if we combine the mortality from the several causes—diarrhœa, convulsions, dentition, and atrophy. This I have done in the last column of Table II., and the result is a column of figures which represents a mortality having a slight tendency to increase between the years 1875 and 1886. After the latter year the mortality from the combined causes began to fall, and has continued to diminish in a tolerably even curve until the present time: we shall inquire into the probable causes of these phenomena presently. It is advisable at this point to clear the ground by expressing the opinion that a review of the causes of infantile mortality, having any useful end in view, must practically resolve itself into an investigation into the circumstances connected with the mortality from diarrhœa and those other causes of death which depend upon the defective food supply of infants. Prematurity and congenital defects, as causes of death, we can hardly at present hope to modify. Tubercular diseases as a factor in infantile mortality have gradually, during recent years, lost most of their significance. Respiratory diseases, as represented by pneumonia and bronchitis, evidently depend almost entirely upon seasonal influences; and though the mortality from these diseases fluctuates widely from year to year, the oscillations depend chiefly upon the character of the seasons, and the means of a series of years show little movement either of advance or of retardation. Zymotic diseases, other than diarrhœa, constitute the other important heading. The infantile deaths under this column are due for the most part to measles and whooping cough. There has been a marked fall in infantile deaths under this heading, as shown in the table, and the present rate in Sydney is between 5 and 6 per 1,000 births.

The scheduled causes of infantile deaths which depend upon improper food, or dietary diseases, as they may be called, account for about one-half of the total infantile mortality. They have, therefore, a far more important influence in determining the amount of infantile mortality than any other group, or combination of groups, of diseases. The facts that they are admittedly dependent upon improper feeding, and that their fatality has largely diminished and is still on the down grade, argue that, as causes of death, they are controllable. Can they not be more thoroughly controlled?

Dr. Hope, the Medical Officer of Health of Liverpool (England), recently remarked that in cities an infantile mortality of 100 deaths per 1,000 births might be regarded as an ideal minimum of inevitable deaths during the first year of life. This dictum, of course, applied to England, and to English conditions. In Australia (the statement is based upon Sydney figures, but I have

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no doubt they are, on the whole, applicable to the other large towns of Australia), where the infantile deaths from tubercular, respiratory, and zymotic diseases are very much less than the English experience, the figure for infantile deaths which must be regarded as unavoidable should be considerably lower than the standard set up by Dr. Hope.

It is not my intention, in this short address, to enter at all deeply into the question of the influence of meteorological conditions upon the mortality from dietary diseases. Ballard, in his classical report to the English Local Government Board, in 1888, went very fully into this subject, and his conclusions are well known. His "working hypothesis" connected the essential cause of diarrhœa with the life process of some unknown micro-organism resident in the more superficial layers of the soil, the vital manifestations of which are dependent, among other things—perhaps, principally—upon conditions of season, and on the presence of dead organic matter, and which finds, especially at certain seasons, nidus and pabulum in human food where it manufactures a substance which is a virulent chemical poison. This "working hypothesis" up till the present time sums up the knowledge we possess as to the essential cause of diarrhœa. We have made but little advance in this direction.

Other propositions of Ballard's are certainly not applicable to Sydney conditions, however true they may be in England. His conclusions on the relation between the summer increase of diarrhœal mortality, and the temperature registered by a 4-foot earth thermometer, do not hold good here. There are, it is true, no records of the temperatures registered by the 4-foot earth thermometer in Sydney, but there are complete records of the temperature of the earth 5 feet below the surface for the past thirty years, and these differ so slightly from the 4-foot observations that they may be regarded as practically identical. Ballard's observations referred to are as follows:—

(*a*) The summer rise of diarrhœal mortality does not commence until the mean temperature recorded by the 4-foot earth thermometer has attained somewhere about 56° F., no matter what may have been the temperature previously attained by the atmosphere:

(*b*) The maximum diarrhœal mortality of the year is usually attained in the week in which the temperature recorded by the 4-foot thermometer attains its mean weekly maximum:

(*c*) The decline of the diarrhœal mortality . . . . . coincides with the decline of the temperature recorded by the 4-foot earth thermometer, which temperature declines much more slowly than the atmospheric temperature.

In Sydney a comparison of the records of 5-foot earth temperatures for thirty years with the weekly records of diarrhœal mortality shows (1) that in many years the 5-foot earth thermometer does not register below 56° F. at any time, consequently that temperature cannot be regarded as a critical one; (2) that the maximum diarrhœal mortality of the summer in Sydney is reached in December, whereas the mean weekly maximum of the 5-foot earth thermometer is generally not reached till March, and never before the end of January; (3) that the decline of the summer diarrhœal mortality in Sydney does not begin till March. All these points are shown in the attached diagram, which exhibits the rise and fall in the diarrhœal mortality in Sydney and suburbs, and the variations in the meteorological data, from week to week during the years 1902-1905.

While, however, this is the case as regards the earth temperature, the relation of shade temperature to diarrhœal appears to merit investigation.

The summer rise in diarrhœal mortality in Sydney usually begins about the last week in October or the first week in November, about a fortnight

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after the mean temperature in the shade permanently attains a height of 60° F., and the diarrhœa rise continues to about the middle of December, when the apex is attained, and the descending curve begins, falling before the apex of the mean temperature curve is reached (during January and February). Having once begun, the fall in diarrhœal mortality continues evenly, apparently without reference to the temperature curve, until the minimum of the mortality curve is reached in September. Thereafter a slight rise occurs in October, but the real rise is deferred till November.

If one allows for the average duration of illness in fatal cases of diarrhœa, it will appear that the beginning of the summer epidemic manifestation of diarrhœa in Sydney is very nearly coincident with the time at which the mean temperature in the shade touches 60° F. Out of 279 cases of fatal diarrhœa in infants in the city of Sydney, which I investigated during the years 1902-1905, the average duration of illness was found to be twenty-eight days, and the duration of illness in individual cases was as follows :—

During the	first	week of illness	107	died, or	36.2	per cent.	of all cases
"	second	"	58	"	20.8	"	"
"	third	"	35	"	12.5	"	"
"	fourth	"	19	"	6.8	"	"
"	fifth	"	5	"	1.8	"	"
"	sixth	"	11	"	3.9	"	"
"	seventh	"	4	"	1.4	"	"
"	eighth	"	16	"	5.7	"	"
After the	eighth	"	30	"	10.7	"	"

Allowing for two days' delay in registration of the deaths, which appears to be about the average, and a very short incubation period, it would seem from the above figures that about a week after the critical mean temperature in the shade, if there be one, has been recorded, a response should appear in the rise of the curve in the registered mortality from diarrhœa, and this is seen to be the case after a mean temperature in the shade of 60° F. has been reached.

When the epidemic mortality has once received a start, it gathers force rapidly throughout a period from three to four weeks, after which it begins to decline without further relation to the shade temperature curve. A fairly good simile for the phenomenon is the pressing of the trigger of a gun, and the outburst diarrhœal mortality is the explosion which follows. The best expression of the facts which I have come across in medical literature is one by Ernest Holt, who says, "It appears that an average mean temperature . . . of about 60° F. is required to start the epidemic. Not many cases are seen until such a temperature has lasted for some days. The explanation of the high mortality appears to be . . . . that the majority of the susceptible infants are unable to withstand the first very hot month. Humidity and rainfall, according to the most careful investigations of Siebert in New York and Baginski in Berlin, do not influence either the prevalence of summer diarrhœa or its mortality."

The last statement of Holt's is striking, in view of the contrary opinions held by most English authorities, notably Hope, of Liverpool, and Newsholme, of Brighton, on the influence of rainfall upon summer diarrhœa. Personally I have always held the opinion that copiousness of rainfall during the hot summer months bears a very close inverse relationship to the prevalence of diarrhœa and cognate disorders; and, without being able to produce any convincing statistical evidence to back this opinion, I must confess to retaining it still. But an investigation into the records of meteorological data in Sydney for twenty-eight years, and their coincidence with fluctuations in the summer mortality from diarrhœa is disappointing. All one can definitely gather

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from them is that, on the whole, diarrhœal mortality has been higher in dry and hot summers than in cool and wet ones, and *vice versa*. It appears to be very difficult, if not impracticable, to separate the influences of drought and heat in this connection. Even when they are combined, and both excessive, there are exceptions to the general proposition that under such circumstances the mortality from diarrhœa is coincidently high.

For instance, the summer of 1904-5, with the highest mean temperature in the shade (with one exception, that of 1895-6), and the lowest rainfall (without exception) for twenty-eight years, also witnessed the lowest diarrhœal mortality experienced in Sydney for twenty-eight years. But whatever may be the effect of meteorological conditions upon the incidence and fatality of diarrhœa and its cognate causes of mortality, there must be some other cause or causes at work of almost equal or even greater importance. If not, there could be no progressive change in the mortality from this group of diseases such as has been experienced in Sydney during the past thirty years; and this is a most comforting reflection, otherwise we should have a very hopeless task set us in the reduction of infantile mortality from these causes, since we have no control over meteorological conditions.

I have already drawn attention to the course of movement shown by the Sydney mortality curve from diarrhœa and its kindred diseases, and indicated that the level of the curve showed no particular tendency towards movement in either direction until after the year 1886, when a downward movement set in which has been progressive until the present time. In the eleven years prior to 1886 the infantile mortality rate from those diseases which I have discussed fluctuated between 84 and 111 per 1,000 births, with a slightly higher tendency towards the close of the period in 1886 than at its beginning, in 1875. Since 1886 the rate has fallen, till in 1904 it stood at 46 per 1,000 births. What have been the causes at work in Sydney which have so reduced the rate ?

Infantile diarrhœa is, no doubt, directly due to food infection. This much is proved by the relative immunity from the disease of breast-fed children. In a series of sixty infants under three months of age who died from diarrhœa in the city of Sydney during 1902-5, and whose cases I investigated, I found that only 6.6 per cent. had been entirely breast-fed, while 93.4 per cent. has been wholly or partially fed on other foods. During the year 1904 I made an inquiry into the methods of feeding of 621 living infants under the age of three months. The results showed that 72 per cent. were entirely breast-fed, and the remaining 28 per cent. were wholly or partly fed on other foods. If these sets of figures are comparable—as I have no doubt, in the main, they are, since my inquiry into the feeding of living infants was conducted in the districts and among the classes of population which chiefly furnish the diarrhœal mortality—the mortality among infants under three months old fed wholly or partially otherwise than on the breast is, in Sydney, thirty-seven times as great as among infants of the same age wholly breast-fed. This startling discrepancy is only to be accounted for on the theory that, by some means, the essential cause of diarrhœa reaches the infant through its food. Food, however, is only to be regarded as the vehicle of the infection. Ballard regarded the upper layers of soil as the *nidus* of the infective material, and a large mass of evidence which has been accumulated since his classical report was published has done nothing to weaken his hypothesis. Perhaps the weight of evidence is rather in favor of the surface of the soil instead of its upper layers being regarded as the *habitat* of the diarrhœa organism or organisms, and diarrhœa is now rightly considered as a filth disease, and every movement in domestic and municipal sanitation which makes for cleanliness is to be looked upon as one step towards the diminution of this most fatal disorder.

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The sanitary history of Sydney during the past thirty years lends support to this view. Prior to the year 1888 the water supply of Sydney was obtained from the Botany swamps, situated about five miles south of the centre of the city, in a low-lying peaty and sandy morass, on the shore of Botany Bay. The catchment area and conservation beds were liable to pollution from many sources, and the district of Botany itself has been for many years a hot bed of diarrhœa and typhoid fever. Since the year mentioned, the water supply of the city and suburbs has been obtained from a catchment area situated on mountainous sandstone country about fifty miles to the south-west of Sydney —a source which is almost unimpeachable on sanitary grounds.

The change in the system of sewerage in Sydney has been no less important than that in the water supply. In 1877 the system of sewers in the city was about as bad as it was possible to be. The sewers were of various sizes and shapes, constructed in brick and stone, and all discharged into the harbor. The tank stream sewer, running through the centre of the city, and entirely built over by dwellings and business places, was in a foul dilapidated state : the sewage stagnating along an uneven invert, while the sewer air found vent through innumerable unplanned crevices and openings, not to mention the unventilated house drains. The suburbs were entirely unsewered, and cesspits of the foulest description, and the most insanitary construction, were common both in city and suburbs. In that year the sanitary arrangements of the city were considered to be so defective that the Government of the day appointed a board to report on the subject, and the report brought to light many glaring evils. As a result of this report, the present excellent system of sewers began to be constructed in the year 1885, and was steadily pushed forward until the year 1897, when the system serving the city was completed. Even in the year 1890 there was still a considerable number of filthy cesspits in the city ; and in the suburbs, as late as 1899, there were known to be 1,559 of these abominations, and probably as many more existed unaccounted for. Almost all these insanitary conditions have now been removed, and the city and all the more populous suburbs of Sydney are as well off as regards sewerage and water supply probably as any large city in the world.

In the year 1886 the Dairies Supervision Act came into operation, and was enforced year by year with gradually increasing stringency, improving vastly the conditions of the metropolitan dairies, and, as a consequence, raising the quality of the milk supply.

Finally, the advent of plague in 1900 caused the municipalities of the metropolitan district to exercise their powers to make by-laws for the suppression of filth on premises, and to enforce them when made, and generally to put their municipal houses in order with such vigor as was before undreamed of.

It might have been anticipated that all these advances in sanitary conditions would have had a salutary effect on the infantile death rate from diseases caused by food pollution, and the coincidence of sanitary improvement with progressively falling death rates is so striking as to be convincing evidence of the casual relation of the one to the other.

But there is one field of municipal endeavor in the prevention of infantile mortality, the soil of which has been but lightly stirred hitherto, but which, with diligent tilth, should yield a goodly crop. It is that necessary to break the vicious chain of events which carries the infective material from the soil into the stomach of the infant by preventing the pollution of the food supply, *i.e.*, the milk. The Dairies Supervision Act has already proved a step in this direction in Sydney. By enforcing many improvements in the construction of the dairies and milk shops it has lessened greatly the opportunities which

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existed for filth and micro-organisms to find their way into the milk. A vigorous enforcement of the adulteration of food clauses of the Public Health Act has also been of avail. The suppression of the practice of adulterating milk by the addition of water has diminished the chances of micro-organisms finding their way into the indispensable food of young children, and the prohibition of the use of preservatives has compelled the milk vendor to sell fresh milk only, and limited his opportunities of passing a stale and germ-laden fluid on an unsuspecting public.

The question, however, is constantly present in one's mind as to whether more pollution does not enter the infant's milk in the home of the infant himself than at the dairy and in the course of transit to the consumer's dwelling. Newsholme believes that the home is the most common site of infection; and there are many reasons in support of his contention. No one who has been accustomed to visit the homes of the poor in the crowded slums of great cities will need to be reminded of the innumerable chances of pollution of the food supply of the infant, once he has been permitted to receive nourishment otherwise than from his mother's breast. Imperfectly cleansed bottles with long rubber tubes, in which particles of sour milk curd adhere and decompose, setting up rapid changes in every fresh meal that is introduced into them; dirty methods of storage; the access to food of flies and other insects with feet swarming with micro-organisms, after visiting heaven knows what foul garbage; dust from streets and yards, carrying particles of manure, &c.—these are only some of the dangers to which the infant life of the poor neighborhoods of our cities is exposed. To minimise these dangers there are, it seems to me, two lines of procedure open. One is entirely new to us in Australia, but evidences of its value in the saving of the infant life are rapidly accumulating in France, in England, and in the United States · I refer to the establishment of infant milk depots, and model dairies established and conducted by private philanthropy, or, preferably, under municipal control; such, for instance, as that of Battersea in London, or Rochester in the State of New York. In these depots sterilised milk, produced at a model dairy, is modified, according to the requirements of the age of the child for whom it is intended; is put up in bottles of such a size that each contains one meal; and is sold to poor mothers in baskets, each of which contains enough meals for one child for twenty-four hours, at a price which barely pays expenses. At Battersea the charge for supplying an infant in such a manner is 1s. 6d. a week. The children supplied from the depot are registered, and their homes visited from time to time by a woman inspector, who notes the progress of each infant, and reports at the depot. She also instructs the mother in the feeding of the child. Such statistics as are available in connection with these depots, as well as those in France and other continental countries, are of the most gratifying character, and show an immense saving in infant life from their operations. Indirectly also they are of great value in educating the public in the best methods of feeding young children, and the model dairies connected with the depot are valuable as object lessons to the ordinary dairy farmer, whose ways and methods in the production of milk are never ideal, and often disgusting. To all who are interested in this subject, a perusal of Dr. McCleary's valuable little book on infantile mortality can be most heartily recommended. The other precedure to which I allude is purely educational in its method, and has been in operation in the city of Sydney for rather more than a year. A daily list of all births registered in the city is obtained from the registrars, and, within a few days after registration, the home of each child born in a poor neighborhood is visited by a woman inspector of the municipal council, who interviews the mother, talks to her confidentially on the management of the child, advises her as to the methods she ought to follow, and earnestly inculcates cleanliness, leaving,

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also, at each house a copy of a brief and plainly worded leaflet setting forth the dangers of infantile diarrhœa, and giving instructions on the feeding of infants. The principal points impressed on the mother by the inspector are the great superiority of breast-nursing over every other form of feeding: should breast-feeding be impossible, the use of properly modified fresh cow's milk is advocated, and warnings against the long tube bottle and the use of starchy or patent foods are tendered. The conditions as to cleanliness and general sanitary state of the dwelling are noted by the inspector, and reported by her at headquarters, where any action which may appear to be indicated by the information in the report is taken by the Medical Officer of Health.

It is evident that the value of such a proceeding as this must largely depend upon the personal tact of the visiting inspector, and the municipality of Sydney has been fortunate in obtaining the services of a woman who is in all respects exceedingly well fitted for the work. It is worthy of note that in only two or three instances has there been the slightest objection, or resentment, on the part of mothers to these domiciliary visits.

Since this action was inaugurated in May, 1904, over 1,400 homes of newly born children have been visited in this manner. The experiment is yet too recent for me to be able to offer you any useful statistics on its effects; and, moreover, such statistics would be obscured, and their value diminished, by the unusually low infantile mortality which appears to have prevailed throughout eastern Australia during the summer of 1904-5, and which has evidently largely depended on causes beyond human control.

There are many other aspects of this most important and fascinating subject to which I might refer, but I feel that I have already taken up too much of your time and attention. I trust that our experiences in Sydney may be of some slight value to sanitarians in other States, and particularly to the members of this Section.

TABLE I.

DEATHS OF INFANTS UNDER ONE YEAR PER 1,000 BIRTHS REGISTERED.—MEAN OF TEN YEARS' OBSERVATIONS—ENGLAND AND WALES, 1892–1901; NEW SOUTH WALES AND SYDNEY, 1895–1904.

	England and Wales.	New South Wales.	Sydney and Suburbs.
Measles	3.0	.4	.6
Whooping cough	5.7	3.3	3.1
Pneumonia and bronchitis	25.2	10.3	11.2
Diarrhœal diseases	29.1	30.7	40.9
Convulsions	17.8	8.2	6.5
Dentition	2.4	1.1	.7
Suffocation	2.0	.6	.8
Atrophy, debility, and inanition	20.8	15.6	17.6
Premature birth	19.1	14.3	16.0
Congenital defects	3.9	3.9	4.6
Tubercular diseases	7.5	3.5	3.9
Syphilis	1.4	1.0	2.0
All other causes	15.9	14.5	15.
All causes	154	107	123

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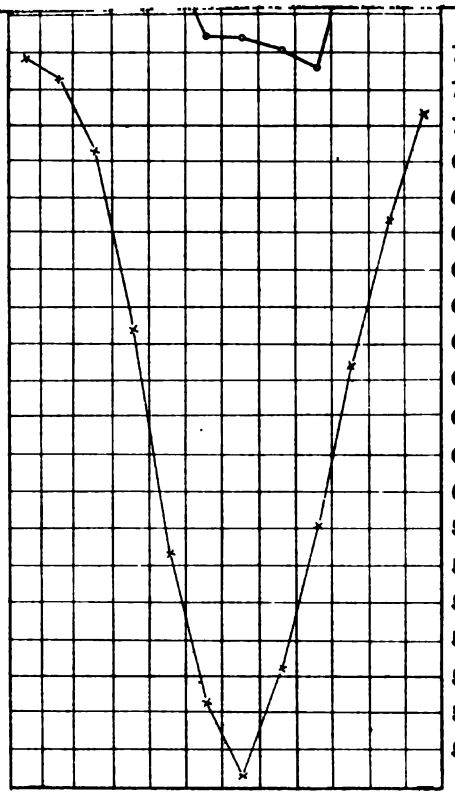
SYDNEY AND SUBURBS.



Mean Monthly Shade Temperature— 30 years.

SURVEYOR GENERAL'S OFFICE, ADELAIDE.

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Earth July 1st, 1902, to June 30th, 1905.

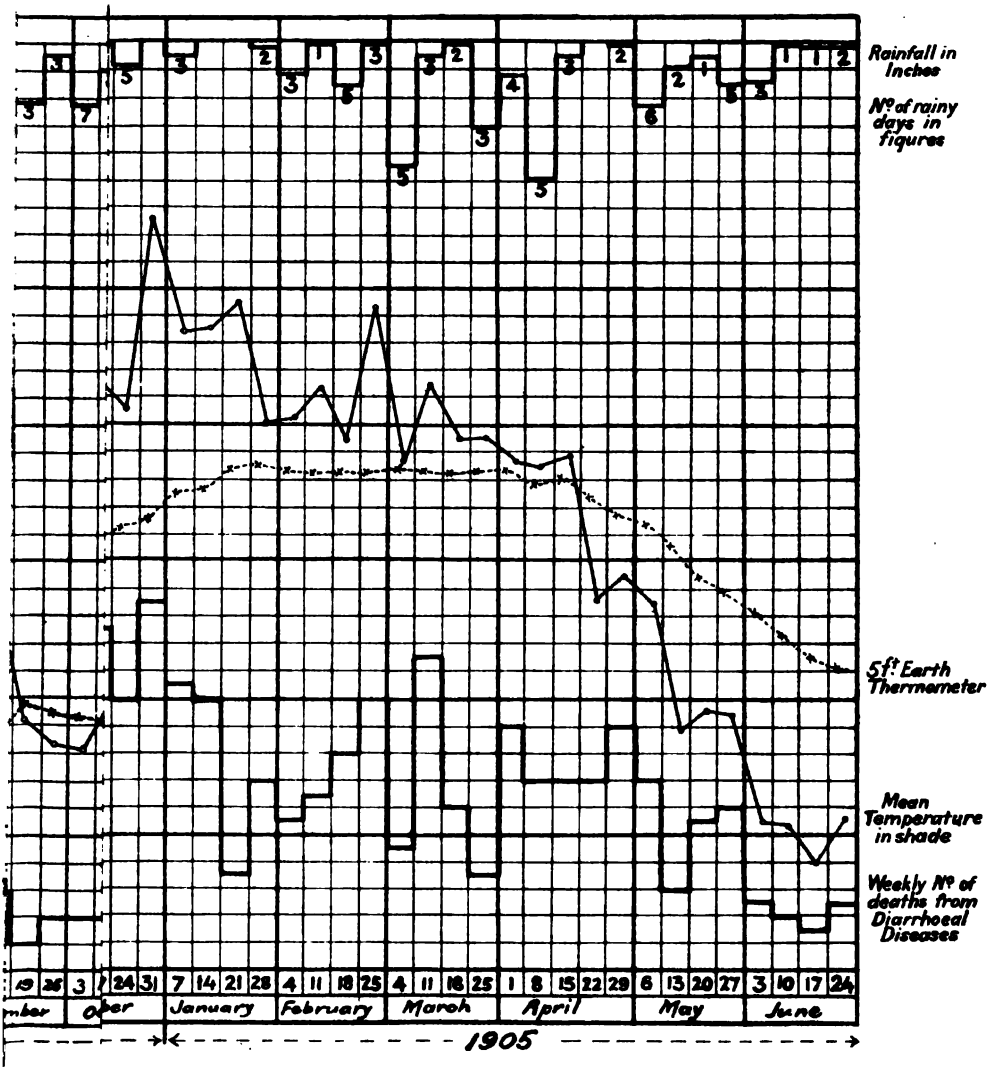
Rainfall in Inches
No of rainy days in figures
5ft Earth Thermometer
Mean Temperature in shade
Weekly No of deaths from Diarrhoeal Diseases
19 26 3 10 24 31 7 14 21 28 4 11 18 25 4 11 18 25 1 8 15 22 29 6 13 20 27 3 10 17 24
mber
October
January
February
March
April
May
June
1905

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TABLE II.

SYDNEY AND SUBURBS.

Deaths of Infants Under One Year per 1,000 Births for the Years 1875-1904, inclusive.

1 Year.	2 All Zymotic Diseases (except Diarrhœa).	3 Pneumonia and Bronchitis.	4 Diarrhœal Diseases.	5 Convulsions.	6 Dentition.	7 Atrophy, Debility, and Inanition.	8 Prematurity.	9 All Congenital Defects.	10 Tubercular Diseases.	11 All Causes.	12 Combined Infantile Mortality from Causes in Columns 4, 5, 6, and 7.
1875....	22.0	14.1	24.7	18.6	8.4	43.6	15.0	2.6	10.9	176	95.3
1876....	19.0	12.2	26.1	20.1	5.2	40.7	13.0	3.1	13.2	169	92.1
1877....	15.9	10.8	32.5	16.6	7.4	28.4	11.9	3.6	10.5	156	84.9
1878....	25.8	13.1	29.6	19.8	5.0	32.5	11.9	3.6	9.5	173	86.9
1879....	9.5	11.5	36.3	20.6	5.1	30.8	10.4	1.8	9.8	153	92.8
1880....	18.4	21.1	32.2	24.2	5.6	43.2	13.6	3.2	7.5	192	105.2
1881....	12.1	14.8	32.1	22.8	4.3	35.5	8.9	2.5	9.2	162	94.7
1882....	12.7	17.1	42.0	20.6	5.9	36.9	12.1	3.6	9.3	183	105.4
1883....	10.3	13.8	31.9	19.0	4.6	38.8	12.9	2.7	9.2	163	94.3
1884....	12.3	15.7	36.1	21.3	3.4	40.5	10.3	4.1	9.7	172	101.3
1885....	10.9	14.5	39.9	22.3	6.8	42.2	13.3	2.5	11.1	186	111.2
1886....	11.3	14.5	37.3	17.4	5.9	37.2	12.4	4.6	11.3	173	97.8
1887....	6.3	10.5	34.6	14.2	6.3	22.1	12.6	3.4	9.5	140	77.2
1888....	6.9	12.3	39.0	14.5	5.2	30.9	14.3	2.9	9.3	152	89.6
1889....	14.8	16.2	44.4	14.0	6.6	25.5	13.6	4.3	8.7	172	90.5
1890....	6.2	14.6	31.2	13.3	3.9	20.0	15.9	3.1	6.2	135	68.4
1891....	8.7	17.7	34.5	13.6	4.0	23.9	14.8	3.8	5.1	148	76.0
1892....	7.8	11.0	30.5	10.4	5.0	23.7	14.4	2.5	5.5	130	69.6
1893....	12.6	11.7	37.9	13.5	1.6	25.7	13.9	4.5	5.8	147	78.7
1894....	10.0	10.6	35.4	10.9	.2	23.7	14.7	4.5	5.4	134	70.2
1895....	7.0	10.1	39.2	9.0	.7	21.7	15.1	3.8	5.4	130	70.6
1896....	3.7	9.7	45.4	9.5	.4	24.5	14.4	4.2	5.4	139	79.8
1897....	3.5	12.8	43.4	8.9	.4	21.2	17.1	3.7	3.9	129	73.9
1898....	14.7	17.9	46.1	9.5	.3	22.8	15.9	5.0	5.5	154	78.7
1899....	9.2	10.7	33.6	6.1	.2	16.8	17.1	4.6	4.7	120	56.7
1900....	2.8	8.3	36.4	7.6	.3	16.0	15.4	4.2	3.6	109	60.3
1901....	10.3	11.8	37.4	5.5	1.4	17.1	15.5	4.2	2.9	120	61.4
1902....	5.8	10.7	39.0	2.7	1.5	12.5	16.4	4.6	3.8	112	55.7
1903....	6.7	12.5	44.2	3.9	.8	12.5	14.5	5.4	2.3	116	61.4
1904....	5.1	8.4	31.0	3.2	.7	11.4	18.8	6.5	2.0	98	46.3

SEWAGE TREATMENT.

A REVIEW, BY E. S. STOKES, M.D., &c.

The necessity, in the interests of public health, for the appropriate treatment—as apart from the mere disposal—of sewage appears to have met with adequate recognition only within comparatively recent years.

The phenomenal overgrowth of cities and towns, especially during the latter half of the last century, and the concomitant advance of public opinion in hygiene, have been the principal factors in bringing into prominent notice the exigencies of the situation.

Matters first came to a head, so to speak, in England, after the passage of the Public Health Act of 1848, by virtue of which a large number of sewers were installed. There does not, however, seem to have been at this time any very serious or systematic endeavor, on the part of local authorities

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concerned, to purify the sewage before its discharge—which was, in most cases, into rivers. In consequence the rivers became seriously polluted, and a general outcry arose. In 1857 the first Sewage Commission was appointed, and, after sitting assiduously for over eight years, they declared that "the right way to dispose of town sewage is to apply it continuously to land; and it is only by such application that the pollution of rivers can be avoided." Obviously this opinion did not meet the requirements of the day, because we find a second Commission appointed two years later, in 1868. This Commission endured only for six years, *i.e.*, until 1874, when they presented their fifth and last report. The opinion of this Commission on the comparative methods of the classes of processes for the treatment of sewage, viz., chemical precipitation, intermittent filtration, and broad irrigation, may be stated thus:—(1) All these processes are, to a great extent, successful in removing polluting organic matters in suspension, but intermittent filtration is the best; broad irrigation ranks next, and chemical precipitation processes are less efficient. (2) But for removing organic matters in solution, the processes of downward intermittent filtration and broad irrigation are greatly superior to upward filtration and chemical processes.

The Commission defined "broad irrigation" as "the distribution of sewage over a large surface of ordinary agricultural land, having in view a maximum growth of vegetation (consistent with due purification) for the amount of sewage supplied"; and "intermittent filtration" as "the concentration of sewage at short intervals, in an area of specially chosen porous ground, as small as will absorb and cleanse it; not excluding vegetation, but making the produce of secondary importance. The intermittency of application is a *sine qua non* even in suitably constituted soils, wherever complete success is aimed at."

Nothing further was done in the way of appointing Sewage Commissions for the next eight years, but during this time the condition of the Thames became so bad from the quantity of sewage flowing into it that a third Commission was created in 1882, to investigate and report upon this overwhelming nuisance. After two years they reported that evils did exist, imperatively demanding a prompt remedy, and that by chemical precipitation a certain part of the organic matter of sewage could be removed. They also reported, however, "that the liquid so separated would not be sufficiently free from noxious matter to allow of its being discharged at the present outfalls as a permanent measure. It would require further purification; and this, according to our present state of knowledge, can only be done effectually by its application to land."

Guided by these reports, since 1884 the Local Government Board of England have required that any scheme of sewage disposal for which money is to be borrowed with their sanction should provide for the application of the sewage, or effluent, to an adequate area of suitable land before its discharge into a stream. This requirement has been a fruitful source of dispute between local authorities and the Central Board, especially in cases where the former satisfied themselves, by extended experiment, that they were able to produce an effluent better in many cases than the water, in the stream into which it was to be discharged, and, naturally, they considered it a gross injustice that they should be compelled to outlay large sums of money in the purchase of land which they did not consider they required, and which, if purchased, they could not use. Furthermore, the recent evolution of biological methods of sewage purification, and the undoubted degree of success attained by several of these processes, added great weight to the contention of local authorities that land treatment as demanded by the Local Government Board was not absolutely necessary.

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Mainly to settle this point, we find the fourth Sewage Commission appointed in 1898. To date they have issued four reports, with numerous appendices, containing valuable information; but so far their conclusions have been very few and very guarded. In an interim report issued in 1902, they state in reply to the question, "Are some sorts of land unsuitable for the purification of sewage?" "We doubt if any land is entirely useless; but, in the case of stiff clay and peat lands, the power to purify sewage seems to depend on the depth of the top soil. There are of course numerous gradations in the depths of top soils which are met with in nature, and it is not easy to draw the line between lands which contain a sufficient depth to justify their use and lands which do not. We are, however, forced to conclude that peat and stiff clay lands are generally unsuitable for the purification of sewage; that their use for this purpose is always attended with difficulty; and that, where the depth of soil is very small, say 6 inches or less, the area of such lands which would be required for efficient purification would in certain cases be so great as to render land treatment impracticable."

In the same report they answer the question—"Is it practicable uniformly to produce, by artificial processes alone, an effluent which shall not putrify and so create a nuisance in the stream in which it is discharged?" thus—"We are satisfied that it is practicable to produce, by artificial processes alone, either from sewage or from certain mixtures of sewage and trades refuse—such, for example, as are met with at Leeds and Manchester—effluents which will not putrify, which would be classed as good according to ordinary chemical standards, and which might be discharged into a stream without fear of creating a nuisance. We think, therefore, that there are cases in which the Local Government Board would be justified in modifying, under proper safeguards, the present rule as regards the application of sewage to land. No general rule as to what these safeguards should be can be laid down at present; and, indeed, it will probably always be necessary that each case should be considered on its own merits."

It is of interest to note, then, that in England, during the past forty-eight years, *i.e.*, since 1857, Commissions on Sewage have been sitting for fully twenty-three years, and that the last Commission is still at work endeavoring to solve the sewage problem. Also, during this time several Rivers Pollution Commissions have existed, which dealt largely with the pollution of rivers with sewage. So that, practically, during the past half century there have been very few years in which a Commission of one sort or the other was not engaged in making inquiries into some aspect of the subject.

The results attained do not seem altogether commensurate with the time and energy expended; but perhaps these seemingly barren results are due to the complexity of the problem. The first Commission went bald-headed for land treatment; the second was very strong on this point, but recognised chemical processes; the third appeared more kindly disposed towards the latter, but still demanded final land treatment before the discharge of effluents into streams; whilst the present Commission has gone so far as to express the cautious opinion that land treatment is not always necessary, and may, indeed, under suitable safeguards, be at times omitted.

But whilst Commissions have calmly pursued their way, and given birth to ponderous tomes and appendices, their efforts have not enabled them to give voice to an *ex cathedra* pronouncement as to which of the methods in use or advanced can be relied upon to effectually purify sewage under any conditions. Certainly the earlier Commissions were somewhat positive in their expressions, but, as subsequent development has shown that their conclusions were based upon what we now know to be insufficient evidence, the

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present Commission hesitates—no doubt, wisely—to say much on this point until they are able to speak in the light of more extended experience. I think it is rather too much to expect at any time a dogmatic answer to this question, but the least we might hope for would be a clear enunciation of the principles involved in the purification of sewage, together with a generalised account of their application under the variety of circumstances met with in practice.

Beyond all the work done by and on behalf of the above Commission, we have records of prolonged and expensive experimental investigation undertaken by interested corporations, both in Europe and America, and exhaustive reports on the subject by engineers, chemists, and biologists. Out of this class of information it is possible to glean, here and there, some facts which seem to indicate the direction in which we must turn our eyes to discover the real solution of the problem.

I propose shortly to review the several methods of treatment which are now in use, or which have been suggested or used at various times; and, in doing so, shall endeavor to trace as far as possible, in proper sequence, the principal developments that have occurred during the past fifty years.

In his evidence before the present Commission, Mr. Adrian, Assistant Secretary to the Local Government Board, subdivided the years from 1842 into three periods, viz. :—(1) 1842-1857, when the primary aim was to secure prompt removal of sewage from the neighborhood of dwellings; (2) 1858-1870, which was characterised by the abandonment of the notion of possible injury to health as a consequence of irrigation of land by sewage, and by the growth of the belief in the ideal system of sewage disposal as being that which should aim at its profitable utilisation by direct application to land, and should thus facilitate the protection of rivers from contamination; (3) the period from 1870 to the present time, with the dominant idea that the prevention of the pollution of streams is an indispensable requisite of every system of sewage disposal which can lay claim to efficiency.

In view of the already mentioned requirement of the Local Government Board as to the whole or partial land treatment of sewage before discharge into a stream, it is only natural that this form of treatment should have been a prominent feature in the past; and there is no doubt that, under suitable conditions and efficient management, it, either as intermittent downward filtration or broad irrigation, has produced most satisfactory results; and it is also certain that under contrary conditions it has caused grave nuisances.

The most recent statement on this subject will be found in the report of Drs. McGowan and Houston and Mr. Kershaw, on land treatment, to the present Commission. Their conclusions, generally, are favorable to land treatment. In summarising their opinion with regard to suitability of various soils, they say, with regard to filtration, that excellent results can be obtained from a light loamy soil overlying a porous subsoil; that a sandy soil and subsoil, and also a partially peaty soil overlying gravelly sand, are capable of yielding good results; that peat, pure and simple, is not well adapted for sewage purification, and that, with regard to chalk, they were unable to speak with confidence, owing to insufficient data. They further state that, with regard to broad irrigation, "heavy loam and clay soil, although not so well suited for sewage purification purposes, may yield fairly good effluents if the volume of sewage per acre is relatively small."

Thus almost any kind of soil can be used for the purpose of sewage purification, provided of course that the volume is proportionate to the purifying capacity of the soil in question. We are far from advocating the treatment of sewage on land which is, practically speaking, not well suited for the purpose,

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but this does not invalidate the truth of the proposition that the matter is nearly always one of degree of suitability, and seldom one of intrinsic disability.

Stress is laid upon the necessity for efficient screening and settling before applying sewage to land, unless it is delivered in a thoroughly disintegrated condition—such as may at times be seen in cases where the outfall works are situated some considerable distance from the gathering ground, and where, consequently, the sewage becomes altered during its long passage, or where the sewage is broken up by pumping.

The advantage of cropping is pointed out, and the best crops are stated to be those which can be more or less continuously sewaged without detriment, *e.g.*, quickly growing plants like rye grass, mangold wurzel, &c.

Some of their conclusions on other points may be quoted :—

" The effluents from land processes of sewage treatment are not, from the bacteriological point of view, in a proper condition for discharge into drinking water streams."

" The effluents from land possess a bacterial flora characteristic of sewage, and the microbes characteristic of soil (in the sense of being peculiarly abundant in soil) are relatively absent from land effluents.

" As a result of its treatment on land, and judged by the bacteriological tests employed in this investigation, sewage does not seemingly become modified in its biological characters to any material extent. The bacteria, however, were reduced in number to a marked extent.

" The few samples of subsoil water collected in the neighborhood of some of the sewage farms were usually found to be pure, both chemically and bacteriologically ; but this, of course, must not be regarded as proving that wells sunk in such situations are safe for domestic use, or free from serious objections.

[A footnote in reference to this says :—" Two years' work by one of us in the chemical and biological qualities of the Chichester well waters showed that shallow wells, sunk in polluted soil and subsoil, may show, on searching bacteriological examination, unequivocal evidence of excremental, and therefore potentially dangerous, pollution."]

" Generally speaking the discharge of effluents into streams did not exercise any marked prejudicial effect in the water of the stream ; indeed, the reverse was sometimes observed. There seems to be no reason to doubt that the effluents from properly managed sewage farms would, when discharged into non-polluted streams of relatively large volume, neither give rise to nuisance, nor, as far as may be judged by rate of absorption of oxygen, prove injurious to fish.

" There seems to be no reason to doubt that, with proper management, land can purify sewage for a practically indefinite period.

" As regards the likelihood of sewage farms being dangerous to health, we can do no more than tentatively express the opinion that no convincing proof has yet been furnished of direct or widespread injury to health in the case of well-managed farms. It may be possible that the foul emanations from a badly-managed or over-sewaged farm constitute an indirect source of danger to health by lowering the vitality of weakly and susceptible individuals."

To my mind, these quotations, in the main, fairly sum up the situation as regards land treatment at the present day ; but they can only be considered as confirmatory of previous views, and not as expository of new discoveries of any essential character. Whilst, therefore, we cannot accept that land treatment under certain conditions is an efficient method of purifying sewage, we must not ignore the fact that these certain conditions—nature and area of land available are purely local matters, and that many towns are so situated that they cannot obtain a sufficient area of suitable land. The recognition of these difficulties, and the general struggle for improvement in all directions, aided by

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increased knowledge—chemical and biological—so noticeable since the middle of the last century, have co-operated in the evolution of other methods of sewage treatment, the earliest of which may be classified under the heading of "Chemical processes."

Chemical processes for the treatment of sewage have been instituted with several objects in view beyond mere purification. The earliest chemical process actually used (1846) was the lime process, and this was employed apparently only for clarifying. Very many modifications of the lime process have since been introduced—such as combination of lime and tar, lime and tar and calcined chloride of magnesia, lime and carbolic acid, lime and clay, lime and prepared alkali waste, and lime and herring brine. The addition of some of these chemicals was with the idea of effecting disinfection. The clay was used with lime in connection with the manufacture of Portland cement. In addition, lime has been used to neutralise acidity of sewage, due to the entrance of trades wastes.

Alumina processes have also been extensively used. Salts of aluminium, either alone or in connection with lime, zinc salts, carbonate of soda, iron salts, permanganates, clay, &c., have been tried or recommended at different times. The well-known A. B. C. process (alum, blood, and clay) appears to have been one of the most successful of these, although at present time the blood is omitted.

Certain salts of iron form useful precipitants. Both ferrous and ferric salts have been employed—most generally the ferrous sulphate. The perchloride has been tried in combination with a similar salt of magnesia. Holden's process consists of the use of sulphate of iron, clay, lignite, charcoal, coal dust, and lime. It is stated that sewage treated with this shows considerable amounts of nitrates and nitrites, which is, of course, a sign that a certain degree of oxidation of nitrogenous matter in the sewage had taken place—a rather unusual event to occur after the action of a chemical precipitant. Ferrozone is a proprietary substance made up of a combination of salts of iron and alumina, and is recommended as an efficient precipitating agent.

At first exaggerated ideas prevailed as to the manurial value of the sludge obtained by the above chemical process. But experience and analysis both go to disprove this. The sludge contains very little nitrogen and potash, and perhaps a slightly greater amount of phosphorus. With the object of trying to arrest some of the more valuable constituents of sewage, a number of processes have been tried. The originators of the magnesia process hoped to precipitate ammonia and phosphates as triple phosphates, but failed to carry it out. Several phosphate processes have been experimented with. The process consists in the employment of soluble or superphosphate of magnesia, which was added to and mixed with sewage, and then precipitating it with lime or other precipitating agent; the idea being that, when superphosphate of magnesia was added to the sewage, it neutralised the ammonia, forming an insoluble triple phosphate of magnesia and ammonia, which is precipitated out of the liquid. For a variety of reasons all these processes failed.

Other chemicals have been used for the purpose of deodorising or sterilising sewage. Dibdin considers that it is quite possible to deodorise large volumes of sewage with manganates or permanganates and sulphuric acid. Manganese oxides are deposited in the sludge, and there possibly effect further oxidising effects. Chlorine and chlorine compounds act as conveyers of oxygen, and in that capacity have been applied to sewage, most commonly directly in the form of chloride of lime or bleaching powder, but also indirectly as hypochlorous acid formed by the action of an electric current passed through either the sewage itself or a saline solution—such as salt water. There is no doubt that chlorine in this form exerts a powerful destructive

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effect in the lower forms of life, but it is by no means satisfactorily shown that this electrolytic process is efficient in the purification of sewage. This process may be adopted for the sterilisation of an otherwise purified effluent which is discharged into a stream used for drinking purposes.

Although largely used and experimented with, chemical processes have failed to secure of themselves any very appreciable measure of success as agencies for the complete purification of sewage. The effluent produced may be clear, owing to the removal of suspended matter, but the organic matter in solution is left unaffected, and may even be added to in some cases by the solvent effect of the precipitant; consequently these effluents undergo secondary decomposition, and require further treatment before being allowed to flow into streams. This treatment usually consists of passage through filter-beds, or of methods mentioned under the heading of land treatment.

With the aid, then, of other processes, chemicals may be said to have attained the object aimed at, but at the cost of a serious disability—I refer to the production of sludge from the action of the precipitant in the sewage. This sludge consists of the matters in suspension plus the precipitants, and, of course, is always being produced and constantly calling for removal. At first it was believed that it would possess considerable manurial value, but this, as already stated, was shown to be incorrect; in fact, in this capacity it would not pay for cartage away. The perennial sludge difficulty with chemical processes undoubtedly stimulated experiment in other directions, the results of which now appear before us in the various methods usually classed under the term " bacterial " or " biological purification."

The explanation that formerly held with regard to the purification of sewage by land treatment, or the purification of effluents from chemical processes by filters, was that, apart of course from the mechanical straining, the organic matter was directly oxidised by the oxygen of the air by a simple chemical combination. The most important oxidation products being nitrates, it is only natural that attention should have been specially centred in these compounds. It is stated that, in 1862, Pasteur regarded it as probable that nitrification was the direct result of bacterial activity. In 1872 the Berlin Sewage Commission reported that sewage matters were converted into nitrates, not by a simple molecular process, but by organisms present in natural sewage and soil. In 1876, at the Paris Sewage Farm, Schloessing and Muntz demonstrated this by sterilising the soil with vapor of chloroform. These experiments were subsequently amply confirmed by other observers, but none of them were successful in isolating an organism able to build up nitrate from ammonia. In 1886 Heraeus claimed to have isolated pure cultures capable of transforming ammonia into nitrous acid. Other investigators, notably Frankland and Warrington, closely examined Heraeus' work, and came to the conclusion that the nitrous acid formed was due to the reduction of nitrate in the medium, or, at times, to absorption of the same from the air. The search after the nitrifying organisms, however, though at first negative, was not fruitless. Almost about the same time Jordan in America, the Franklands and Warrington in England, and Winogradsky in Germany, appear to have arrived at the conclusion that these organisms would not grow on the ordinary media, and they therefore tried weak ammoniacal solutions in combination with small quantities of salts of the alkalis, *i.e.*, a medium containing nothing but inorganic matter. Frankland and Winogradsky succeeded in isolating an organism that would build up nitrites, and Jordan one that completely nitrified (but this latter was probably a mixture). Since that time (1890) a vast amount of experimental investigation has been put in hand, so that we are now in possession of the main facts concerning both the building up and breaking down of nitric acid by bacteria. The first complete investigation into the treatment of sewage

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by filtration through specially prepared artificial beds was undertaken by the Massachusetts State Board of Health, from 1887 to 1900. They clearly established the fact that sewage could be completely purified by intermittent filtration through appropriate filters, and that the purification was due to bacterial agencies. All these inquiries, however, had main reference to oxidation, but, as it has been shown the nitrifying organism will not act upon nitrogen in organic combination, it must be apparent that there exists a preparatory stage in which organic nitrogen is converted into ammonia. The fact that solid excrementitious matter undergoes apparently spontaneous disappearance by liquefaction in cesspools had been noted undoubtedly before this, but no great importance appears to have been attached to such phenomenon until 1891, when Scott-Montcrieff began his experiments on a practical scale at Ashtead. His upward filtration tank is so well known that I need not attempt to describe it; it is sufficient for me to say here that his demonstration of the disappearance of sludge by natural agencies in the tank attracted general attention, and opened up a new era for sewage treatment.

In 1895, Cameron, of Exeter, introduced his "septic tank," and added further proof that the solid matter of sewage undergoes spontaneous liquefaction. Both Cameron's and Montcrieff's processes appeared to obviate the sludge difficulty of the precipitation processes, and the sewage problem seemed solved. But whilst it cannot be now claimed that these processes achieved everything that enthusiasts believed them capable of at first, still it must be admitted that Cameron's "septic tanks" especially have had a tremendous influence in shaping subsequent events in sewage treatment. Chemical processes have been dropped, and no one now thinks of aught but bacteriological installations. A host of inventors have arisen with many and wonderful valves, syphons, distributors, *et hoc genus omne*. We have few discussions on the relative values of the various systems as such, but on all sides we have advocates of closed septic tanks and open septic tanks, contact beds and continuous filtration, Montcrieff's tanks, Dibden's beds, ærating filters (such as Ducat's, Lowcock's, and Waring's, &c.), each being able to show that his system will effect a purification of over 90 per cent.

To attempt to describe fully the complex changes in dead organic matter that go on quietly in nature's laboratory would take us to realms beyond the scope of this paper; but a brief account may be acceptable. The final products of the process are fully oxidised compounds; nitrogen becomes converted into nitric acid, carbon into carbonic acid, and hydrogen to water. Organic matter, however, seldom oxidises directly to these ultimate compounds, except, perhaps, under such energetic influences as fire or electric discharges, but undergoes a preliminary disintegrating transformation to simpler compounds capable of easy oxidation by bacterial agency. In the first stage—which is found to proceed most rapidly under anærobic conditions—we see proteid matter converted into peptones, and peptones again split up into amido-compounds; amido acids are further decomposed into fatty acids and ammonia, and basic amines into hydroxy-compounds with evolution of of free nitrogen. Urea is decomposed into carbonate of ammonium generally before the sewage is received for treatment. The organic acids are reduced with the production of acid carbonates, free carbonic acid, hydrogen, and methane. Acetic acid is generally the penultimate product, but this eventually passes into the compounds and gases just mentioned. In formation of $C O_2$ here, the oxygen is derived from the original substance, and the process cannot be considered a parallel to what we shall see occurs in the later stage. Cellulose and fibrous matter may be hydrolysed with the formation of carbonic acid and marsh gas, and probably, at times, of fatty acids. Carbohydrates and fats, similarly, are decomposed into fatty acids and gases.

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The bacteria at work in this stage are anærobes—obligatory and facultative —and the absence of free oxygen probably causes the latter to break up a good deal more organic matter in their search for oxygen than they would do if it were readily available from the air. We observe here the gradual formation and splitting off of ammonia from organic nitrogen, with the concomitant elaboration of nitrogen free compounds. We also note the evolution of gases, hydrogen, nitrogen, and marsh gas, which escape into the air, and ammonia and carbonic acid, which remain in solution. We further see that no oxidation products are formed, with the exception of carbonic acid, as already explained.

The next stage, which does not seem to be generally recognised as distinct from the others, is one of partial oxidation. Here we find the free ammonia acted upon by organisms which produce nitrous acid, and, in addition, important chemical reactions have been recorded between the nitrous acid so formed and transitional products carried over in solution from the first stage—such as ammonia, amido-acids, and amines—resulting in the evolution of considerable volumes of free nitrogen. Nitrosification is said to proceed most rapidly in the presence of diffuse light and a moderate supply of oxygen.

In the final stage nitrites are converted into nitrates, and the carbonaceous residues oxidised as completely as possible.

The conditions for nitrification by bacteria are as follows :—1. The antecedent formation of first ammonia and then nitrous acid from the organic nitrogen ; (2) the presence of some fixed base ; (3) darkness, and free admission of air ; (4) absence of excess of alkalinity. Warington found that nitrification could not proceed if the alkalinity as ammonia exceeded 44.6 parts per 1,000,000.

Carbonic acid gas is also formed in abundance in this stage, and, unless provision is made for its rapid and continuous removal, nitrification will be delayed—if not altogether inhibited.

An interesting feature in connection with nitrification is the rapidity with which it takes place. Scott-Montcrieff found that in passing a tank effluent over his differential trays, in eight to ten minutes nine parts of nitric-nitrogen per 100,000 were formed—a very high figure. Probably in contact beds most of the nitrate production occurs as the beds are slowly emptying ; and whilst standing full the changes described as belonging to the second stage are happening.

A consideration of the above account will throw some light on the failure of such systems as Ducat's, Waring's, and Lowcock's, which sought to treat raw sewage in filters by direct oxidation. We see how nature does not provide for the direct oxidation of nitrogen, and therefore the systems are based upon incorrect principles. Any measure of success they may have attained can be accounted for by the fact that the sewage they were treating had undergone, before reaching the works, the changes pertaining to the first stage. The reason that nature does not permit of the direct combination of nitrogen with oxygen is obvious when we think of the composition of the atmosphere. If we could oxidise the nitrogen in the proteid molecule, by causing any extra blast of air to be directed on to it, we should soon see the whole world converted into an alkali desert, and we ourselves would be as Lot's wife.

Although bacterial purification of sewage has been sprung upon us of late as if it were a new invention—like wireless telegraphy—we must not fail to recognise that it is no new process, but that the identical changes so fully elucidated by numerous recent investigations have been going on in the world as long as living matter existed thereon. In short, it is an integral part of the economy of nature ; and, without the intervention of agencies to effect the changes necessary for the transformation of effete animal matter into substances suitable for absorption by the vegetable kingdom, life, as we know it, would soon disappear from the face of the earth. What we really have been

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doing is, so to speak, to harness the forces of nature, so that we can use and regulate them to work—not in any way contrary to their disposition, but under conditions most favorable for efficiency.

Glancing back over the last fifty years we can now recognise the effect of these unseen agencies in the evolution of sewage treatment. First we have land treatment, which has been generally successful, only failing when applied under inappropriate conditions: this is bacterial treatment pure and simple. Next we see the introduction of chemical processes, all of which ended in failure and necessitated subsequent treatment of the effluents, either on the land or on specially prepared filter beds. Once again bacteria are called in to the rescue. And, finally, we come to the present time, when, with a full appreciation both of the changes involved in the disintegration of organic matter and the building up therefrom of innocuous and important inorganic compound, and of the agencies whereby this metamorphosis is effected, we find ourselves surrounded by a multitude of systems of bacterial treatment—as numerous almost as the roads to heaven—each of which has its army of adherents ready to pledge themselves in all sincerity that theirs is the only system worth a second thought.

Out of all the apparent confusion can we gather any hope for the future? I think we can. It may be risky to attempt to prophesy, but in this instance I believe I shall be on safe ground in expressing the opinion that in the application of bacterial treatment we shall find the true solution of the sewage problem. It will not be solved by somebody's patent filter, or valve, or such-like, but the correct answer will be arrived at only by prolonged and careful study of each individual sewage we propose to treat, and the application thereto of a line of treatment—the details of which must be determined by actual experiment—based upon the broad principles already enunciated.

Finally, I am well aware that I have said nothing new nor original, and that I have not referred to many points of interest in connection with the subject; my object being to submit this review in the hope that it may be of interest, and possibly of some use, to the members of this Section. A wise physician makes full use of the *vis medicatrix naturæ*. We should never fail to remember that there is also a *vis purificans naturæ*.

TYPHOID FEVER IN NEW SOUTH WALES, 1898-1904.

By R. J. Millard, M.B., Ch.M., Sydney, D.P.H., Cambridge,

In 1898 notification of certain infectious diseases, including Typhoid Fever, became compulsory in New South Wales under the Public Health Act of the State. The notifications, sent in the first instance to the local authorities, are by them forwarded to the Board of Health of the State and recorded in the Notification Bureau of the Department of Public Health. Statistics for seven years have thus become available, and the object of the present paper is to review these statistics concerning Typhoid Fever, and the results of some investigations into outbreaks of the disease during this period.

For permission to use these official data I am indebted to the Chief Medical Officer of the Government and President of the Board of Health (Dr. J. Ashburton Thompson), under whose instructions I made the investigations into country towns typhoid referred to later on.

The importance of the subject is very great. Every year some hundreds of persons die of this disease in New South Wales, and some thousands are

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incapacitated from wage-earning or enjoyment of life by an illness which has an average duration of many weeks. Moreover, the most susceptible persons are adolescents and young adults, and the disease not infrequently permanently impairs the constitution and development. A similar state of affairs exists throughout the civilised world, and for many years the attention of hygienists and of sanitary authorities everywhere has been largely occupied with measures to elucidate and reduce typhoid incidence and mortality.

I have tabulated the figures under two main heads, Metropolitan and Country. It is necessary to separate metropolis from country because the conditions of life are largely dissimilar. The metropolis possesses an extremely good water supply, is extensively served by a very excellent system of sewers, has the advantage of energetic sanitary administration, offers facilities for hospital isolation, and last—but not least—being on the sea coast, has very different climatic conditions from the inland regions which form the greater part of the country districts.

For details concerning Typhoid Fever in the metropolis I am largely indebted to the excellent annual reports of the Medical Officer of Health for the Metropolitan Combined Districts (Dr. W. G. Armstrong).

Fatality.—The fatality has been :—

Metropolis.....	5,790 cases	568 deaths	— 9.8 per cent.
Country Districts.....	16,217 "	1,809 "	— 11.2 "
Whole of New South Wales	22,007 "	2,377 "	— 10.8 "

These percentages are very low. In England the fatality per cent. has generally been about sixteen.

Age and Sex Distribution has not differed, generally speaking, from that observed in other countries. The incidence has been heaviest on adolescents and young adults. The sexes have been affected fairly equally.

Incidence Rate.—Reckoned on the population figures obtained at the Census of 1901, the incidence of typhoid in the seven years has been as follows :—

	Population.	Typhoid Cases.	Annual Rate per 1,000.
Metropolis	487,900	5,790	1.69
Country Municipalities	369,476	11,682	4.52
Unincorporated portions of the State	497,470	4,535	1.30
All New South Wales	1,344,846	22,007	2.31

For comparison with these figures, it may be mentioned that the incidence rate of London, for the ten years 1893-1902, was .8. The incidence rate of the Sydney metropolis is thus more than twice, and of the combined country municipalities more than five times, the London rate.

The combined unincorporated districts have a rate lower than that of the metropolis, and less than one-third that of the country municipalities. This comparatively low rate is probably due mainly to the fact that these districts are all rural, and include few centres of population sufficiently large to suffer from their defective sanitary arrangements : most of the population is scattered on farms, stations, mining settlements, and small townships. Another contributory cause of the low rate is probably that many cases of typhoid in these country districts are never seen by a medical man, and consequently are never notified.

In the combined country municipalities the rate is 4.52. The marked excess of this over the metropolitan rate is mainly due to the climatic differences referred to more fully later on ; but is also largely attributable to the fact

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that these country municipalities possess many of the defects, and lack most of the advantages, usually attaching to urban conditions. Defective sanitation, lack of sewers, faulty methods of disposal of excreta and house refuse, especially in towns where considerable numbers of people are aggregated—these are, no doubt, important factors in producing the higher incidence on the country municipalities. Another point which deserves mention is the relative densities of population per acre and per house. In the country municipalities, containing, as most of them do, large areas of which only the central part is populated, the density of population per acre for the municipality is very much less than in the metropolis; but the number of persons per dwelling was found at the Census of 1901 to be the same, namely, 5.4, both for Sydney and suburbs and for the total country municipalities, while the sizes of the dwellings were smaller in the country municipalities. Of the total dwellings in either division, 37 per cent. in the country and only 32 per cent. in Sydney and suburbs had four rooms, or fewer, so that the country people were definitely more crowded in their dwellings. In connection with infantile diarrhœa, Newsholme considers density of population per house to be of more importance than density per acre, and it is at least interesting to find this theory supported by our New South Wales statistics concerning Typhoid Fever. Increase of numbers per house means diminution of power to isolate infectious cases, and this must necessarily influence the spread of the disease.

In the Metropolis the incidence rate has been heaviest on those municipalities in which the sanitary conditions were worst. Botany has had a bad pre-eminence in this respect throughout the period under review. From time to time different localities have been the subject of special mention in the annual reports of the Medical Officer of Health, on account of epidemic or increased endemic incidence. No outbreaks due to water-borne infection occurred. This was to be expected, as the metropolitan water supply is exceedingly good. Small localised outbreaks due to milk infection occurred in 1898, at Burwood, twenty-three cases, and in 1904, at Surrey Hills, again twenty-three cases. In 1900, in the Ultimo portion of the city, seventy-two cases occurred in a small area of which the Medical Officer of Health reported that "the only satisfactory thing, from a hygienic point of view, was the water supply. The dwellings were ill-constructed . . . . . and ruinous . . . . . yards badly drained, unpaved, and often filthy . . . . . old-fashioned sewers . . . . . house connections very defective . . . . . a few of the houses served with filthy cesspits . . . . . much of the area liable to flooding in heavy rain storms."

The municipality of Balmain (population 30,256) in 1901 had 183 cases, and in 1903 had 150 cases, the mean number for the other five years of the period 1898-1903 being 66.2. The increased incidence was in each instance carefully investigated, and it was found that the ordinary epidemic causes could be excluded. In 1901 the Medical Officer of Health suggested that the extensive disturbance of the upper layers of the soil, consequent on the active construction of sewers in Balmain, might have played a part in disseminating the disease. In 1903 it was concluded that "the diffusion of the disease was essentially due to the many grossly insanitary and unwholesome premises, and to the inadequate methods of disinfection adopted."

In the Country there have been marked differences in the incidence rate in different districts. The most striking general difference has been between the comparatively low rates in the coastal and the comparatively high rates in the inland districts. This is illustrated by the maps which I have had prepared, and, in more detail, by Table A, in which I have set out (1) the population at last Census, (2) aggregate typhoid cases for seven years, (3)

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incidence rate per 1,000 of population per annum, and (4) mean annual rainfall, for all municipalities throughout the State. The district divisions, viz. :—A, Coastal; B, Tableland; C, Western Slopes and Riverina; and D, Western Division, are those adopted in the Statistical Register of New South Wales for 1903; and the rainfall figures are those published by the Government Astronomer. I have inserted the figures for the metropolis at the head of the table, but have not included these in calculating the aggregate coastal incidence rate, for the reason stated above, that metropolitan sanitary conditions are so much superior to those of country towns. A measure of this superiority is in fact afforded by these figures. The metropolitan typhoid incidence rate for seven years is 1.69 per 1,000, while the other coastal towns, generally similar to the metropolis in climate and rainfall, and containing in the aggregate a population of 183,122, have for the same period a mean rate of 2.34 per 1,000. For the population figures I have drawn on the Census returns of March 31st, 1901. This date is very nearly midway between January 1st, 1898, and December 31st, 1904, the period for which the notifications are here tabulated.

It will be seen that the aggregate incidence rates and mean rainfalls for these four Divisions form a regular gradation, the incidence rate increasing with the diminution of the rainfall as follows :—

A. Coastal Division (excluding Metropolis)	Rainfall 40in.	Incidence rate	2.34
B. Tablelands	" 29in.	"	" 4.74
C. Western Slopes and Riverina	" 21in.	"	" 7.46
D. Western Division	" 13in.	"	" 10.03

Corresponding variations have been recorded for India by Major A. R. Aldridge, in a paper on "Enteric Fever and Sewage Disposal in Tropical Countries" (*Journal of Hygiene*, July, 1902). He says—"Of these twenty-four stations, the thirteen having the highest admission rates are geographically very distinctly separated from those having low rates, all being situated in or closely bordering on the dry dusty alluvial plain of Upper India . . . On the other hand, of the eleven stations having a relatively low rate, four are situated on or near the coast with a damp climate throughout the year, while the remainder are situated on rocky soils of volcanic origin, and are relatively free from dust."

The divisions of the State which I have tabulated differ in other respects than in rainfall. Thus the Coastal Division is, generally speaking, free from the extreme heat which characterises the interior. As regards dust, too, this depends not only on the rainfall but also on the winds. In the inland divisions dry west winds blow in the summer time and dust storms are common. The difference in rainfall may be supposed to exert a twofold influence —on the soil and so on the saprophytic existence of the typhoid bacillus, and on its dissemination in dust and in the water supply. Dwellers in the Coastal Division have less difficulty in getting a satisfactory source of supply than in the waterless west, where the choice is limited. And this suggests another difference, namely, in mode of living and diet. It is probable that, on the whole, the dwellers in the Coastal Divisions have a higher standard of living than the others; though, as only municipalities are under consideration, the difference is not, I think, very great.

As regards general sanitation, the divisions are in many respects similar. Sewerage is practically non-existent in all. The individual municipalities vary in the energy with which they avail themselves of their statutory powers for preservation of the public health; but, generally speaking, more attention is paid to these matters inland than in the coastal division—partly because the coastal towns lack the annual stimulus of a heavy typhoid list, and

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partly because they are generally older towns and slower to avail themselves of the comparatively recent legislation on the subject. Thus cesspits and inefficient scavenging are at least as common in the coastal as in the other divisions.

The cleanliness of any town depends largely on the contour of its site. Other things being equal, a hilly well-drained town will be cleaner and more healthy than a town on the plains. In this respect most of the coastal towns are superior to the western towns, which are, many of them, on very flat country. But the tableland towns generally are quite as well situated as the coastal; yet the tableland incidence rate is twice as heavy as the coastal.

The relative abundance of flies in the coastal and other divisions during the typhoid season is a matter on which I have no certain data, but it appears to be a general belief that flies are very much more numerous inland than on the coast.

It will have been noticed that while the divisional incidence rates form a regular gradation from coastal westwards, yet there are in each division municipalities which have rates differing widely from the mean. This is only to be expected in dealing with numerous comparatively small centres, where an inconsiderable outbreak in any one year will suffice to greatly exalt the incidence rate for the seven years period. These individual centres necessarily vary greatly in their local sanitary conditions, and in most cases a cause for the exaltation of the rate is to be found in some gross neglect of sanitation, such as continuance of the primitive cesspit, formerly so common in all our towns.

Such departures from the mean do not, however, invalidate the general observation that, in the four great divisions of the State, Typhoid Fever and rainfall have been inversely proportional. They may be taken as indicating that, though rainfall and the conditions accompanying its abundance or scarcity are apparently the factors having most influence on the incidence of the disease, yet that local circumstances may override this influence: which may be used to point the moral that even in the favorably situated coastal towns it is not safe to neglect sanitary principles and practice.

Annual Variation.—For the seven years the mean number of notifications per annum has been—New South Wales, 3,144; metropolis, 827; country, 2,317. Notifications above the mean were received in 1898 for country and metropolis, in 1900 for both metropolis and country, 1901 for metropolis only, 1903 for both metropolis and country. The highest figures for the two divisions respectively were, in 1900, metropolis, 1,045; and in 1903. country, 4,022. The year 1903 was remarkable for a very large amount of typhoid throughout the country districts, the figures for this year totalling more than the sum of the figures for 1902 and 1904. The metropolis shared in this rise, but only to a moderate degree. This year was marked, meteorologically, by the break-up of a long period of drought. In 1902 the rainfall throughout the State had been abnormally low, everywhere below the average, and in many places more than 50 per cent. below. The drought continued into the early part of 1903, but about the latter half of April there were general heavy rains. It is noteworthy that the greatest typhoid incidence was toward the end and after the break-up of the drought. And this necessarily suggests that cases may have been in part due to contamination of water supplies by the washing of long accumulation of surface dirt into wells and underground tanks. In the several outbreaks which were specially investigated by me there was, however, no evidence to support such a theory, wells and underground tanks being comparatively uncommon, and it is more probable that this was merely the autumnal incidence somewhat prolonged in consequence of the large amount of infectious material throughout the country, and of the mild weather which accompanied the rains.

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Seasonal Variation.—The notifications received during each fortnight of each year are tabulated in Table B, and in Tables C to I are shown curves obtained with these figures. These tables do not accurately indicate the actual seasonal incidence, as they are based on the dates of receipt of notifications at the Notification Bureau. The actual date of infection of any case is probably, as a rule, three or four weeks earlier. The tables for successive years are, however, comparable with one another, and show a striking similarity. Thus, in all, the curve begins to rise about the end of October and continues to rise, with perhaps a few breaks, until April, after which a descent begins, which is not complete until July. The figures then remain low until the October rise recommences.

No exact interdependence of these curves with meteorological conditions can be demonstrated, and in some respects they seem to be mutually independent. Thus the curves are similar for droughty and other years alike. In this connection I have collated and examined the rainfall and typhoid incidence for each month of the seven years in the metropolis and in certain representative country towns—Broken Hill, Tamworth, Bathurst, Wagga, and Albury—but without being able to satisfy myself of any direct connection between the two. The most obvious connection appears to be with the temperature, the summer rise and winter fall of temperature being the only meteorological conditions which recur with absolutely regular periodicity each year. The averages of the mean shade temperatures obtained at the Sydney Observatory during the past forty-four years are as follows :—

January...	71°.5	maximum	July.....	52°.3	minimum
February..	71°.0	"	August	54°.8	"
March	69°.3	"	September ..	58°.9	"
April	64°.5	"	October.....	63°.5	"
May.....	58°.4	"	November...	66°.9	"
June.....	54°.4	"	December...	69°.9	"

This may be taken as indicating, approximately, the seasons in other parts of the State also ; but the extremes of heat and cold are much more marked inland, where it is common to get summer maximum over 100° and winter minimum below 32°. The typhoid rise begins in the early summer, and coincides with the general rise of temperatures. The ascent continues, however, for some time after the shade-temperature has reached its maximum, and the decline does not begin until autumn. An important point, which should not be lost sight of, is that in the winter months, though cases continue to occur, anything in the nature of an epidemic is extremely rare. Why should this be so ? Infectious material is being produced, and food and water supplies are exposed to risks of contamination, yet the infection is not disseminated. Obviously some necessary conditions are lacking in winter which are supplied in summer.

In the multiplication of cases of typhoid two factors are of main importance : (1) the persistence and propagation of the bacillus in its saprophytic existence ; (2) the distribution of the virulent bacillus to susceptible individuals. As regards (1), repeated observations have shown that the typhoid bacillus is extremely resistant to cold. Curschmann ("Typhoid Fever," p. 35) states "The bacilli persist at temperatures as low as 10°C., and even repeated freezing and thawing of fluid containing the bacilli do not appear to destroy them (Chantemesse and Widal, Janowsky) . . . . . Prudden observed that typhoid bacilli kept in ice at a temperature between 1°C. and 11°C. retained their vitality for three months." But although it retains its vitality, the bacillus is probably in a state of hibernation at these low temperatures. In the laboratory it is found that the optimum temperature for culture is 37°C., and the limits 9°C. and 42°C. Thus it may be supposed that the lowered temperature of winter, while not killing the bacillus, tends to inhibit its growth. How is the infection handed on from one summer to another ?

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The notification figures show that throughout the country the occurrence of new cases ceases almost entirely in the winter, and examination of records of individual isolated country towns usually shows a complete cessation for many months. Yet in these same towns when the appropriate season comes round typhoid breaks out again, and frequently shows a special affinity for certain areas or premises which have in previous seasons been connected with other cases of the disease. It is impossible to avoid the conclusion that the case-free interval is bridged over by the saprophytic persistence of the bacillus. It is true that the researches of Horton Smith and others have shown that the bacillus may persist in the patient's urine for months and even years after convalescence, but the seasonal curves recurring year after year with absolute regularity show that such dissemination of bacilli is insufficient by itself to cause any considerable spread of the disease. For, just when there are the greatest number of convalescents at large, namely at the end of the autumn, the case incidence falls abruptly. The saprophytic theory is obscured by the conflicting results obtained in various experiments on the vitality of the typhoid bacillus in soil. Thus, on the one hand, Dr. Sidney Martin, in his report on the Antagonism of the Soil to the Typhoid Bacillus (L.G.B. Report, 1900-01), concludes as a general result of his investigations " that the typhoid bacillus has commonly only a short existence in the soil; that it is destroyed by the products of the putrefactive bacteria which exists in most cultivated soils." On the other hand, Professor Firth and Major W. H. Horrocks, in their " Inquiry into the Influence of Soil, Fabrics, and Flies in the dissemination of Enteric Infection " (*B.M. Journal*, September 7th, 1902), came to the following conclusions (*inter alia*) :—

1. That there is no evidence to show that the enteric bacillus, when placed in soil, displays any disposition or ability to either increase in numbers or grow upwards, downwards, or laterally.
3. That the enteric bacillus is able to assume a vegetative existence in ordinary and sewage-polluted soil, and survive therein for varying periods, amounting in some cases to as much as *seventy-four days*.
4. That the presence or absence of organic nutritive material in the soil appears to be a largely negligible factor . . . . . .
5. That an excess or great deficiency of moisture in soils appears to be the dominant factor affecting the chances of survival of the enteric bacillus in, or at least the possibility of recovering it from, the soil.
6. From fine sand allowed to become dry the bacillus can be recovered on the twenty-fifth day after inoculation.
7. From fine sand kept moist with either rain or dilute sewage bacillus cannot be recovered later than *twelfth day*, probably because it has been washed into the deeper sand layers.
9. From ordinary soil kept damp by occasional addition of rain water the bacillus can be recovered up to and on the *sixty-seventh day*.
10. From same soil kept damp with dilute raw sewage, recovery up to *fifty-third day*.
11. From same soil, kept damp with dilute sterile sewage, recovery up to *seventy-fourth day*.
12. That in similar soil, after heavy rainfall, the bacillus at once disappears from the surface layers.
13. That from same soil allowed after inoculation to become so dry as to be readily blown about as dust, the bacillus can be recovered up to the *twenty-fifth day*.
20. That the bacillus is able to survive in surface soil an exposure to 122 hours direct sunshine extending over twenty-one consecutive days. (Experiments conducted at Netley, in the months of June and July, 1902.)

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And in a later communication (*B.M.J.*, October 4th, 1902), Professor Firth draws attention to a paper by Professor E. Pfuhl, of Berlin, who found that after inoculation of various soils he could recover the enteric bacillus for various periods, as follows :—Moist garden earth, eighty-eight days ; dry sand, twenty-eight days ; and moist peat, twenty-one days.

As regards (2) —distribution—it is probable that flies and other flying insects play an important part in the distribution of infectious material. It is, I believe, a fact that flies are almost extinct in winter, and that they gradually increase through the summer until the first nip of returning cold weather kills them off in thousands. Thus the increase and decrease of flies are respectively coincident with increase and decrease of typhoid. How far they are casually related is probably not susceptible of demonstration ; but observations are on record which show that the same individual flies may frequent latrines and mess tents, and that it is possible for them to carry infectious material on their feet.

In an article on Typhoid Fever in South Africa (*B.M.J.*, March 8th, 1902) A. B. Dunne, M.B., B.C., Cantab. (late Civil Surgeon, South African Field Force), speaks of the " plague of flies " at Bloemfontein during the epidemic of enteric in 1900, and of the noticeable diminution in cases admitted to hospital coincident with the killing off of the flies by the cold nights of May and June.

So far I have dealt with typhoid mainly as occurring endemically in New South Wales. It may not be out of place to add something concerning some epidemic and exaggerated endemic manifestations which it has been my duty to investigate.

The year 1903 was remarkable for an unusually heavy incidence of typhoid throughout the State generally, and for epidemic outbreaks at several country towns.

1. Of these, Coonamble was the most striking example. Here in a town of 1,680 inhabitants there occurred 256 cases of typhoid fever in the eight weeks January 30th to March 26th. The disease manifested itself in typically epidemic form. For the first four weeks of the year only two cases were notified, and then in the next four successive weeks there occurred sixty-one, forty-six, seventy-one, and forty-six cases respectively. After this, as a direct result apparently of the measures adopted, the numbers dropped abruptly, and in the next four weeks were eleven, nine, five, and seven. The water supply of the town was above suspicion, being derived from an artesian bore 1,320 feet deep, and supplied to the town mains under continuous pressure from an overhead tank 70 feet above ground level. Samples taken from different points in the town were examined in the biological and chemical laboratories of the Department of Public Health, and found free from any indication of pollution. As regards household milk supply, the majority of patients had either used condensed milk or none at all. The cause of the epidemic outbreak appeared to be the consumption of summer drinks, and of a new and popular concoction known as " milk-shake," at a particular shop in the town. In this shop there were employed (1) a youth who, while mixing drinks, &c., was " out of sorts," and who a fortnight later was found to have advanced typhoid fever, and (2) a man, who at the same time was the sole attendant on his wife then ill with typhoid. Of the sixty-one cases occurring in the first fortnight (January 30th to February 12th) forty-one had consumed " milk-shakes " from this shop, and sixteen were secondary cases in already infected households. In the succeeding weeks of the epidemic, multiple cases in households became a prominent feature. Altogether sixty-one households had more than one case, and of these households four had six cases, five had five cases, eight had four cases, seventeen had three cases, and twenty-eight had two cases. These cases occurred, generally speaking, at such intervals after the primary case as to

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leave it scarcely open to doubt that they had been infected directly or indirectly from the primary case, and not from the one common cause. Opportunities of such infection were many. Isolation at home was impracticable, and the members of the household generally shared the duties of nursing, with its manifold risks. Besides this, the method of disposal of infectious excreta was calculated to promote infection. Instead of using the special closet pails provided by the council, most householders attempted to "burn" the stools and urine. The *modus operandi* was to make a fire, and pour the stools, &c., over it. Perhaps the fire was made in a hole. In any case the result was merely that the fire was extinguished and the liquid material soaked into the ground practically unaffected. In this way the back yards became infected. Agencies for carrying infective material into the houses and into food were supplied by flies, and by the small whirlwinds of dust prevalent at that time of the year. The measures adopted to check the epidemic comprised rapid extension of the local hospital from twenty to 150 beds, removal thither of every case as it occurred, and disinfection of premises, especially of back yards. With these measures the outbreak ceased rapidly, as above stated; and it is interesting to note that only nine cases occurred in Coonamble in the following year, 1904.

In the same year, 1903, examples of increased incidence almost amounting to epidemics were afforded by Tamworth, Bathurst, Cobar, and other country towns.

2. Tamworth, population 5,799, had 126 cases in the period January 14th to May 20th. The heaviest incidence was in the fortnights ending April 8th and 22nd, and May 5th, when twenty-four, twenty-nine, and thirty-one cases respectively were reported. No epidemic cause could be ascertained. Water supply was not obtained from a common source by any considerable number of cases. Almost every household used rainwater caught on the roof and stored in iron tanks. Milk was very scarce in consequence of the drought, and was not used at all by the majority of the patients. Employment or school attendance could not be incriminated. Association with previously infected premises was a most striking feature of the outbreak. A map of the town on which were "spotted" residences of all cases which had occurred in successive years, demonstrated this association very clearly. With few exceptions, the cases reported in 1903 occurred on premises or in blocks which had been invaded at least once, and in some instances every year, since 1898. This was especially noticeable in West Tamworth, where the majority of the cases occurred. Here again, as at Coonamble, the disposal of the infectious excreta was almost universally faulty, the practice having been to bury it in the back yards. This practice had prevailed for many years, and it was easy to believe the soil had been in this way repeatedly infected. But it was less easy to explain why the outbreak should have been so much more extensive in 1903 than in previous years, unless the explanation was to be found in the climatic peculiarities of this season. The summer had been unusually dry. In January and February respectively only .47 and .38 inches of rain fell at Tamworth. This was considerably less than for the same period in any of the five preceding years. In March 2 inches, and in April 2.5 inches fell, and the rapid increase of typhoid cases coincided with this rainfall. It should, however, be noted that at the same period there was a markedly exaggerated incidence of typhoid fever in other country towns where the same relationship to rainfall could not be demonstrated. The only condition in common was that the country had suffered from a long severe drought, but how this stimulated the typhoid bacillus to such vigorous vitality was not clear.

3. Bathurst, population 9,327, had 135 cases in the twenty-four weeks ending June 17th. The cases were fairly evenly distributed over this period,

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the heaviest incidence being in the middle eight weeks ending April 22nd, in which seventy-three cases were reported. For the previous five years the notifications had been—1898, 35; 1899, 14; 1900, 115; 1901, 36; and 1902, 22 cases. In 1903 the gradual increase of cases made it improbable that there was any epidemic cause such as infection of water supply, and investigation of individual cases confirmed supposition. The town water supply was as a matter of fact a good one, derived from a shaft and a tunnel in the drift of the Macquarie River, well above the town, whence the water is pumped to a high-level reservoir and distributed under continuous pressure through the mains. Of 1,691 premises supplied by the town water mains only 105 were invaded by the disease, and there was no indication of any special incidence along the course of any particular main or mains. The spread of the disease appeared due to defective sanitary conditions combined with imperfect isolation of patients and disinfection of excreta. Bathurst is an unsewered town, and in consequence the disposal of household slops is generally unsatisfactory. At best the slops are but led to the nearest street gutter, and as a rule the house drains are not watertight. The disposal of household garbage and refuse was in 1903 equally unsatisfactory. Systematic regular removal was not enforced, and consequently on most premises there was an ashpit or ash heap, where the accumulated garbage of months was thrown, and which was probably never thoroughly cleared away. Moreover, on this heap bedroom slops were commonly cast, and in some instances even typhoid stools were buried there. Instructive examples of the spread of the disease under these circumstances were observed, of which two may be quoted here :—

(1) In a small terrace of six houses in Piper Street, four cases occurred, three houses being invaded. The first case, Veronica G., aged 12 years, was notified on December 24th; three weeks later, on January 13th, her sister Ruby, aged 16 years, was notified; four weeks later Linden H., living three doors away, and three days after him Marie J., aged 45 years, living next door to the first case. A defective open gutter ran down immediately at the back of the terrace receiving the slop waters from all the houses, and close by was an ancient "ash heap" common to all the houses.

(2) In another household four cases occurred, as follows :—

D.R., female, 17 years—Notified February 14th, removed to hospital February 12th, discharged March 18th.

A.R., male, 12 years—Notified March 26th, removed to hospital March 24th, discharged March 16th.

L.R., female, 6 years—Notified April 20th.

M.R., female, 15 years—Notified April 30th.

On these premises the usual drainage and garbage defects existed.

4. Cobar, population 3,371, had 118 cases in the twenty weeks ending May 20th, 1903, and of these, ninety-seven cases occurred in the latter half of this period. For the previous five years the cases notified had been successively 12, 23, 68, 38, and 14. As regards causation, it was not possible at Cobar to exculpate the water so satisfactorily as at the other towns already mentioned. The town supply was indubitably not of good quality, and it was being newly laid on at the time when the cases were becoming numerous. But many of the cases occurred on premises not yet connected with the mains, where rainwater only was used for drinking purposes, and throughout the town there were instances of recurrence of the disease on premises infected in previous years, and of secondary cases in an already infected household. These circumstances brought this outbreak very closely into line with the outbreaks at the other towns. Here also there were no sewers, and

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the yards were fouled with slop waters and house refuse. The "spot" map showed that in certain blocks typhoid had recurred year after year, suggesting very strongly that the source of infection was persistent and localised on these premises.

As examples of the handing on of infection from case to case there may be instanced three hotels, of which one had seven and the others each six cases :—

1. Hotel X.—Seven cases—
   - i. H. G., 40 years, lorry-driver, lodged at hotel. Onset about 25–12–03. To hospital, 2–1–03; discharged, 6–2–03.
   - ii. J. E. McL., 23 years, miner, lodger. Onset, 10–3–03; hospital admitted, 17–3–03; discharged, 2–4–03.
   - iii. J. K., 45 years, blacksmith, lodger. Onset, 3–4–03; hospital admitted, 10–4–03; discharged, 12–5–03.
   - iv. J. O'D., 34 years, tailor, boarder only. Lodged at his shop. Onset, 6–4–03; hospital admitted, 8–4–03; discharged, 7–5–03.
   - v. J. 40 years, miner, arrived from Canbellego ill on 23–4–03. Hospital on 26–4–03. N.B.—This patient evidently did not receive his infection at this hotel, but he contributed to infect the premises, and is included on that account.
   - vi. W. R., 28 years, engine-fitter at mine, lodger. Onset, 3–5–03; hospital, 10–5–03.
   - vii. D. McP., 33 years, miner, lodger. Onset about 13–5–03. Under treatment at hotel, 20–5–03.
2. Hotel Y.—Six cases—
   - i. M. H., 11 years, schoolgirl. Onset about 10–2–03; hospital admitted, 13–2–03; discharged, 22–2–03; re-admitted, 3–3–03; discharged, 25–3–03.
   - ii. N. M., 26 years, servant, came from Dubbo, and after staying for about a week at the hotel, fell ill about 10–3–03; hospital admitted, 17–3–03; discharged, 7–4–03.
   - iii. A. H., 33 years, cook, mother of H. M. (No. 1). Onset, 14–3–03; hospital admitted, 21–3–03; discharged, 2–4–03.
   - iv. C. R., 22 years, miner, lodger. Onset about 1–4–03; hospital admitted, 5–4–03.
   - v. J. K., 30 years, miner, lodger. Onset about 15–4–03; hospital admitted, 22–4–03.
   - vi. W. H., 38 years, miner, lodger. Onset, 25–4–03; hospital admitted, 2–5–03.
3. Hotel Z.—Six cases—
   - i. W. B. G., 22 years, assistant C. P. S., boarded only, lodged elsewhere. Onset about 4–3–03; hospital admitted, 8–3–03; discharged, 4–4–03.
   - ii. B. D., 22 years, female, cook's assistant. Onset about 5–3–03; hospital admitted, 9–3–03; discharged, 22–3–03.
   - iii. P. F., 31 years, part proprietor of hotel. Onset about 15–3–03; hospital admitted, 22–3–03; discharged, 18–4–03.
   - iv. A. R., 25 years, barmaid. Onset about 15–3–03. Nursed at hotel in room adjoining bar.
   - v. C. W., 37 years, female cook, shared room with B. D., and helped nurse her. Onset, 22–3–03; hospital admitted, 29–3–03; discharged, 28–4–03.
   - vi. C. F., 34 years, part proprietor. Onset about 23–4–03. Being nursed in hotel.

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In considering the data and experience relating to Typhoid in New South Wales in the seven years to 1904, four points appear to me to especially merit emphasis:

1. That typhoid has very regularly recurring seasonal variations, which correspond closely to those observed in other parts of the world. Moreover, there is a close resemblance in this respect between typhoid and infantile diarrhœa, which also is believed to be due to a micro-organism having naturally a partially saprophytic existence.

2. That for its dissemination the typhoid bacillus is very dependent in some way on external conditions, as exemplified in the difference between the incidence rates of the coastal and inland districts. How these conditions act must be largely a matter of speculation, but in the present example the rainfall would appear to be the most important constituent of the climatic differences.

3. That infection of water supplies has not, in New South Wales, been an important factor in spreading typhoid fever. It is necessary to insist upon this, because the medical and lay mind has become strongly imbued with the theory that in water, and in water alone, is to be found the *causa causans*. As the Frenchman in difficulties of another kind promptly suggests, *cherchez la femme*, so the average would-be sanitarian, confronted with a typhoid outbreak, at once turns to the water supply, and is satisfied if he can ingeniously prove that it has been exposed to the danger of contamination by sewage. I do not for a moment suggest that the water supply has not been the vehicle of infection in many epidemics. The circumstantial evidence is very strong, and is vouched for by witnesses of the highest standing. But other means of spread have, as a matter of fact, proved to be more common, and therefore of more importance, in New South Wales; and it is to be feared that the too earnest attempt to sheet home the crime to the water supply often diverts attention from simpler and more obvious causes, as, for instance, the fouling of the back yard by infectious excreta.

4. That infection from a previous case, either directly in the sick-room, or indirectly from excreta imperfectly disinfected or improperly disposed of, has been a factor of great importance. It is not only in New South Wales that the casual importance of association with infected persons has been noted. Recently the trend of skilled opinion has been strongly in this direction, and many observers reporting on outbreaks have emphasized the influence of this, rather than the more conventional epidemic cause—water-borne infection. As an example may be quoted the very complete official report on typhoid in in the volunteer camps of the United States Army during the war with Spain in 1898 (summarised in the *B.M. Journal* of July 26th, 1902); and additional evidence in abundance is furnished by the many articles and discussions reported in the medical journals, more especially in connection with Typhoid Fever in South Africa and India. A notable apostle of this doctrine is Professor Koch, who, in an address at the Kaiser Wilhelm's Academy, on November 27th, 1902, emphasized the importance of personal, as distinguished from water-borne, infection (*B.M.J.*, February 28th, 1903).

From this it results that the two essentials in dealing with any outbreak are (1) to isolate the sick, (2) to disinfect their excreta. Proper isolation and treatment can rarely be secured except in hospital, and therefore every case should, as early as possible, be removed thither. The necessity for this is not sufficiently appreciated, and too often the medical attendant consents to or even advises the home treatment of the patient, though it must be obvious that it will be impossible to secure effective disinfection of soiled clothing and of excreta. And this brings me to the all-important matter of the disposal of excreta. It is now well known that the bacilli may occur in both urine and stools, and consequently both these matters are to be disposed of. Where sewers are available, disposal is simplified; but it is otherwise in the

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unsewered country towns. Here various expedients are adopted. Burning is frequently attempted, on the alleged advice of the medical attendant, but is manifestly impracticable unless there is available sufficient absorbent material, such as sawdust, to soak up the liquid, and a furnace into which the mass may be cast. Otherwise the net result is a mild degree of desiccation, with, presumably, little injury to the vitality of the bacillus. Burying on the premises is wonderfully common.. Throughout the country I have found this practice adopted; and again the medical attendant is generally credited with advising it. Even when the local council is willing and anxious to supply a special pail for the removal of typhoid excreta, I have found the householder diligently digging little potholes about his back yard, and sowing his soil with infectious material. Sometimes he attempts a preliminary "disinfection" with chemicals, but it may be taken as a general rule that the average householder cannot be trusted to use disinfectants effectually. The most effectual and simplest method of disinfection is *boiling*. This may be easily done in the back yard in a kerosine tin over an improvised hearth of a few bricks. If the infectious slops as produced are poured into the tin and kept covered, it will probably be sufficient to boil the contents night and morning. After boiling, the excreta, now sterilised as regards typhoid bacilli, may be emptied into a closet-pail for removal in the usual way. Little, if any, nuisance is caused by the boiling if care be taken not to allow the contents of the vessel to get burnt. At the temporary hospital at Coonamble, all excreta were thus disposed of in several large washing-coppers, which were placed at a convenient distance from the wards. The contents of bed-pans and urinals, and any soiled waters, were cast direct into the coppers, under which fires were kept burning. At suitable times the council's contractor removed the contents to the ordinary nightsoil depot. As there were at one time over 140 typhoid patients in the hospital, this method was fairly thoroughly tested. The result was eminently satisfactory, both as regards absence of nuisance and non-infection of the nursing staff.

TABLE A.

TYPHOID FEVER IN NEW SOUTH WALES, 1898–1904.

Typhoid Incidence and Rainfall in the Municipalities of the Several Divisions of the State.

Municipality.	Population, Census 1901.	Typhoid Cases Notified 1898–1904.	Rate per 1,000 per Annum.	Rainfall, Mean Annual, in Inches.
A.—COASTAL DIVISION.				
Metropolis	487,900	5,790	1.69	50
I. North Coast—				
Ballina	1,819	11	.86	75
Casino	1,926	111	8.23	44
Coraki	770	23	4.26	50
Grafton (North and South)	5,147	93	2.58	39
Kempsey	2,329	4	.24	50
Lismore	4,378	78	2.54	55
Maclean	1,333	7	.75	51
Murwillumbah (incorporated, 1902)	772	8	—	—
Port Macquarie	1,160	2	.24	63
Taree	871	1	.16	47
Ulmarra	1,722	4	.33	40
Wingham	556	2	.51	46
Aggregate North Coast (deducting Murwillumbah)	22,011	336	2.18	51

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TABLE A—*continued.*

Municipality.	Population, Census 1901.	Typhoid Cases Notified 1898-1904.	Rate per 1,000 per Annum.	Rainfall, Mean Annual, in Inches.
A—Coastal Division—*continued.*				
II. Lower Hunter—				
Dungog	1,082	56	7.39	35
Gosford	751	27	5.13	50
Greta	861	56	9.29	34
Maitland (East and West)	10,073	388	5.50	34
Morpeth	1,288	25	2.77	41
Muswellbrook	1,710	65	5.43	24
Newcastle and Suburbs	54,991	1,142	2.96	48
Raymond Terrace	823	13	2.25	38
Singleton (North and South)	2,872	134	6.66	30
Aggregate Lower Hunter	74,451	1,906	3.66	37
III. County of Cumberland—				
Auburn	2,948	16	.77	31
Bankstown	1,246	6	.68	31
Cabramatta and Canley Vale	980	6	.87	31
Campbelltown	2,152	9	.59	29
Castlereagh	609	7	1.64	29
Dundas	1,087	6	.78	36
Ermington and Rydalmere	1,231	20	2.32	36
Granville	5,094	46	1.29	31
Ingleburn	362	—	—	30
Liverpool	3,901	34	1.24	30
Mulgoa	476	1	.30	29
Parramatta	12,560	143	1.63	36
Penrith	3,539	55	1.41	29
Prospect and Sherwood	3,259	13	.57	36
Richmond	1,202	7	.83	32
Rookwood	4,496	16	.50	31
Smithfield and Fairfield	1,643	7	.60	31
St. Mary's	1,840	5	.38	29
Windsor	2,039	46	3.22	28
Aggregate County of Cumberland (omitting Metropolis)	50,664	448	1.26	31
IV. South Coast—				
Bega	1,898	62	4.66	34
Berry	1,990	13	.94	64
Bowral	1,752	26	2.12	39
Braidwood	1,551	75	6.90	26
Broughton Vale	322	—	—	64
Camden	1,719	11	.91	29
Gerringong	1,051	4	.54	51
Illawarra, Central	4,664	16	.49	43
Illawarra, North	3,190	25	1.11	43
Jamberoo	1,291	—	—	53
Kiama	1,769	16	1.29	55
Mittagong	1,210	20	2.36	39
Moruya	1,099	4	.52	38
Moss Vale	1,385	6	.61	40
Nowra	1,904	9	.67	40
Picton	1,053	4	.54	30
Shellharbor	1,929	4	.29	53
Shoalhaven South	909	—	—	40
Ulladulla	1,765	4	.32	48
Wollongong	3,545	14	.56	43
Aggregate South Coast	35,996	313	1.24	44
Grand Aggregate Coastal Division (omitting Metropolis and Murwillumbah)	183,122	3,003	2.34	40

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TABLE A—*continued.*

Municipality.	Population, Census 1901.	Typhoid Cases Notified 1898-1904.	Rate per 1,000 per Annum.	Rainfall, Mean Annual, in Inches.
B.—Tableland Division.—				
I. Northern Tableland—				
Armidale	4,249	76	2.55	32
Bingara	879	44	7.15	30
Glen Innes	2,918	90	4.40	33
Hillgrove	2,274	37	3.25	36
Inverell	3,293	185	8.02	30
Manilla (incorporated 1901)	888	17	—	—
Quirindi	1,676	86	7.33	28
Tamworth	5,799	379	9.33	28
Tenterfield	2,604	5	.27	34
Uralla	681	4	.83	34
Walcha	980	16	2.33	30
Aggregate Northern Tableland (deducting Manilla)	25,353	922	5.19	31
II. Central Tableland—				
Aberdeen	749	43	8.20	20
Bathurst	9,223	393	6.03	24
Blayney	1,529	151	14.10	30
Carcoar	578	52	12.85	30
Cowra	1,811	71	5.60	26
Cudgegong	2,985	18	.86	26
Gulgong	1,579	23	2.03	27
Hill End	643	3	.66	28
Katoomba	2,270	22	1.38	60
Lithgow	5,268	127	3.44	35
Molong	1,254	58	6.60	30
Mudgee	2,789	97	4.96	26
Orange (West and East)	6,331	238	5.37	34
Wellington	2,984	144	6.89	23
Scone	1,145	53	6.61	24
Murrurundi	1,235	5	.57	32
Aggregate Central Tableland	42,373	1,498	5.05	30
III. Southern Tableland—				
Bombala	986	6	.86	24
Burrowa	839	14	2.38	19
Cooma	1,938	38	2.80	20
Goulburn	10,612	218	2.93	26
Grenfell	869	57	9.37	26
Queanbeyan	1,219	20	2.34	24
Tumut	1,391	142	14.58	32
Yass	2,220	45	2.89	24
Young	2,755	42	2.17	25
Aggregate Southern Tableland	22,829	582	3.64	23
Grand Aggregate Tableland Division (deducting Manilla)	90,555	3,002	4.74	29
C.—Western Slopes and Riverina Division.				
I. North-Western Slope—				
Coonamble	1,680	419	35.63	21
Gunnedah	1,910	76	5.68	25
Moree	2,298	83	5.16	23
Narrabri (West and East)	2,963	336	16.20	25
Warialda	875	5	.82	28
Aggregate North-Western Slope	9,726	919	13.49	24

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TABLE A—*continued.*

Municipality.	Population, Census 1901.	Typhoid Cases Notified 1898–1904.	Rate per 1,000 per Annum.	Rainfall, Mean Annual, in Inches.
C—Western Slopes and Riverina Division—*contd.*				
II. Central Western Slope—				
Condobolin	1,078	84	11.13	18
Cudal	599	9	2.14	25
Dubbo	3,409	221	9.26	25
Forbes	4,294	358	11.91	19
Narromine	931	53	8.13	18
Parkes	3,181	174	7.81	20
Peak Hill	1,107	10	1.29	22
Warren	1,175	81	9.84	18
Aggregate Central Western Slope	15,774	990	8.96	21
III. South-Western Slope—				
Albury	5,823	111	2.72	28
Cootamundra	2,424	159	9.37	24
Corowa (incorporated 1903)	2,046	7	—	—
Gundagai	1,487	45	4.32	24
Junee	2,190	77	5.02	21
Murrumburrah	1,448	18	1.77	24
Temora	1,603	77	6.86	20
Wagga Wagga	5,108	260	7.27	22
Wallendbeen	736	9	1.74	22
Wyalong	1,575	63	8.31	14
Aggregate South-Western Slope (minus Corowa)	22,334	819	5.24	22
IV. Riverina—				
Deniliquin	2,644	59	3.19	16
Hay	3,012	212	10.05	14
Hillston	843	5	.84	14
Moama	928	5	.76	17
Narrandera	2,255	32	2.02	18
Jerilderie	744	1	.38	15
Aggregate Riverina	10,426	314	4.30	16
Grand Aggregate Western Slopes and Riverina Division (omitting Corowa)	58,260	3,042	7.46	21
D.—Western Division.				
I. East of the Darling—				
Balranald	741	41	7.90	12
Bourke	2,609	245	13.41	16
Brewarrina (incorporated 1901)	683	8	—	—
Cobar	3,371	367	15.55	15
Gladstone (incorporated 1899)	1,171	36	—	15
Nyngan	1,455	50	4.90	16
Wentworth	642	21	4.67	12
Aggregate East of the Darling (omitting Brewarrina and Gladstone)	8,818	724	11.84	14
II. West of the Darling—				
Broken Hill	27,500	1,872	9.72	9
Silverton	286	—	—	9
Wilcannia	935	39	5.95	13
Aggregate West of the Darling	28,721	1,911	9.50	10
Grand Aggregate Western Division (omitting Brewarrina and Gladstone)	37,539	2,635	10.03	13

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TABLE B.

TYPHOID FEVER—FORTNIGHTLY SUMMARIES OF NOTIFICATIONS, 1898–1904, INCLUSIVE, IN METROPOLIS, COUNTRY, AND NEW SOUTH WALES, AS A WHOLE.

	1898.			1899.			1900.			1901.			1902.			1903.			1904.		
	Metropolis.	Country.	N.S.W.	Metropolis.	Country.	N.S.W.	Metropolis.	Country.	N.S.W.	Metropolis.	Country.	N.S.W.	Metropolis.	Country.	N.S.W.	Metropolis.	Country.	N.S.W.	Metropolis.	Country.	N.S.W.
January 14	53	99	152	36	49	85	42	51	93	26	67	93	31	84	115	32	145	177	48	112	160
" 28	42	130	172	30	75	105	92	98	190	29	99	128	41	94	135	42	178	220	44	125	169
February 11	37	144	181	54	92	146	105	137	242	45	136	181	40	119	159	30	228	258	72	160	232
" 25	49	203	252	38	142	180	94	179	273	67	115	182	45	113	158	60	427	487	57	171	228
March 11	55	232	287	42	153	195	84	194	278	39	114	153	46	117	163	47	339	386	38	121	159
" 25	36	173	209	77	174	251	73	239	312	49	148	197	47	125	172	35	344	379	55	92	147
April 8	43	211	254	57	181	238	78	237	315	61	113	174	46	122	168	45	308	353	49	77	126
" 22	51	269	320	37	161	198	52	192	244	73	133	206	39	120	159	44	277	321	36	80	116
May 6	47	200	247	50	142	192	52	186	238	40	138	178	25	83	108	55	364	419	25	52	77
" 20	59	132	191	41	155	196	32	120	152	56	112	168	16	82	98	51	270	321	32	59	91
June 3	46	109	155	39	120	159	25	109	134	28	82	110	20	102	122	51	277	328	26	52	78
" 17	17	43	60	27	81	108	14	63	77	21	56	77	21	74	95	26	115	141	21	54	75
July 1	18	40	58	32	50	82	16	41	57	20	43	63	15	46	61	26	79	105	13	24	37
" 15	16	36	52	30	31	61	18	41	59	18	27	45	12	25	37	31	57	88	13	14	27
" 29	15	20	33	14	24	38	18	18	36	12	15	27	9	23	32	19	27	46	12	8	20
August 12	8	13	21	14	18	32	7	12	19	9	16	25	6	21	27	13	20	33	7	12	19
" 26	7	13	21	16	19	35	10	14	24	4	12	16	5	17	22	8	25	33	10	5	15
September 9	7	12	19	11	18	29	5	9	14	7	4	11	11	13	24	15	16	31	14	8	22
" 23	8	17	25	10	22	32	5	8	13	15	6	21	11	20	31	8	15	23	9	8	17
October 7	4	17	21	12	14	26	—	12	12	8	6	14	16	20	36	4	17	21	9	26	35
" 21	11	23	34	4	23	27	10	13	23	8	16	24	6	20	26	13	19	32	10	20	30
November 4	23	22	45	6	32	38	14	29	43	14	32	46	10	28	38	29	23	52	9	23	32
" 18	12	59	71	12	42	54	14	27	41	18	49	67	17	31	48	30	34	64	31	46	77
December 2	39	66	105	21	36	57	41	68	109	29	64	93	20	68	88	42	62	104	30	36	66
" 16	49	88	137	15	43	58	67	89	156	55	90	145	26	107	133	27	112	139	44	78	122
" 31	72	107	179	61	100	161	77	183	260	82	176	258	105	227	332	50	244	294	69	118	187
Total for year	824	2,478	3,302	786	1,997	2,783	1,045	2,369	3,414	833	1,869	2,702	686	1,901	2,587	833	4,022	4,855	783	1,581	2,364

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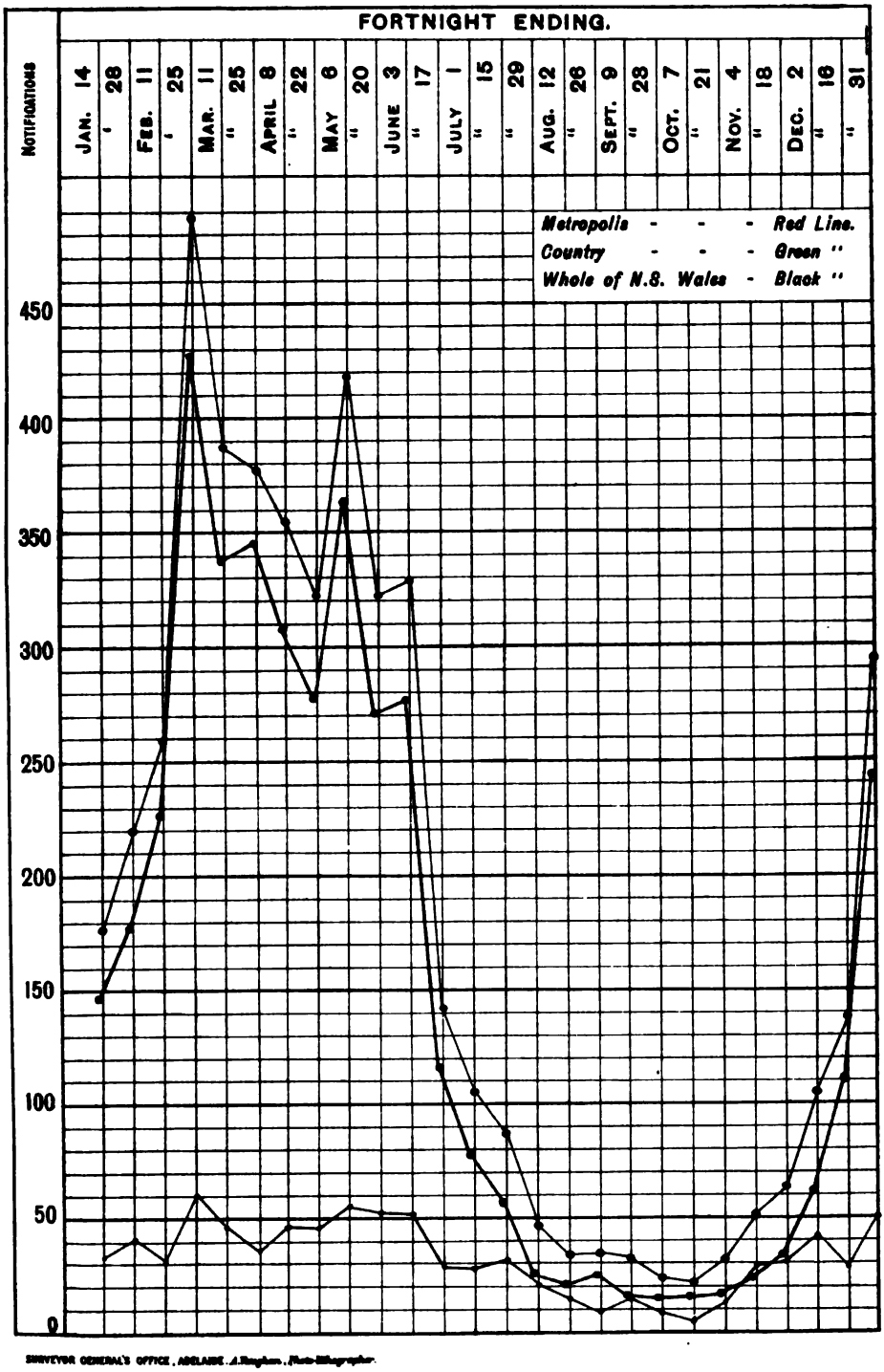
Typhoid Fever Notifications received at Notification Bureau during each fortnight, for Metropolis, Country, and whole of N. S. Wales.



SURVEYOR GENERAL'S OFFICE, ADELAIDE. J. Bingham, Photo-Lithographer.

R. J. MILLARD.
30/8/06.

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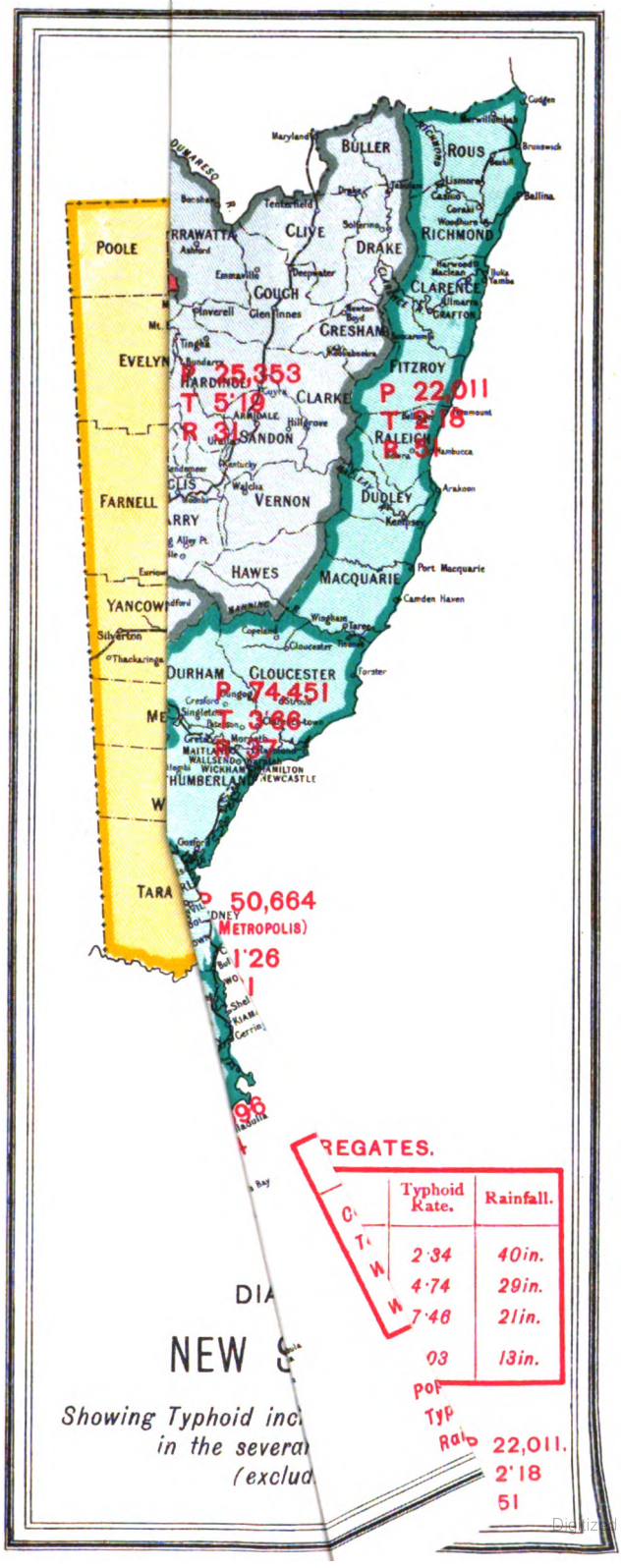
—1904.



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SUMMER DIARRHŒA IN INFANTS: FROM THE PUBLIC HEALTH POINT OF VIEW.

By W. F. Litchfield, M.B.

To get a proper idea of the subject of Summer Diarrhœa in infants, it is necessary to take a glance at infantile mortality. This is calculated as the number of deaths in children under 1 year of age to every 100, or 1,000, births during the same period. The mortality of infants varies greatly in different countries. I may mention the following:—

New Zealand (10 years ending 1900)	8.3
Norway and Sweden	10.0
New South Wales and Victoria (10 years ending 1900)	11.2
Sydney " "	12.3
England and Wales (10 years)	15.0
London "	15.0
Manchester "	20.0
Berlin "	30.0

Thus it will be seen that Australia stands in a favorable position in this respect. Norway and Sweden is quoted by European authorities as having the lowest infantile mortality of any country. That of New Zealand, however, is lower; and, in 1904, Sydney, with its half million inhabitants, showed an infant mortality of under 10 per cent., to wit, 9.8 per cent. I notice that certain European authorities* speak of 10 per cent. as being a normal standard for infantile mortality. What they mean is that under present social conditions it is not likely to be less, but that an attempt should be made to bring it down to that level.

My own personal experience in Sydney led me, some time ago, to believe that acute diarrhœa directly or indirectly played by far the most important part in infantile mortality. Of 173 consecutive deaths under 1 year amongst my out-patients, 62 per cent. were due to enteritis, 16 per cent. to congenital syphilis, and 20 per cent. to other diseases, including congenital defect. The same was noticeable with regard to the case incidence: nearly all the bad cases could be traced to an accute attack of enteritis. A study of the general statistics confirms that view. I find that 50 per cent. of all deaths under 1 year in Sydney may fairly be attributed to diarrhœal disease. About 30 per cent. may be grouped under the heading of congenital defect and congenital disease, including congenital syphilis, while the remaining 20 per cent. is made up of a host of diseases, of which the respiratory take the chief place. Under diarrhœal disease I have included diarrhœa, enteritis, cholera infantum, dentition, tabes mesenterica, and atrophy: my own clinical experience is that they should be so included. Moreover, the seasonal incidence of these deaths corresponds in the main with deaths from diarrhœa. When you subtract the deaths due to congenital defect and congenital disease, which may fairly be called unpreventable, you have five-sevenths of all other deaths directly due to diarrhœa.

I find that during 1904 (a very favorable year as regards diarrhœal mortality), in Sydney alone, 626 children under 1 year died from this disease; and when you remember that there are a large number of children over 1 year, and a goodly number of adults, who succumb annually to the same disorder, you will perceive at once that it is by far the most deadly of Australian diseases. It causes many more deaths each year than does consumption, which has always been supposed to hold the pride of place as a death-dealer.

* M. Budin, of Paris, and the Ed. *B.M.J.*

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I have been able to calculate corresponding figures for England and Wales* to those just given. They are :—

Diarrhœa	56	per 1,000
Congenital diseases	26	" "
Respiratory diseases	30	" "
Other diseases	39	" "

(See diagram.)

It will be noticed that, in relation to the total deaths, diarrhœa is more important in this country, while both absolutely and relatively respiratory disease is less fatal here than in England.

The disease is closely associated with summer weather conditions. Ballard made the important discovery that the mortality corresponded with a rise in the temperature, not of the air but of the ground, as tested by the four-foot thermometer. In England the disease is almost solely confined to the third quarter of the year. In this country the mortality begins to rise in October; it is most severe in November, December, and January, and then lessens month by month till September is reached, when it is lowest (see diagram).

Next, the evidence when carefully considered points to the disease being an acute specific infection. The seasonal incidence of the disease, its epidemicity, sudden onset, definite course, its pathological anatomy, and its behaviour in institutions, make that clear to my mind. Lately there has accumulated a good deal of evidence to show that the dysenteric bacillus of Shiga is an important, if not the sole, immediate cause of its production. This is a very important addition to our knowledge on the subject, and we owe it to American bacteriologists. Duval and Basset, Martha Walstein, and others have recorded series of cases of Summer Diarrhœa, from which the dysentery bacillus has been isolated. The most complete series was recorded by La Fetra and Howland. They found the bacillus in sixty-two out of sixty-four consecutive cases of Summer Diarrhœa, some of which were very mild. In conjunction with Dr. Hepsley, I found this bacillus in the stools of several cases of children suffering from diarrhœa in Sydney.† In a further number we established a specific agglutination re-action with the bacillus: moreover, we were unable to make any clinical distinction between those cases from which we obtained the bacillus and re-actions from the general run of other cases, and in every *post-mortem* examination made by us an inflammation of the large bowel was present. However, if it be thoroughly grasped that Summer Diarrhœa is an infection, then much of the confusion that exists with regard to it. and infantile mortality in general, will disappear.

The idea that it is caused by ptomaines is not supported by facts. The characteristic of ptomaine poisoning is the grouping of a number of cases around a certain food supply. Nothing of this is seen in Summer Diarrhœa. Moreover, the chief anatomical lesions are in the lower parts of the bowel, whereas they should be in the upper part of the bowel if ptomaines were the cause of the disease.

The part played by so called mal-feeding will be considered presently.

The next important thing to notice is that the mortality occurs chiefly in hand-fed infants. This looks at first sight as though the disease were a food infection. A careful consideration of the facts, however, seems to me to make it clear that that is not so. In the first place, infants fed exclusively on the breast do not altogether escape the disease. Richards,‡ in two series found that 14

* Culled from a table in article by G F. McCleary, in *B.M.J.* of August 13th, 1904.

† *A.M.G.*, January, 1899.

‡ H. M. Richards, *Journal of Hygiene*, July, 1903. This is an extremely important article (the factors which determine the local incidence of fatal diarrhœa).

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per cent. and 11 per cent., respectively, of all deaths from diarrhœa were in infants fed solely by the breast. Holt, in a large number of deaths from diarrhœa, mentions 3 per cent. as being exclusively breast-fed. The case incidence, moreover, is much higher than the mortality incidence, because the prognosis in breast-fed children is better than in hand-fed infants, when the disease is contracted. In my own series there were 5 per cent. of purely breast-fed infants among the deaths; and I can say that a considerable number of infants who get no food but the breast contract enteritis every summer. Out of 224 cases recorded by me 44 were purely breast-fed.

Secondly, fatal diarrhœa is not uncommon in infants fed on foods that may be considered unimpeachable. I have seen this over and over again, in institutions and in private practice.

Thirdly, about one-half of the deaths occur in children who are being fed on one or other of the patent foods of which condensed milk is the chief. These foods are sterile. Richards gives the following figures:—In one series of 183 deaths, 33 per cent. were in infants on condensed milk. In another series of 191 deaths, 44 per cent. were in those fed on the same food. In my series of 242 cases (not deaths), only 59 were on cows' milk, 44 were on the breast, 47 on the breast and foods, 73 on condensed milk, and 17 on other foods.

Fourthly, sterilisation, in the shape of boiling the milk, or by the use of sterile foods, has been more or less universally adopted of late years, and yet the infantile mortality from diarrhœa has not correspondingly decreased. It is not possible, of course, to say that it is never communicated by means of food, any more than it is to say that scarlet fever, diphtheria, or typhoid fever is not communicated at times by that means; but I am convinced that food is not the vehicle of the germ in the majority of the cases.

The relation of mal-feeding in general to Summer Diarrhœa needs mention. It is customary for a general charge to be levelled against mal-feeding that it is the cause of the diarrhœal mortality. But if what has been said above about diarrhœa being an infection, and that that infection does not come through the food, is correct, it is obvious that mal-feeding *per se* is not the cause of that mortality. I must say that within my own experience it is very rare to see a death that could be put down to mal-feeding pure and simple. There is always some disease, such as syphilis or an acute enteritis, at the back of the trouble. One certainly sees a number of weakly infants of the illegitimate class that have not had diarrhœa; but here a general neglect is as much or more to blame than the food, and, as I have said, they do not die unless something more serious happens. Moreover, one does not find in the mortality tables a place for mal-feeding. If mal-feeding were a serious factor in itself for diarrhœa mortality, one would not expect to find an absence of the latter in two such warm months as September and October; yet such is the fact. What is the mortality among hand-fed infants? I doubt whether it is higher than the mortality among illegitimate infants; that is, 28 per cent. for Sydney. The mortality of infants boarded-out under the Infants Protection Act, and under the State Children's Relief Act, in Sydney, is put by Mr. Coghlan at 40 per cent. These infants constitute the least favorable class in the community, and the death-rate for hand-fed infants generally must be lower. My point is that it is not the mal-feeding that kills, but an acute specific infection; this will be clearer directly.

The fact remains, however, that breast children are largely immune to diarrhœa, and maternal nursing is the only sure prophylactic against that disease. My explanation of this fact is that the child receives from the mother protective substances, technically known as immune bodies, which prevent the disease from developing. There is no direct proof of this; and

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I arrive at it partly by a process of exclusion. It is known, however, that such substances in the case of other diseases are transmitted through the milk, and we have the circumstance that adults are resistant to the enteritis of infants. It may be argued that hand-feeding predisposes to a diarrhœal infection, by lowering the bodily resistance of the child. This may be so; but there is no evidence to show that it is an important factor. Richards, in his article, decides against it; and it is a common experience that healthy children, as well as poorly nourished ones, go down every summer to the disease in question. How, then, is the infection in Summer Diarrhœa conveyed? Well, there are lots of ways that an infection may be spread; but, in the case of the enteritis of young children, there is a good deal of evidence to show that it is spread through the air by means of dust. M. Richards concludes that the infection is closely associated with organic matter in the soil, and is spread by means of the dust. He looks to road sanitation as the most important element in prophylaxis.

The following facts support the dust theory:—(1) In wet seasons the mortality from diarrhœa is low. (2) In the suburbs of Manly and Hunter's Hill, which are almost surrounded by water, and which are therefore comparatively free of dust, the mortality from infant diarrhœa is almost nil. (3) At Glebe Point, Sydney, the general wards of the Children's Hospital are on one side of the main street, and the diphtheria cottage is on the other. Every year, in the general wards, sporadic cases of severe diarrhœa occur, while in the diphtheria cottage—during about ten years—no case has been observed. The only difference in the situation of the two places is that the diphtheria cottage stands well back from the road, and is screened by a number of trees and a well-grassed paddock; while the general hospital stands almost flush with the footpath.

Newsholm made an important observation with regard to infant mortality. He found that in overcrowded areas the mortality was not much increased unless there was overcrowding in individual houses; in other words, the number of houses did not matter greatly, but the number of individuals in each house did. This accords with opinions expressed by myself in an article on "Foundling Hospitals" (*A.M.G.*).* In that article I showed that infantile mortality was always enormous in institutions where a number of hand-fed babies were housed together. Some years ago I had an experience of a babies' home in a thickly populated position of Sydney. During the winter we got on fairly well, but during the summer bowel disorder was constant and death very frequent. In spite of careful sterilisation of foods and general cleanliness, wave after wave of diarrhœa used to go through the wards. Even the infants fed at the breast did not altogether escape. It was at that time that the importance of an ærial infection in diarrhœa was first impressed upon me.

We come now to the consideration of preventive measures. Breast-feeding stands easily first in prophylactic measures. The mortality from diarrhœa in breast-fed infants must be very low. Richards found it to be, for Croydon, England, five per 1,000 infants so fed. Partial breast-feeding is better than whole hand-feeding. Unfortunately, however, under our present social conditions a considerable number of mothers cannot suckle their infants. Still I think, if it is thoroughly understood that when an infant is taken off the breast its chances of living are considerably lessened, and that it is one particular disease that is therefore likely to kill it, then some greater effort will be made in individual cases to persevere with maternal nursing. If my theory that human milk contains immune bodies is correct,

* *A.M.G.*, January, 1899.

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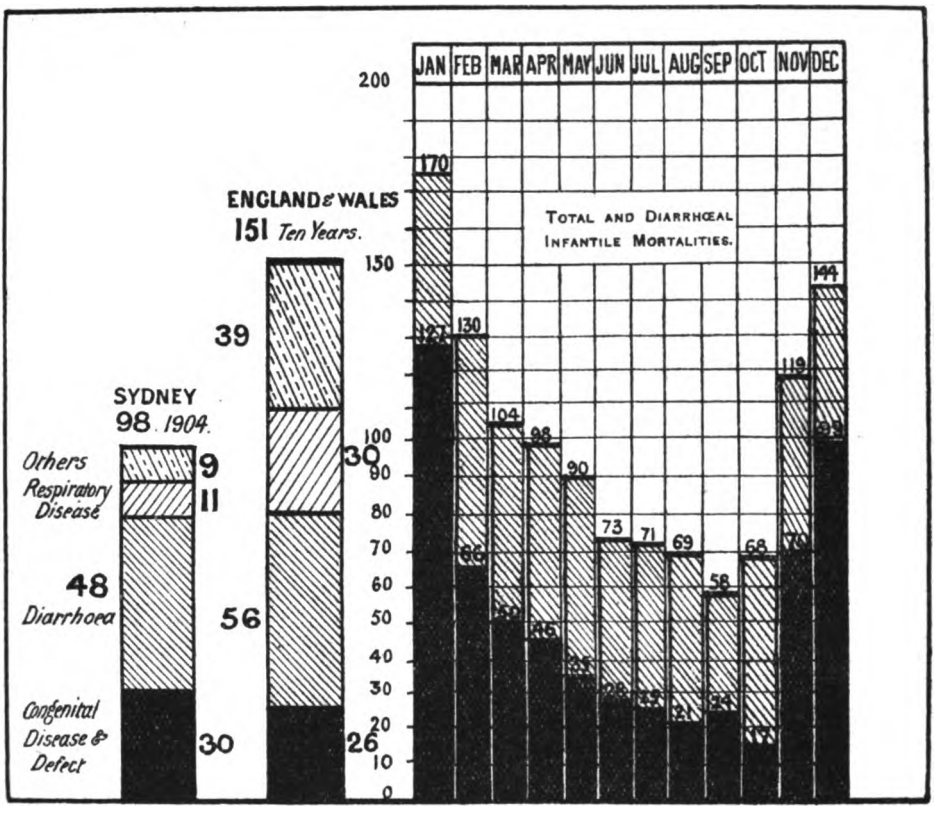
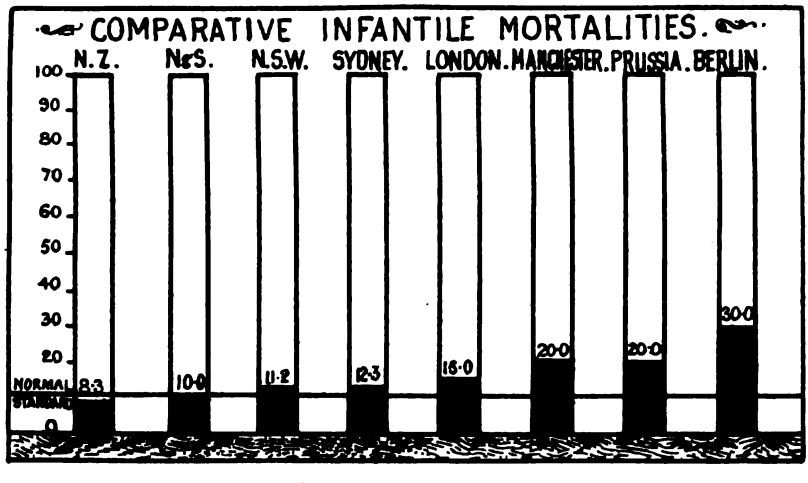


SHOWING DEATHS PER 1,000 BIRTHS IN DISEASE GROUPS. INFANTILE.

SHOWING TOTAL AND DIARRHŒAL MORTALITIES PER MONTH. THE BLACK COLUMNS REPRESENT DEATHS FROM DIARRHŒA. THIS IS FOR SYDNEY — DURING 1904.



SURVEYOR GENERAL'S OFFICE, ADELAIDE.

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it affords a powerful argument with which to impress mothers. In this country the State has a large number of illegitimate children placed in its hands. The authorities should be well seized of the importance of keeping mother and child together during the first year of life.

Next, where hand-feeding is resorted to not more than one child should be reared under the one roof. One fault of the New South Wales Infants' Protection Act is that it permits a foster-mother to take up to three infants to look after. This is a serious blot, for reasons given above.

Next, in all cities a serious effort should be made to keep down the dust. I understand that there is a science in road-making. Well, roads should be so constructed as to yield a minimum of dust. The streets should be well watered. This especially applies to the summer months, for it is then that the disease is most prevalent. I think that tree-planting in the streets would have some effect in keeping down the dust. Houses well back from the road, and screened by trees and shrubs, might well be encouraged. Infants, too, should have a better chance in places away from the main thoroughfares. Then surely some discretion should be used as to the time of day when, and the place whereto, children are taken out for their daily walk. If a choice of residence is possible, some foliaceous waterside suburb, away form the main thoroughfares, ought to be favorable.

There can be little doubt that babies born at the end of the summer, say about February, are more favorably situated than those born at later periods. It would be interesting to have statistics showing the mortality of infants born in the different months. However, I do not suppose that we are ever likely to have a "baby season."

The advisability of cleanliness of person, food, and surroundings I need scarcely emphasize. The extension and perfection of the sewerage system, by keeping down the organic content of the soil, and by washing away diarrhœal discharges, must be of service as a preventive measure.

Such, then, are my views on the important question of Summer Diarrhœa in infants. The main point that I have tried to bring out is that it is an acute specific infection, and the present general tendency to put all the blame on the food is wrong. I know that in this I have run counter to those who have been blaming the milk supply for the excessive mortality in infants, but I think that a calm view of all the facts will convince the majority that milk has been unjustly blamed in the past.

TWENTY YEARS OF SANITARY PROGRESS IN MELBOURNE.

By James Jamieson, M.D.

The completion of twenty years of service as a health officer in a large Australian city may serve as my excuse for bringing before this Congress a short record of what has been attempted, and, in part, accomplished.

A comparison of different parts of the period is the easier, because the administration, with certain improvements, has been the same and the statistical methods similar. On the whole, indeed, the conditions have been very uniform, unless in one very important particular. The population of the city of Melbourne has not varied greatly during the period, and there has been only a trifling increase in area. In the year 1891 there was an improvement in the water supply, in respect both of quantity and quality, new sources being tapped; but no filtration method has at any time been introduced, otherwise than privately. The great change has been in the adoption of a system of

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underground sewerage, bringing with it not only a drying and cleansing both of surface and subsoil, but also the almost complete abolition, in sewered areas, of the old and abominable pan system of nightsoil collection. Work was actually begun in 1897, and since that time has been steadily continued, so that it is now approaching completion, not only in the city but in most of the more central municipalities by which it is surrounded.

The city of Melbourne, with its population of rather less than 70,000, has the disadvantage of being thus surrounded, on all sides, by other independent municipalities, though it has compensation in possessing, in and close about it, a large area of public parks and gardens.

The staff most directly concerned in sanitary administration has been increased of late years, and may be described as fairly adequate. It consists of an Inspector of Dairies, Factories, and Lodging-houses; a Senior Inspector of Nuisances, with four assistants; and a female inspector (whose time is chiefly taken up with exercising supervision over cases of infectious diseases).

The meat supply is now carefully regulated; all carcasses, whether slaughtered in or brought into the district, must be branded, and special officers not only inspect the stock at the markets, but visit the butchers' shops at short regular intervals.

The milk supply is guarded in a double way. A qualified veterinary surgeon makes quarterly inspections of all cows kept in the city, or depastured in the parks adjacent; and the quality of the milk sold is frequently tested chemically by the City Analyst. On two occasions, also, a large number of samples were examined bacteriologically at the Melbourne University Laboratory, to which the City Council makes an annual grant. On the whole, it may be said, I think, that the food supply in all its kinds is fairly well guarded.

The important thing, however, is to discover what have been the results of the work, so far as shown by mortality returns. Dividing the twenty-years period, 1885–1904, into four divisions, it appears that the general rate of mortality, per 1,000 of population, fell successively in the following way on the average of the quinquennial periods:—1885-89, 19.48 per 1,000; 1890-94, 17.09 per 1,000; 1895-99, 14.65 per 1,000; 1900-04, 14.06 per 1,000.

The latest returns are the most favorable:—1902, 15 per 1,000; 1903, 13.8 per 1,000; 1904, 12.82 per 1,000.

While it may rightly be claimed that these results are satisfactory, it must also be admitted that most cities in Europe and America, as well as in Australia, show an improvement in their death rates, more or less closely resembling that here shown. It is only by examining certain details that the gain can be shown to have been greatly in the prevention of the diseases usually spoken of as preventable.

The infective diseases more strictly epidemic, as measles, scarlet fever, and influenza, vary so greatly at different periods, from causes so little known, that they cannot supply materials for a useful comparison. The prevalence of typhoid, on the other hand, is everywhere taken as providing a fair test of the sanitary condition of a town or district.

The number of deaths in quinquennial periods from typhoid has been:—1885-89, 223; 1890-94, 140; 1895-99, 103; 1900-04, 44.

These figures are sufficiently eloquent, if mere comparison of periods is made, though the population has fallen a little between the first and the last. The rate per 100,000 allows of comparison with other places. Things were at their worst in 1889, when the rate was 93.5, and at their best in 1903-4, when it was only 8.1. Taking London as the standard, a comparison shows that while, in 1889, typhoid was about five and a half times more fatal in Melbourne, in 1903-4 the rates were very nearly equal; that of the other great English towns averaging about 50 per cent. higher.

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A variety of causes have, doubtless, contributed to the bringing about of this very remarkable improvement; but the main credit must be given to the introduction of the drainage system. In some German cities, notably Munich and Berlin, where a drainage system was also of late introduction, a similar result was got. Confirmation of this opinion is also found in the circumstances that, in the suburban municipalities, as a whole—in many of which the old methods of drainage and nightsoil removal are still obligatory, while the works are incomplete—the typhoid rate in 1903-4 was almost twice as great as in the city. In view of that fact we may hope that the limit of benefit has not even yet been fully got.

Though diphtheria is very distinctly a disease spreading mainly by personal contact, it is often taken as also supplying some test of the sanitary condition of a district. Taking the four quinquennial periods, as before, the deaths from diphtheria and croup numbered, in 1885-89, 131; 1890-94, 86; 1895-99, 53; 1900-04, 29.

It is true, of course, that diphtheria is a disease which has always shown great periodical fluctuations of prevalence, but the reduction of fatality has been so great and continuous that some other than meteorological and other general conditions must be assumed to have been operative. The benefit got has probably resulted largely from the system of notification, with consequent exclusion from schools, not only of children actually affected, but of other children from the house. Supervision of cases, with cleansing and disinfection of houses after recovery, has doubtless also had a share in the result.

The subject of infant mortality is one which, for many reasons, is at present receiving great attention. In a leading article in the *Lancet* of May 20th, 1905, a very strong statement on the subject is made:—"It is a strange satire upon preventive medicine to have to admit that, although our general death rate has undergone, and is still undergoing, a steady decline, our infantile mortality should show not only no substantial fall, but even in some instances a substantial rise." The following figures show that, however true this opinion may be as respects the English towns and cities, it does not correctly represent the condition in Melbourne. Taking again the periods of five years, and the average rate of mortality per 1,000 births under 1 year of age, this is the result:—1885-89, 194; 1890-94, 152; 1895-99, 156; 1900-04, 110.

In the two years, 1903 and 1904, the rates were respectively 105 and 83.4. In ordinary years the infant mortality rate is, in the main, co-incident with the mortality from the diarrhœal diseases, and, in fact, is essentially a question of feeding. It varies with season and atmospheric temperature, and the influence of these is chiefly by way of causing easy contamination or deterioration of food, and, above all, of milk. High temperature may be taken as acting chiefly in two ways: by increasing the amount of dust, carrying germs of various kinds, and by favoring germ-growth in milk, however contaminated. The deleteriousness of dust will depend on the amount of organic or living material which it carries, and that again on the comparative cleanliness or foulness of soil and surface adjacent to houses. It is also apparent that the keeping quality of milk will depend on the cleanliness and care with which it is collected, conveyed, and distributed. And there is the rather incalculable and unmanageable element of ignorance and carelessness on the part of mothers and nurses. The problem of prevention is clearly very difficult and complicated, and one which may be approached in many ways. Education on matters of general hygiene may do much, but, unfortunately, is slow in operation; but municipal action can do a great deal, though even that may be slow of effect.

Melbourne, in some respects, is not in a favorable position for attacking the problem. It is a dusty town, with a high summer temperature. It seems

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hardly possible to allay the dust nuisance; but the dust may be rendered less harmful, as a food contaminant, by good drainage and careful street and lane cleansing. The improvement of our infant death rate is probably, therefore, due in part to the steady improvement in these respects, which certainly has been going on.

Safe-guarding the milk supply may be taken as the most important measure for the lessening of infant mortality; and for several years I have been in the habit of ascribing to measures of this kind the reduction in our diarrhœal and infant mortality rates. Quite a large proportion of the work performed by the City Analyst has been the examination of samples of milk. Whenever, in his opinion, there is clear evidence of adulteration, prosecution is instituted, and most of the benches of magistrates recognise the importance of the charge by fining severely. The result has been seen in the returns, which show a steady increase in the percentage of those milks which pass the "limit," and even pass the "standard." In 1904, further, out of eighty-three samples examined, only two were reported as containing boracic acid, or other preservative.

Bacteriological examination has confirmed what was shown by chemical examination. In the three years, 1900-02, ninety samples of milk were tested, by inoculation, for the detection of the tubercle bacillus, and five gave a positive result, though, in two of the guinea pigs used, only local lesions were produced. Of the ninety, fifty-four were tested in 1902, with a positive result in two only. In 1904 samples to the number of thirty were again tested, with a negative result in all. In the report for 1904 the following further statement was made:—"Incidentally, another important fact, bearing on the purity of the milk supply, has been brought out as a result of these examinations, *i.e.*, on previous occasions numbers of guinea pigs have invariably succumbed to peritonitis (septicæmia) within a few days of the injections. No such deaths occurred in the present instance; an observation which more or less justifies the following deductions:—(*a*) Absence of excessive numbers of bacteria, indicating general cleanliness in milking, &c.; (*b*) Absence of inflammatory condition of the udders."

I do not feel, therefore, that I can echo the pessimistic utterance of the *Lancet* (June 17th, 1905):—"We should be wrong probably in saying that there was not a pint of milk sold in London exactly as it comes from the cow, but we should not be far out if we said that at least—and probably more than—nine-tenths of the supply is not in its perfectly pure natural condition."

When it is seen that the infant mortality of London in 1904 was 146, and of the chief English towns 166, there is evident reason for statements of the kind quoted. There is, perhaps, reason also for the establishment of municipal milk depots, a step of not indisputed wisdom which has been taken in some English towns and cities. I have not felt called on to propose the adoption of any such measure, which does not seem urgently necessary, in view of the facts and figures adduced. It may happen that the low rate of mortality of 1904 will not be sustained, as the summer of that year was exceptionally cool and wet—though certainly not so cool and not wetter than that of England. But, the standard having been set, it will be incumbent on us to strive to keep up to it.

No field of sanitary activity has excited more attention, of late years, than that of the prevention of tuberculosis. Everywhere over the civilised world the "crusade against consumption" has been taken up; and, so far as evidence is yet obtainable, with a fair measure of success. If it has not been greater, the explanation may be found in the fact that the disease is, on the average, of rather long duration, and that the time is short since strict or systematic measures began to be taken.

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It may be proper to show, first, whether there has been actual evidence of diminished prevalence and fatality in recent years; and, as figures are comparatively small, I have thought it better to compare the totals of successive quinquennial periods with the mortality rate per 10,000 of population: --

Mortality from Tuberculosis.

	Total Deaths.	Annual Rate per 10,000.
1890-94	1,021	28.2
1895-99	784	22.4
1900-04	711	21.4

There is a marked difference between the periods, and the contrast is even more pronounced when the first three and the last three years are compared :—

Mortality from Tuberculosis.

	Total Deaths.	Annual Rate per 10,000.
1890-92	665	30.2
1902-04	404	20.0

On either method of comparison a very manifest improvement is shown, the mortality rate having been reduced by quite one-third, in the fairly remote periods shown in the second table. The disappointing feature is the comparatively slight gain in the last of the three periods of the first table as compared with the second; the most satisfactory is that the last year, 1904, gives the lowest number of deaths, 116, and the lowest rate, 17.1.

That there has been substantial gain cannot be doubted; the question remains about the causes by which it has been produced. It must be confessed that no large share can be ascribed to the special precautionary measures which have been coming into use since the institution of the so-called "crusade against consumption." Rather more than five years ago serious endeavors to carry out these measures were begun in the city. In every instance of death reported as due to phthisis the house was visited, instructions given for cleansing and disinfection, and wherever necessary the room or rooms chiefly occupied were cleansed by washing down and stripping roof and walls, and renovating afresh. This is ordered to be done by the owner; but if fumigation is regarded as sufficient, the walls are rubbed down and fumigation by formalin done by the corporation officers. When articles of clothing or bedding could not be easily washed, an endeavor is made to have them burned. It was soon evident that such action, only or chiefly after death, did not properly meet the needs of the case; but, in the absence of any provision for notification, nothing seemed possible but to appeal to the hospital authorities for reports of cases admitted to the wards or attending as outpatients. There was no great response for a time, and it has only been since notification has been in force, even rather ineffectively, that cases in considerable number have become known. It is clear, therefore, that the great reduction between 1890 and 1899 could not have been due to the measures just described. I believe they were due mainly to increased attention to general sanitary precautions; better house construction; more regard to reasonable ventilation; and great improvement in the making and cleansing of streets and lanes, and the regular removal of household waste. A portion

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of credit must also be given to the effect of legislation, in regard to shops and factories, by the prevention of over-crowding and the insuring of proper lighting and ventilation.

But there was another form of action which, I believe, had considerable influence in many ways, and not least perhaps in the way of phthisis prevention. Even before I entered on duty there had been instituted a system of house inspection, taken up chiefly with the internal condition. When, from structural defect or bad repair, a house has fallen into a condition making it unfit for human occupation as a dwelling, it is so reported in a certificate signed by the Health Officer and Building Surveyor. The owner has about six weeks allowed to make repairs and improvements, which must again be certified as satisfactory by the same officers. If he fails to do so, or to get extension of time for reason given, the house is condemned, and must be vacated. This compels action of some definite kind, one direct result in many cases being that the house is pulled down. Special attention has always been given to signs of dampness on walls or under floors, the repairs demanded including means for its correction. In the nineteen years 1886-1904, no fewer than 2,006 houses were thus certified, and no fewer than 546 were pulled down without much delay. Nearly all the others were repaired, to the satisfaction of the officers, within about the time allowed. It thus happened that, all through the period, without special thought of phthisis prevention, action was taken on a large scale which must have been beneficial in that way.

The lesson is that anything of the right kind, in the direction of sanitary improvement, brings its reward, and leads to benefit in ways not only foreseen and intentional, but in others that were not expected—perhaps not intended.

THE ADMINISTRATION OF THE HEALTH ACT, 1898, IN ADELAIDE.

By T. Geo. Ellery, Esq.

I would like, in the first place, to compliment Dr. Jameson on his paper and the good results that have followed during his long tenure of office. I am conscious of being placed at a disadvantage in following so eminent a hygienist, and I cannot be expected to deal with a similar subject in the same high scientific manner; but, as I appear before you as an administrative unit who has only a plain tale to unfold, I feel assured of your attention.

In dealing briefly with my subject, I hope to initiate a discussion on the equipment necessary to ensure effective sanitation in Australasian cities.

I have used the term "Australasian cities," not so much on account of any inherent difference between these and the cities of other countries as to show that we have considered this question at least from the Australasian standpoint, and in the light of local experience. The fact that the cities of Australasia itself differ so much in many ways will complicate the arrival at general conclusions, but the co-operation in the discussion of representatives from these cities should lead to something definite being laid down in the direction indicated.

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By sanitary efficiency I mean reasonable practical results in all the branches of sanitary science embraced within the scope of the Health Acts in force in the various cities.

The Health Act of South Australia deals with the sanitation of premises and their surroundings, food supplies (chiefly meat and milk), and infectious diseases; and I propose to show what is being done, and the results, to some extent, of the work in Adelaide.

1. *Sanitation.*—Adelaide has a population of 39,240 (Census, 1901), with 10,498 tenements (houses, offices, shops, and hotels), within an area of 1,042 acres (exclusive of park lands, gardens, and squares). There is a staff of four inspectors, or one for every 10,000 of population or every 2,600 tenements. These inspectors, although not in possession of the Royal Sanitary Institute's diploma, on account of there being no facility for obtaining it in this city, are fully trained and competent men. Each has special duties assigned to him, and also takes part in the systematic house-to-house inspection of the city, in addition to doing a fair share of the clerical work attached to the service of notices. The special duties are allotted by the Medical Officer of Health as follows:—Butchers' premises and slaughter-houses, hide and skin marts, offensive trades premises, produce stores and salerooms, cowyards and dairies, public w.c. and urinal accommodation, livery and bait stables, marine stores, restaurants and bakehouses, are inspected and reported on monthly; public and private schools, common lodging-houses, and banana-ripening rooms, quarterly; boarding-houses, hotels, private hospitals, and maternity homes, yearly; markets, spitting on footpaths, and a general observation of smoke emissions also receive attention. In addition, one of the inspectors acts as disinfection officer at the final disinfection of houses, and another carries out duties chiefly in regard to obtaining samples of milk for analysis under the Food and Drugs Act. The results of these arrangements have been found to be satisfactory. As regards house-to-house inspection, it required three years to complete the first sanitary survey of Adelaide. This was partly owing to the survey being the first systematic inspection made, and, as a result, necessitated more work than otherwise would have been the case; but, with the staff at its present strength, it is not anticipated that at any time the ground will be covered more than once in two years. With so long an interval between systematic inspection of premises, minor nuisances recur and may pass unobserved for a time, but this is met to some extent by noting the places likely to give trouble and visiting them at frequent intervals. I may mention that the card system of recording the sanitary history of the houses is in operation.

It is difficult to state the result in terms, but I think the members of this Section will find, if they apply a personal test, that the general sanitary condition of the city is good.

During the past five years 417 houses have been condemned as unfit for human habitation, and 243 of these were pulled down, while the others were made habitable.

In all minor matters the sanitary inspectors sign and serve their own notices, and copies of them are registered in the Secretary's office. If any are not complied with in the specified time, the Health Officer returns the docketed copy to me with a minute as to what action he desires taken. If legal proceedings are required, the notice must be adopted by the Board of Health, and the necessary authority to prosecute be given.

Major matters—such as declaring premises unfit for human habitation, ordering old insanitary houses to be demolished, requiring premises to be partly rebuilt, requiring extensive paving and drainage works, &c.—are

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remitted direct to the Board of Health, who make the necessary orders, under seal, and the notices under the orders are signed by myself as Secretary; the same book records being kept as in the case of inspectors' notices.

Under section 76 of the Health Act, legal action may be taken by the inspectors, with the Secretary's authority, in regard to nuisances without serving any notices whatever.

The removal and disposal of house refuse are under the City Surveyor's department, and he is no more satisfied with the methods adopted than the Health Officer or myself. The refuse is conveyed in open carts to a "tip" on the park lands, where the evils inherent in all tips are minimised by covering up the refuse as soon as it is tipped, and by preventing rag and bone picking at the tip. I may add that the substitution of properly covered vans for the present carts is under consideration, and that a refuse destructor would have been in course of erection now but for influential local opposition to the site selected. As I have strong views on these matters, I would like to hear Dr. Jameson's opinion of the operation of refuse destructors in Melbourne.

The deep drainage system is under the control of a Government department, and, so far as can be ascertained, no outbreak of disease has been associated with it. The drainage system has been established for over twenty years.

Factories are also under Government inspection.

2. *Food Supplies.*—Small cattle are slaughtered on butchers' premises in the city, large cattle at the city slaughter-house. This latter is an antiquated structure, and would have been replaced by a modern abattoir but for the reason which has delayed the erection of a refuse destructor. Inspection is consequently superficial.

Milk supplies are protected by combined action on the part of the city and suburban local boards, which ensures inspection of premises and examination of cows within the metropolitan area. A small combination, comprising four local boards, employ a veterinary inspector. A larger combination comprises the city and fifteen other local boards, who have appointed a veterinary surgeon with a special training in dairy science as Inspector of Cattle, who is under my control with Dr. Borthwick as Consultant Medical Officer. The periodic visitations of these inspectors is supplemented by the more frequent visits of the local sanitary inspectors. There are altogether 728 registered dairies, over 4,000 cows, and 25 milkshops within the area. Premises have been rendered much more sanitary, cows are better tended, and adulteration with water is less frequent than formerly; so that a better and purer milk has been the result of the combined action. The Inspector of Cattle has a fully equipped chemical and bacteriological laboratory on the Town Hall premises for the analysis of milk.

Water supplies are under the control of a Government department as far as the city is concerned, and I may say that no outbreak of disease has been traced to the water supply.

3. *Infectious Diseases.*—The diseases are specified by the Health Act. The usual restrictive sections of health Acts are operative, and compulsory removal to a hospital is permitted under certain conditions. This latter power it has so far been unnecessary to exercise, as no difficulty in this direction has been experienced. Power is given to erect isolation hospitals, but this has not been taken advantage of. At present the Adelaide and Children's Hospitals, as well as private hospitals, take in cases of typhoid fever; the Children's Hospital has made admirable provision for diphtheria in children; some of the private hospitals have limited isolation accommodation for

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infectious diseases generally; the Government finds provision on the Quarantine Station for exotic diseases, like plague and small-pox. The staff engaged in the administration of this part of the Act are a trained nurse with a special knowledge of the theory and practice of disinfection and a sanitary inspector who is told off for special duty when required. The apparatus at their disposal are formalin lamps (Alformant) and Equifex sprayers; the latter having almost completely displaced the former. A metal box is provided for the removal of infected articles, which cannot be otherwise disinfected, to be burned at an open grid. As soon as a case is reported the trained nurse visits the house, advises as to the best mode of isolating the patient in the house, and shows how to prepare and use disinfectant solutions. She also leaves (after explaining them to the person in charge) printed forms for guidance in maintaining effective isolation and disinfection during the progress of the case. Particulars, specified on a card, in regard to employment, milk supply, school attended, &c., are obtained by the nurse for information and guidance of the Health Officer. If the patient's family is poor, disinfectants are supplied gratis, and in that case bacteriological examinations can be also obtained free by the medical attendant. The trained nurse keeps in touch with the case throughout, and at its termination directs and oversees the disinfection of the contents of the room, leaving the latter ready for final disinfection by the inspector: this he accomplishes by spraying it thoroughly with formalin solution, and if anything requires to be destroyed by burning he also sees it done personally. Unfortunately, Adelaide does not possess a steam disinfector; but as this formed part of the plans for a destructor, its absence is similarly accounted for.

As to results, I shall place a few figures before you, supplied by the Health Officer. During the last five years, *i.e.*, since the Health Act came into operation, there have been notified 230 cases of typhoid fever, of apparently local origin. Of these 161, or 70 per cent., were removed to hospital (public or private), leaving 69 to be treated in their own homes. Secondary cases occurred in 6 of the 69 houses, or in 8.7 per cent. Of diphtheria, out of a total of 222 local cases, 138, or 62 per cent., were removed to hospital, and among the 84 isolated at home secondary cases occurred in 5 houses, or in 6 per cent. Of scarlet fever, out of a total of 365 local cases, 112, or 30 per cent., were removed to hospital, and among the 253 isolated at home secondary cases occurred in 28 houses, or in 11 per cent. In all these instances of secondary infections the houses were of the poorest sort, where effective isolation was impossible.

The following tables contrast average rates for the period of five years immediately preceding the passing of the Health Act, and the period of five years during which the Health Act has been in operation:—

	Per 1,000 Population.		Per 1,000 Births.
	Birth Rate.	Death Rate.	Infant Mortality.
First period	27.79	18.88	160
Second period	23.84	16.99	138
Last year of second period	22.35	15.77	117

It should be stated that, while correction has been made for deaths in the Adelaide Hospital, Destitute Asylum, and Lunatic Asylum, this has not been done in the case of the Children's Hospital, private hospitals, maternity homes, and private houses. This accounts for the apparently higher death

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rates compared with some other Australian cities. The same vitiation affects the next table. This want of uniformity in regard to statistical rates ought to engage the attention of this Section :—

	Mortality per 10,000 Population.			
	Typhoid.	Diphtheria.	Diarrhœa.	Phthisis.
First period	4.6	1.2	8.1	19.1
Second period	1.8	1.3	4.6	16.8
Last year of second period	0.7	0.7	3.0	15.8

I wish to emphasize the fact that the condition of water supply and drainage have been the same during these ten years and for a period antecedent sufficient to eliminate any effect on them. The diminution in the above death rates must thus largely be accounted for by more effective sanitary administration. During the first period no attention was given to infectious disease, and ordinary sanitary work was at a very low ebb. With the Health Act came a revival in the direction I have already indicated.

I venture to submit that the results obtained by the trained nurses' work in regard to isolation and disinfection have been satisfactory, and might raise the question whether it is necessary to erect expensive isolation hospitals to accommodate all, or nearly all, cases of infectious diseases. Without entering into a discussion on this matter, I may point out that experience in Adelaide shows that it is absolutely essential to have hospital accommodation sufficient to take in cases occurring in lodging-houses or in houses too small to afford satisfactory isolation, among dairy employés and others concerned in the manufacture or distribution of food, and among domestic servants.

I have attempted to describe the sanitary administration of Adelaide, with some of the results obtained under it. The appointment of a trained nurse to supervise cases of infectious diseases at their own homes is the special feature which I would urge other cities to imitate. Her work has been invaluable, and could in no sense be replaced by that of a male inspector; moreover, its educational value must tell indirectly. The other feature of administration which has been prominently successful is the combined action of the metropolitan boards of health in appointing a highly qualified veterinary surgeon to safeguard the milk supplies. So satisfactory has this action proved that it appeals to me as a solution of many problems in the sanitary administration of such areas, and I should like an expression of opinion on the results obtained by any similar combinations from health officers with experience of their working. I have also pointed out in what directions we fail in equipment. In my opinion Adelaide requires the addition of at least one more inspector to relieve the others of special duties. Adelaide is a city naturally favored in its situation and environment, and its staff of inspectors cannot be regarded as a standard for other cities less favorably placed. Adelaide also stands in need of an improved system of removing house refuse, a refuse destructor, an abattoir on modern lines, a steam disinfector with the necessary adjuncts, and a small hospital for the isolation of the cases I have mentioned. I take it we may safely affirm that other cities of Australasia require similar equipment, if not already provided. I trust that city health officers will give us the advantage of their experience generally, and so enable this Section to formulate some resolutions which will carry authority when presented to administrative boards,

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NOTES ON THE PUBLIC HEALTH LEGISLATION OF AUSTRALASIA.

By J. R. Baker, LL.B., Cantab.

It may be fairly said that the Legislation of Australia and New Zealand for the promotion and protection of Public Health, has not kept pace with the advances in the application of the principles of public hygiene. No doubt of recent years Act after Act has been placed on the Statute books of the different States, but none of them come up—although some of them approximate—to the ideal.

Speaking of the States now under review, the unavoidable mortality of any particular city differs little from that of any other city of about the same size, but it is in the avoidable causes that there is much room for improvement; and, to effect this improvement, recourse must be had, in the first instance, to the Legislature.

Now, sir, I purpose to approach the subject from the point of view of the layman who partially occupies his attention in administration, and not from the standpoint of the expert. Looking forward from the vantage ground of the expert there is only one unit really worthy of consideration—namely, the expert. From a layman's point of view, handicapped by his limitations as regards his special knowledge, it is apparent from the outset that there are three units, which are elementary, but which each demand attention in their respective spheres of action.

We have, first, the scientific unit—the expert—who is embodied in the highest degree in the medical officer especially versed in this particular branch, and in a lesser degree in his under-studies, the inspectors, of varying degrees of intelligence.

Second—the lay administrator, who is exemplified in central or local boards of health.

Third—the general public, for whose benefit and advantage sanitary laws operate.

These three units make up the whole world within which public health enactments have their place. We find in the vanguard, right in the front, the expert; always enthusiastic, though at times perhaps apparently unreasonable, and always impatient of the drag that is exercised on his actions by the other two parts that make up the whole.

We have next the lay administrator, anxious on the one hand, so far as his knowledge will allow him, to follow the expert, and dragging after him—slowly, perhaps—the third unit, the general public; apprehensive on the one hand of not being able to keep up with the leader, and on the other hand of getting ahead of his followers, at all times, however, a salutary check—or perhaps you would put it a necessary evil—on the impulsiveness of the expert, who is generally ready to break away from the main body and fight too far in front, running the danger of having his lines of communication cut. I claim, on behalf of the second unit, not only is he a necessary link in the chain, not only does he exercise a beneficial break on the impulses of the expert, but, without him and without his co-operation, and without the exercise of his more limited technical knowledge, the administration of public health matters would—at any rate in a democratic community—be practically impossible.

We have in the third place the third unit—the general public, the body that has to be operated upon; always for its good, but not always to its liking. This unit has to be persuaded, urged forward, sometimes coerced, sometimes perhaps cajoled into acquiescing in the forward movement of the expert and

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less speedy movement of the lay administrator. I have no doubt that you, who for the most part consist of experts, are somewhat impatient of the check exercised by the second unit, and I have little doubt that you all really think, although you may not state so in so many words, that you could very well do without this second unit, and the public health matters would benefit generally by its exclusion. You cannot expect me, of course, to acquiesce in this view; nor do I in fact believe it to be a correct one. In my opinion the ideal health legislation would be one that would best effect the synchronised working of all these three units, or at any rate would most approximate to such synchronisation; and it is having this in view, and having the operations of these three units in their various spheres in immediate consideration, that I propose to very shortly review the health legislation of the various States of Australasia and of New Zealand. For the purpose of comparison I deem it advisable to broadly state my ideas of the ideal, without, however, attempting detail.

The Ideal System.

1. A Commissioner of Health, under a Minister, with the necessary officers and inspectors. I say advisedly "necessary," as the expert is apt to overrun the constable in this respect.

The Commissioner would be, of course, an expert specially suited for the purpose—a man of science and a man of tact, not altogether an impossible combination. This quasi-autocrat should have especially the following powers:—

(*a*) Complete control of quarantine. This may become, in the near future, federal.

(*b*) Power to act in all cases of emergency on his own initiative, subject to a modicum of control of his Minister. These cases would be rare, *e.g.*, plague.

(*c*) Power to make by-laws for government of district boards and local boards, subject to the approval of the Government. So far as district boards are concerned, power not to be exercised except in case of default or inefficiency. Minister to be the arbitrator.

(*d*) Concurrent jurisdiction with district and local boards, in any place where neither exist, then exclusive jurisdiction.

(*e*) Power to recover expenses from boards in cases of non-compliance.

2. District or county boards for centres of population, consisting of delegates from associated local boards, with powers limited to—

(*a*) Protection of food supplies.

(*b*) Control of infectious diseases.

(*c*) Construction and control of abattoirs, and, in particular in this respect, an exclusive jurisdiction, *e.g.*, the prohibition of the sale of meat slaughtered elsewhere.

(*d*) The use of hospitals and sanatoria for the isolation of infectious and contagious diseases, with power of compulsory removal of infected persons where necessary.

(*e*) The cremation of refuse.

(*f*) Milk and dairy inspection and regulation.

(*g*) Revenue to be raised from constituent boards.

(*h*) By-laws. In case of dispute Minister to be arbitrator.

3. Local boards, being the present local authorities where they exist, to deal with the more elementary but no less important duties of health authorities.

In order to attain the above ideal the question of increased expenditure must be faced, but this should not fall altogether on the local bodies. Refuse

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destructors must be erected out of municipal funds, and will bring in little or no revenue. On the other hand, abattoirs, erected also by municipal funds, will pay for themselves. But, in my opinion, isolation hospitals and sanatoria should be erected and maintained out of the general revenue of the State.

Matters in connection with water supply and drainage are not included, as in the majority of the States they are in the hands of the Government.

Type I.—South Australia.

I take this State first, not because it is by any means the best—far from it—but because I am better acquainted with the subject-matter. We find—

1. Central Board of Health, with a medical chairman, which has the general control of health matters in the State. Its powers are concurrent with local boards, with a right to recover expenses in any cases in which it has to step in and compel local boards to carry out their duties. This board deals with quarantine and similar matters, and in case of any serious epidemic, such as plague, it is the only body which could deal with it promptly; but its machinery is inadequate. It might with advantage be abolished in favor of a Health Commissioner.

2. Local Boards of Health are constituted by every municipal and district council: they are charged with the administration of the whole of the health machinery within their respective areas. Their powers are full enough for all practical purposes. They fail, however, to satisfy in many respects the requirements of metropolitan areas, by reason of the independent action of each board within its own boundary, and the insufficient appliances for inspection of food supplies and control of infectious diseases. With the help of the Government the metropolitan boards have done what they can, having regard to their independent instead of combined jurisdiction in these matters; but they are not capable of much further expansion in the direction of efficiency, except on the lines of voluntary combination for specific purposes, which have proved to be unsatisfactory. I except from these remarks a voluntary combination in regard to inspection of dairies, which has turned out very well.

3. There is provision for a combination of any number of local boards to exercise all or any of the provisions of the Health Act—called a County Board. This has not "caught on" generally, although a partial suburban combine on a small scale exists, which, so far as I know, works satisfactorily.

We are a long way short of my ideal in this State.

Type I. may be taken as representative of Victoria and Western Australia, where, however, their legislative Acts are more comprehensive—and, let us hope, more effective.

Type II.—New South Wales.

1. There is the Board of Health, which supervises generally the carrying out of the Health Acts by local authorities, and, outside municipal areas, exclusively administers the Acts. It has concurrent jurisdiction with local boards. There is, I believe, no system of local government in New South Wales similar to that of other States, and consequently outside towns the Board of Health takes charge by means of police officers.

2. The municipal councils are local boards of health: they may combine to work disinfectors and establish abattoirs.

3. The Governor may, and has in at least two instances, proclaimed districts. The local authorities remain distinct, but the medical officer of health is appointed by the Governor for the whole of the district, and, by this means, to some extent concerted action is possible. No doubt this is a step in the right direction; but, taken altogether, Type II. does not correspond

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with my ideal in several important respects, more particularly in defective metropolitan combination and absence of administration by means of local bodies which do not exist in New South Wales.

TYPE III.—QUEENSLAND.

1. Here we have a Commissioner of Health acting under a Minister of the Crown, who is charged generally with the administration of the Health Act. There appears, also, to be in connection with the Commissioner a Central Board of Health, but it is merely advisory, and has no executive or administrative functions—in fact it appears to be a body whose duty it is to hold up the skirts of the Lord Chancellor. The Commissioner has the usual concurrent jurisdiction with local boards, and the necessary powers to enforce such jurisdiction.

2. Local boards within their own area administer health matters.

3. There appears also to have been a metropolitan joint board for the prevention of infectious diseases, which had a jurisdiction within a radius of twelve miles of Brisbane. This board, however, did not prove to be a success, and was abolished.

Speaking generally, the Queensland Act seems to place somewhat unrestricted powers in the hands of the Commissioner. Whether these produce friction or not would of course depend to a large extent on the person who administered them. The general scope of the Act recognises, for metropolitan areas, a jurisdiction too divided to coincide with my ideal.

The Tasmanian Act gives the Chief Health Officer under a Minister of Health very large powers. There is no Central Board of Health, and the Act is to some extent intermediate between this and the following type.

TYPE IV.—NEW ZEALAND.

1. We find in New Zealand no trace of the central board, or its equivalent, but we find in lieu thereof that the State is divided into several (at present six) health districts, by proclamation.

2. The Chief Officer of Health, as well as the district officers of health, are appointed by the Government. The latter administer the Acts in the several districts in conjunction with the local boards; their powers seem to a large extent to run together, the district health officer being enabled, in case of default, to carry out the provisions of the Act at the expense of the defaulting authority.

3. The local authorities are local boards of health in their several departments, and, in conjunction with the district officers, administer all health matters. I am given to understand that very little friction arises between the district health officer and the local authorities: this speaks well for the intelligence of both. There being so many towns of importance in New Zealand, it may be thought my ideal would be difficult of application; but I do not see any difficulties in the way of such application, and, though I must plead entire ignorance of local circumstances, there seems no reason why in any country metropolitan combines should not be in vogue and operate for the general benefit.

The ideal which I have set up aims at an improvement in the administration of the higher branches of sanitation. The chief fault in all existing legislation is that there is no discrimination between populous centres and country districts. More especially is there no serious attempt to deal with metropolitan areas, and other populous centres (which, after all, demand the greatest attention in every State), in a truly scientific manner; and it appears to me that the chief advance in future legislation must be in remedying these defects.

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SCHOOL HYGIENE.

By J. S. C. Elkington, M.D., D.P.H.

The enactment of compulsory education by the various Australian States, in common with other civilised peoples, has carried with it certain far-reaching consequences of profound importance to the race, and it is only within the past few years that these consequences have come to be recognised in their true light. It is not my intention here to deal with the social changes which, within the past twenty or thirty years, have arisen from the operation of compulsory education in its spiritual aspect, nor to attempt to forecast in detail the still more profound changes which appear to be inevitable in the near future. These are not by any means foreign to the subject of School Hygiene; but, in the time at my disposal, it is impossible to attempt to discuss anything more than elementary principles, and to incidentally allude to their application.

In adopting compulsory education the State practically reduces education to a State monopoly. Private schools are recognised co-equally, however, with State schools as qualifying children for conformance with the requirements of the Acts, and School Hygiene cannot be regarded as State school hygiene alone. The State responsibilities cover all classes of schools, however, and the State directly undertakes the care of the vast majority of all school children. Hence it behoves the State to lead the way in any system of educational reform, and particularly in that aspect which is now under discussion. Education is a biological process, whereby certain discrete growing organisms are in process of being fitted to their environment in order to render them more useful to that concrete organism, the State. The environment has a physical as well as a spiritual side, and nine-tenths of those children who undergo the process of fitting will depend for their success and happiness in life almost wholly upon the physical side—in other words, upon the integrity of their bodies and not upon the degree of culture of their minds. Some two-thirds of them, moreover, will be the parents of the next generation, and, in the nature of things, the stamina and *æquanimitas*—to use that portmanteau-word in which Professor Osler sums up, as did Pius Antoninus before him, so much of import to the race and to the individual—of that generation will largely depend upon the bodies of the children now in our schools. The greatest of the faults committed in the name of education in the past has been the non-recognition of this physical side, either as an all-controlling factor in the development of the spiritual side, or as an all-important influence upon the child's future. It is impossible to deny that this oversight has resulted in an incalculable waste of public and private money, and in a monstrous injustice done to thousands of Australian children. These are strong words, and I utter them with a due sense of their seriousness. School is not merely a preparation for life, but an important part of life itself, wherein body and mind are alike plastic, and capable of almost infinite development for good or ill. In the German Imperial Health Manual a great truth is summed up in a pithy sentence—"The sense of justice demands that, in a State where attendance at school is compulsory, the children shall be exposed as little as possible to dangers to health while in school." Far-reaching as is this principle, I submit, however, that a sense of justice demands more than this. It requires the practical recognition of the fact that, not only shall children be protected from the many physical risks induced by the entirely artificial process to which compulsory education submits them, but that they shall be practically and intelligently instructed in the few simple rules by which they may carry the process of protection on to their after-life.

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It requires, further, that justice shall be done to the taxpayer, in that the immense amount of money which he contributes for the purpose of compulsory education shall be expended to the best purpose; in that, by sensible instruction and care at school, there shall be saved a proportion of the further sum which he contributes for the treatment of unnecessary preventable disease, and in that large sums will not continue to be wasted in attempting to force symbolic information through eyes that cannot correctly see and ears that cannot properly hear, in order to reach brains somnolent and unresponsive through carbonic-acid poisoning.

It is scarcely necessary to remind this learned audience that the school hygienist is no longer to be regarded as a crier of trifles in a wilderness of fads. His methods and aims are based upon the common sense of everyday professional practice, and upon the elementary laws of physiological knowledge. The educationist and the hygienist must work hand in hand, and nobody recognises this more fully than does the educationist. Professor Nicholas Murray Butler has stated the question from an authoritative educational standpoint—"The most grievous single obstacle in the way of the spread of sound educational principles," says he, "is the popular view that the essentials of education are limited to instruction in reading, writing, and arithmetic. It might fairly be argued, and with no small force, that the possession of so much knowledge alone is a positive detriment to a human being; especially if that knowledge has been gained at the expense of physical and moral habits, which, in educational value, far outweigh any such meagre intellectual attainment . . . . . It is not too much to say that health, its provision and protection, is all-controlling in present day educational theory, although it is unfortunately far from being so in practice. The chief reason for this discrepancy between the ideal and the real is simple ignorance. Teachers and parents do not recognise that eyesight is being impaired, normal growth prevented, blood poisoned, and the body starved because the hours of school life are so often unhealthy and abnormal hours. . . . . School buildings are constantly erected with a view to external effect alone, and an adequate system of ventilation and a proper site are pronounced too costly. It is not true that a child is always and everywhere better off than running at large in any village or city. If the classroom is already overcrowded, if there are already too many pupils assigned to a teacher, then every additional pupil who is brought in injures those who are already there, and receives injury himself." These weighty words were uttered from a chair of Education. Could we, as reasonable beings, expect plainer speaking or more destructive criticism of the "three-R's-at-any-price system" from the most iconoclastic professor of Hygiene that ever laid sacrilegious hands upon a domestic or a national muck-heap?

The work of the educationist and of the hygienist are then complementary. The neglect of hygienic requirements in school life not only injures the most valuable assets possessed by the State, but it absolutely debars the most skilful and accomplished educationist from obtaining the best mental re-action, and hence wastes time and money. In turn, the educationist must awaken the sanitary conscience early in life before any real or lasting advancement can be made in national hygiene. I am not of those enthusiasts who predict that the schoolmaster will be the sanitarian of the future. The schoolmaster will never replace the sanitary administrator, but it is only through his—and particularly her—work and co-operation that we can hope to attain to that national physical morality wherein preventable disease shall appear in its true light as somebody's crime, and the preservation and protection of health shall be accepted as a sacred duty. This imples no ardent forecast of a sanitary millennium, in which all minds

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shall go attired in the chaste drab garb of hygienic seriousness, and the young man's fancy, before turning lightly to thoughts of love, will impel him to look up the family history of the beloved for traces of tuberculosis or alcoholic tendencies. It does, however, imply a hope that, from due recognition by the State of the vast economic importance of School Hygiene and of properly directed hygienic instruction, there will accrue, amongst other important results, a notable increase in the national common-sense, an intelligent appreciation of scientific medicine as distinguished from quackery, and a notable decrease in preventable disease in all its protean shapes and all its far-reaching consequences. The life of civilised mankind grows more and more artificial every year, and the standard for even moderate success rises higher and higher as man's busy brain overcomes the impossible of to-day and fits it into the daily round of to-morrow's work. The need for a sound well-cared-for body remains as great as in the days when the primal savage wrested a living from uncompromising nature. Children must, then, be taught to live; they must be protected against the early consequences of that civilisation which afterwards demands so high a standard of fitness, and which treads down so remorselessly the unfit. The most daring cannot reconcile the cruel demands of racial fitness for the physical extinction of the weak with the humanitarianism which inspires modern hygienic practice. We must not—cannot—obliterate the unfit, we cannot simplify the ever increasing demands of civilisation; hence we must strive, by all possible means, to prevent the originally fit from being rendered unfit, and to eliminate, as far as possible, the conditions which tend to accentuate existing unfitness. It is a common-sense business proposition after all—the fitter the population can be rendered in our schools, the better value do we get for our money. Conversely, every child who leaves school damaged in health by defective school conditions, every child who passes through school life hampered by practically removable causes which have prevented him from getting twenty shillings' worth of useful knowledge for each pound of taxes or fees spent upon him, and every child whose time has been wholly spent in being fitted to a comparatively useless spiritual side of an environment which is almost wholly physical, represents an unwarrantable waste of money, energy, and time—practically amounting to criminal negligence.

Destructive criticism is proverbially easy, and I need scarcely say that School Hygiene does not confine itself to pointing out defects. Practical and readily attainable measures for their removal or mitigation are essential. The object of School Hygiene in all its bearings is to increase and enhance educational results, and it must be severely subordinated to this object by the study of pedagogic requirements, and by due appreciation of the numerous difficulties—structural, administrative, and financial—which stand in the way of a perfect system. All hygienic defects may be classed under two main headings: defects of structure and apparatus, and defects of management. Each is complementary to the other: a defectively constructed school may hamper excellent management, and, on the other hand, ignorant or careless management may render nugatory the advantages of a well-planned and well-fitted building. In practice, however, defects of management are, for several reasons, more important than are defects of structure. A skilful teacher, versed in elementary hygienic principles and practice, can overcome or mitigate much structural wrong-doing; and no proposal to pull down and re-erect on proper lines every structurally defective school building, or part of a school building, can come within the scope of practical politics, however sapiently we may argue from the standpoint of national economy. Further, as has been already indicated, we have to depend largely upon the teacher for the intelligent and effective employment of any improvements which may

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be introduced into future schools. For my present purpose, State schools and State educational administration only will be considered, as affording at once the widest field and the most useful example; although it is necessary to avoid the fallacy of believing that private schools are free from defects, or require less attention. In my experience, defective as State school buildings have often been found to be, they are, *qua* hygienic structure and management, far superior, as a class, to private schools.

The training of teachers and inspectors in School Hygiene, hence, assumes the place of first importance; and it cannot, I think, be argued that the compulsory inclusion in the teacher's curriculum of such training would impose an unnecessary or unbearable burden upon these usually much-trained individuals. Certain pertinent arguments may be here suggested as showing that the question is one of vital interest to the teachers themselves. Where a superannuation fund exists, as in South Australia and Tasmania, wholly supported by State teachers, it is to the indubitable financial advantage of this class of individuals to eliminate or dilute as far as possible all causes which tend to disable or kill contributors, and hence to draw upon the fund. No improvement is likely to cripple the fund by turning teachers into a race of centenarians. The teacher is susceptible to bad hygienic conditions in schools —not to such a marked extent as are the children, perhaps, but nevertheless to a sufficient degree to produce serious damage to a body already weakened by any cause. This effect of insanitary school conditions upon teachers forms by itself an important question in School Hygiene, and one which should appeal strongly to the influential and numerous class which it affects. Further, under a system whereby a teacher's prospects in his profession are largely influenced by the educational results obtained by him, as ascertained by inspection, it is obviously only fair that physical obstacles to mental re-action—such, for instance, as adenoid growths, or myopia—should be assessed at their real value, and that the children who are thus defective should not be lumped in with their normal brethren. For this to be carried to its best conclusion, a system of medical inspection becomes necessary; but, failing this most desirable provision, a fair degree of practical and skilful training of teachers and inspectors would enable the majority of cases of this kind to be at least suspected and allowed for. I am aware that, by such exemption or separate consideration, some special provision for certain defectives would ulitmately become necessary, as has been the case in London, in order to secure such degree of education as they are capable of receiving, and to prevent neglect. In addition to the identification and separate consideration of defectives of these classes, and the consequent removal of much misery and heartburning now undergone by certain unfortunate children who are punished for faults beyond their control, results obtained elsewhere show a distinct increase in mental re-action from measures of School Hygiene. To quote a single instance, it was found by Carnelly that the installation of mechanical ventilation in three schools resulted in an increase of passes in reading, from 95.6 to 99.6; in writing, from 90.4 to 97.8; in arithmetic, from 84.2 to 92.4; and in the grant earned, from 19s. 6d. to 21s. 8d.— as compared to fifteen schools ventilated by natural means only. I do not desire to use this as an argument for the universal introduction of mechanical ventilation, but as an instance of the beneficial influence of improved hygiene upon the prospects of a conscientious teacher working under a system of classification. Reasoning upon these lines, a series of twelve lectures in School Hygiene for Teachers has been delivered in Hobart during the present year, with the cordial co-operation of my valued colleague and friend, Mr. W. L. Neale, late inspector of schools in South Australia, and now Director of Education in Tasmania. The results have been most gratifying; and although sufficient time has not elapsed to permit of the

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collection of any data as to the possible educational influence of such instruction from an examination point of view, a notable advancement has occurred in the hygienic management of a large number of our schools.

For these and other reasons the instruction of all teachers in School Hygiene is likely to be of much benefit to themselves, as well as to their pupils. A further argument in its favor is to be found in the necessity for recognising those signs of fatigue, physical or mental, which are characterised by the late Inter-Departmental Committee on the Model Course of Physical Exercises (England) as forming to the experienced teacher "the gauge on which he keeps his eye in regulating the work of a class." School stress is far more a matter of defective hygiene than of mental strain; and I view with extreme disbelief the attacks which are sometimes made on the State educational curriculum as being, *per se*, a cause of breakdown in children. There may be more of it than is absolutely necessary from a utilitarian standpoint, but, given sound hygienic conditions, there is not enough in the most comprehensive State school curriculum in the Commonwealth to produce any worse effect than a little intellectual dyspepsia.

Two arguments are occasionally heard, which may be here quoted. One is to the effect that physical culture will probably overcome any bad effects from school conditions. I need not attempt here to explain the absolute futility and unsoundness of such a statement, or to demonstrate the impossibility of attempting to overcome the effect of a couple of hours of somnolence in an atmosphere heavily charged with air sewage by a few minutes of club-waving and toe-touching. I have no intention of decrying the excellent results which may be expected to accrue from a well-planned and carefully executed system of physical culture for schools, but it is, in my opinion, a dangerous procedure when carried out in the absence of a system of medical inspection, whereby the weak lungs and weak hearts may be safeguarded. As Dr. Mackenzie states, "The intention of physical culture is not to cultivate the organism in spite of existing defects. That would only lead to disaster, as no doubt it frequently does." The Royal Commission on Physical Training (Scotland) has collected some startling information on this subject, which, despite the difference in conditions, may well be studied by Australian educational authorities. The second of these objections is summed up in that single word "expense," which almost invariably forms the earliest war cry of the opponent to reform. I have already stated certain arguments which go to show that the maintenance of existing conditions is likely to result in the waste of much more money than would be required for the most drastic hygienic reformation of State schools. Setting this aside, however, I am prepared to state, from actual experience, that not only is it no more expensive to erect schools on moderately hygienic lines, but that it is actually cheaper. There is no need, therefore, to perpetuate, in future schools, the hygienic defects of many existing ones.

The addition of an extra subject to the teaching curriculum carries with it no gigantic financial responsibility, and may even be calculated to relieve the State finances to some degree in respect of sick-leave, compensation, &c., granted to teachers for temporary or permanent break-down. It will further tend to limit the interference with educational work, and consequent expense now caused by certain preventable school diseases, and will enable better value to be got for the taxpayers' money. The inauguration of a system of medical inspection of schools will, undoubtedly, require the outlay of money, although not to a very great extent commensurately with the benefits to be expected. Without going at present into its economic features, I would mention that in most of the American States—and particularly in New York, that home of the hard-headed utilitarian—medical inspection is in full swing; that in Switzerland, the admitted leader of the educational world, every school child and every

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school is frequently and thoroughly inspected; that in Belgium, Hungary, and several other European countries, it has existed for years; and that Japan possesses a vast organisation, wherein are utilised the part or whole time services of some 8,400 medical men. The Scottish Commission on Physical Training has recommended it, and England is shortly going to get it—unless the portents read much awry. We may hence assume that it pays, and that it is no new and daring device of wild sanitarians for ruining the country.

The practical instruction of children in the elementary laws of health, and of health-protective purpose, involves no material expenditure, and can be productive of nothing but good. Unfortunately there has been in the past rather too much done in this way for the "child as he should be," and all too little for that common object of the schoolroom the "child as he is." There has been too much book, and too little treatment of the subject in a live practical fashion calculated to render the knowledge of Elementary Hygiene as wide spread as is the knowledge of reading: there has been too much of a tendency to gravely inform the Australian twelve-year-old that "indulgence in alcohol is detrimental to the human organism," and too little instruction in the benefits to be derived from the proper use of a toothbrush, and the disgrace and danger of spitting, with regular practical demonstrations of the methods of ventilating and even of cleaning the schoolroom. The formation of hygienic habits of breathing, and above all the strenuous enforcement of correct habits of working attitude, from the day the child takes his place at his usually misfitting seat, have been neglected in the past. All such teaching must be intensely practical, and the habit-formation tendency of the youngest children must be fully utilised. Physiology is useless until the basic laws of healthy living have been learned from example and precept, and those pretty demonstrations with lamp glasses and a cigar box, or with limewater and a glass tube, so affectionately described in the books, should be only employed after a working school sanitary organisation has been formed amongst the children themselves, coincidently with a rigid standard of personal cleanliness. It is not implied that any preponderating amount of the school day should be occupied with Elementary Hygiene, or that all other teaching should be subordinated to it; its economic importance, however, warrants its inclusion as a frequent subject of instruction. As a pedagogic subject it needs no apology, in that it teaches the child to see, to reason, and to remember; it lends itself both to training and instruction, and it possesses high ethical value. In conjunction with its allies, domestic economy and civics, it practically conforms to that science of Eugenics towards which the highest educational thinkers are striving as the coping-stone of modern education.

It will be well to consider a little more in detail certain of the defects, particularly of structure, to which I have alluded. Here I do not refer to Tasmanian schools alone, but to a number of Australian schools concerning which information has become available, either by study of plans or by personal visitation. The trail of the "practical builder"—that curse of school construction—is to be picked up almost everywhere in the evidences of disregarded orientation, neglected lighting, defective ventilation, absence of perflatory facilities, expensive bell towers, and fretted stone window-borders, dust trap interiors, and all the other evidences of his diseased love of architectural frippery. The educational authorities in Victoria, at the instigation, I believe, of Dr. Norris, have apparently succeeded in checking to some extent the machinations of the school architect; and in Western Australia, well-meant attempts are made to secure hygienic decency in the newer schools. I trust I will be excused for saying, however, that most of the larger and more

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ornate school buildings of the Commonwealth, other than those of quite recent date, display the methods of the slattern—all show and pomp without; all dark, foul, and difficult to cleanse within.

A school building is erected for a particular purpose, namely, to contain. for several hours daily during a large number of days in the year, a number of young animals of naturally dirt-collecting and dirt-depositing tendencies, particularly prone to attack by certain pathogenic agencies, and assembled for the purpose of receiving instruction principally through their eyes and ears, of which the former organs are physiologically liable to injurious strain. It is thus only reasonable that, in planning and constructing such a building. very particular attention should be given to its lighting, to its air supply, and to those internal and external conditions which have to do with preventing the accumulation and facilitating the removal of organic and possibly infective material. The children assembled in such a building come from scores or hundreds of separate abodes, and a school is, hence, capable of forming a remarkably effective centre for the dissemination of disease. That schools do act in this way is well known to us all; and the masterly work of Dr. (now Sir) Shirley Murphy, and of Dr. Kerr of the London County Council Education Department, has put this question on a new and significant footing. Nevertheless, the sole object of the school-builder in the past appears to have been to devise a fabric as much as possible resembling a cross between a Chinese pagoda and a Gothic barn, and to set that building square with the nearest street or road, regardless of the sun's track or of anything else, excepting a desire for apparent neatness and regularity. Having outlined his building, he appears to have proceeded to cut up its interior into various rooms, passages, assembly halls, and so on, according to its size, and to stick on—still with a pathological desire for neatness—such adjuncts as were required to make it resemble the last school of that type which he perpetrated. He then put in the windows at regular intervals all round, quite irrespective of size, or of direction of existing light relatively to the seating lines; pepperboxed a few fireplaces and things about in convenient places, added a belfry, and made a few holes in the ceiling (if there were a ceiling) for "ventilation." If he remembered inlet-ventilation, he specified something like "Tobin's tubes, price not exceeding five shillings each." This may perhaps appear to be a flippant and even malicious description of the methods pursued; but, if I am dealing unjustly with the school designer of former days, he has managed to effectually conceal all evidence in his own favor.

Time does not permit of the detailed consideration of principles of school-construction, but certain general matters in conjunction therewith may be mentioned. Before doing so I desire to disclaim any intention of formulating a cut-and-dried system suitable to all States of the Commonwealth and all parts of all States. Like the children they are intended to accommodate, schools require to be fitted to their environment with a due sense of proportion; and what will suit Hobart, for instance, will obviously not meet the requirements of Normanton, any more than it will necessarily suit the Tasmanian west coast. An "Australian Type" of school building is impossible; and, in fact, "type plans" are open to grievous misuse in any single State unless carefully and intelligently used. Certain architectural principles must be embodied in all, for reasons of economy and of administration; but although the possession of a neat set of plans, all fitting together and interchangeable, like the works of a Waterbury watch, may appeal to some, it will be found in practice that when school type A is converted into school type B, by rule of thumb, the result is seldom satisfactory. It is equally cheap and far more satisfactory wherever the facilities of staffing will permit, and particularly in large schools, to adopt the system of school

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units, whereby, in place of outlining a block and cutting it up into rooms, the block is built up from the rooms to whatever size may be required, with adequate provision for subsequent extension on similarly correct lines. By regarding the teaching unit as the principal objective to be considered in planning and placing the school, and by adopting definite structural requirements for each such teaching unit, the problem of hygienic school construction is much simplified. The adjuncts can be fitted in afterwards; and once this principle is grasped we will no longer find teaching rooms faced to the south, with a blank wall to the northern sky, and an assembly hall fronting east.

The accommodation in any one teaching room of more than forty children is undesirable: for reasons of lighting the rooms should not be over 24 feet in width, and cubic space must not be measured over 13 feet vertical. The existing *minimal* standards of floor space and cubic space are undoubtedly too low, but they have been apt in the past to be regarded as *maxima*. Any notable increase involves large expenditure, however, and as schemes of mechanical ventilation are out of the question—for the majority of Tasmanian schools, at least—ideal conditions of air supply are unattainable in cold weather. The provision of adequate and properly placed ventilating and perflating openings, of effective pattern—*not* builders' "ventilators," but real ones that will, under ordinary conditions, admit or extract a definite amount of air, and can be cleaned—and their intelligent use in conjunction with the windows, by teachers, together with hourly perflation at recess or change of classes, will tend to keep the air sewage within reasonable limits. All ventilating provision should be primarily intended to serve the "breathing line." The requirements for windows, both as regards amount and direction of entering light, and as adjuncts to ventilation, deserve careful study. I am in the habit of advising for Tasmanian teaching units a *minimal* glazed area of one-fifth of the floor space, with bevelling of piers, and the use of practicable windows—preferably of the well-known "Magic" pattern—surmounted where necessary by hopper top-lights with side-checks, and reaching to within 6 inches of the ceiling. The seating-line must be planned with the windows as a definite portion of the lighting scheme, and rigidly adhered to. School lighting has a literature of its own, however, and it is unnecessary to go further into it.

Interiors must be so designed as to avoid the accumulation of dust and dirt, to facilitate its early detection and removal, to assist in the diffusion of light, to relieve eye-strain, and to necessitate as little attention and repair as possible. All these requirements may be easily and cheaply met. School fitments, and particularly desks and seats, form a subject quite capable of occupying the whole of this paper; but, beyond bestowing a whole-hearted condemnation upon that survival from educational dark ages—the long moveable form and desk—this can receive but passing mention. Backs to school seats are essential, to avoid the pernicious effects of a "forward sitting" position, and even where accurate difference cannot be obtained, proper distance should be secured by fixing seat and desk in correct relative position. To my mind the most practical solution of this very serious question is to be found in the "Sheffield" method advocated by Dr. Kerr, wherein separate backed seats, each supported on an iron standard, are used at a long desk similarly supported. This takes up no more space per head than do dual desks; is very easy to clean; is cheap, strong, and simple; and can, by a simple mechanical device, be rendered adjustable in difference for a proportion at least of the scholars in each room. Heating apparatus is of importance, and although the more expensive pattern of school-ventilating stove is often impracticable, a very fair substitute is obtainable at small cost by using an ordinary circular stove surrounded by a metal cylinder to the bottom of which air is admitted by a trunk under the floor. The heat of the flue can be employed to

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pull used air through galvanized iron exhausts opening near the floor at the opposite end of the room. Undue drying of the entering air can be avoided by a pan of water. The saving of fuel, as compared to the wasteful open fireplace, will amply repay the cost of such a device.

Certain adjuncts to the teaching unit, or group of units, require consideration, both in the main building and outside it; as do the arrangements for rotation of classes in respect of the degree of lighting, &c., rendered necessary by their work. All schoolrooms used for teaching cannot, in a large school, face to the north-east; but, as far as possible, only those teaching units which do command the best light should be employed for fine work involving eye strain. The placing of the building on the site should be such as to secure main-lighting from the north-east, and I have no hesitation in stating that this should completely override all considerations of alignment with the street or other relatively subordinate objections. The school is erected for the benefit of the children, and as a necessary adjunct to that education which the State has ordered. No æsthetic considerations will justify any unnecessary risk to their eyesight and health, and at the worst the lack of correct alignment will but form a standing testimony to the care of the State for its most valuable assets.

Accommodation for outer garments and lavatory provision can be secured at little outlay. Latrine provision is of great importance, but need not be here dealt with in detail, beyond a passing reference to the excellent results which have been obtained from the use of sawdust or dry earth urinals, slightly modified from the pattern advocated by the late Dr. Poore.

For obvious reasons the inside of a school requires far more attention than does the outside, and all unnecessary and expensive external architectural vanities should be ruthlessly swept away. In the case of many large schools the money which has been wasted on bell-towers, carved stone porches, and other meretricious adornments of no practical use, would have enabled the interior to be fitted with modern hygienic facilities. There is no need to aim at the severe simplicity of a gaol, but rather, within reasonable limits, to adopt the utilitarian but yet æsthetic principles of modern hospital construction.

By a useful inter-departmental arrangement all plans of new State schools, and additions to existing school buildings, in Tasmania, are now submitted for inspection to my Department, and are not passed until approved by the Director of Education and myself.

The hygienic training of teachers requires to be carried out on practical—even clinical—lines. Reading is, of course, necessary; but in any set course of this nature, practical and clinical demonstrations, plentifully illustrated by lantern slides taken in the locality, and not imported from other places where conditions differ, should form a large part of the training. Women teachers should be practically instructed in the care of infants, and in the principles of clothing, washing, and feeding them. The true position of artificial feeding, as a last resort, and *not* as an admissible alternative to breast-feeding, should be insisted upon. Domestic economy and civics should be freely interwoven with any such teaching, and the whole made to form a living subject, and not a mere collection of book aphorisms and half-digested formulæ. Comparatively little physiology is necessary, and what is employed should be carefully subordinated to its practical application. The use of Snellens' types, together with their range of application in lay hands, the methods of conducting head-and-hand-drills, of detecting adenoids, eye defects, and other physical obstructions to mental re-action, should form a part of the training, freely illustrated by clinical examples and slides. Practical elementary hygiene, including methods of dealing with organic refuse, of ventilating, warming, and lighting—and particularly methods of managing and improvising facilities of

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this character—should be explained and demonstrated. The signs of fatigue, and the conclusions to be drawn from such signs, together with measures for remedying the same, and the principles of correct working attitude are further subjects which may well receive attention. These form but a part of any full course of hygienic training; but, for teachers possessing live knowledge of this character, the systematic observation of pupils for physical unfitness, hygienic school management, and the organisation of practical instruction in the ways of healthy living should present little difficulty.

A definite hygienic purpose can be made to enter into, and influence, every day of the child's school life. In the infant classes habits of correct working attitude and of proper breathing may be fixed for ever by a skilful patient teacher. The spitting habit should be here vigorously attacked. I trust that I may be excused for referring to a remarkable fact stated at the 1905 Teachers' Conference in London, namely, that the work of the Kindergarten teacher and the nursery governess far outweighs in educational importance the most brilliant displays of professorial erudition. This individual dominates the whole educational system; and unless her work is skilfully and conscientiously performed no subsequent teaching can remedy her errors; but, on the other hand, no subsequent mismanagement can entirely negative the beneficial results of skilful infant teaching. It is the age of habit-formation with which she deals, and her fingers mould the nation. No books, no "set talks," no detailed explanations, are of any use in her work. The child is strenuously learning life, and the results of that learning are expressed in fixed habit. He educates himself from anything that is available, and the infant teacher's business is to render available the right things in the right way, and to ensure that they are rightly used. It is the most skilful of all teaching, and the most momentous in its results.

In the middle school a little set instruction, and much practical demonstration and application, will carry on the teaching. The selection at monthly or fortnightly intervals of a sanitary officer for each class, and the deputing to that important functionary of direct responsibility for the ventilation, regular perflation, and general hygienic management of the room, would, in the hands of a judicious and skilful teacher, awaken much interest in such matters amongst the children. To this should be added practical demonstrations of cleansing methods, and of other principles of domestic hygiene, strict adherence to a high standard of personal cleanliness as checked by daily head-and-hand-drills, and occasional talks on elementary principles. Books here require to be very simple, but artfully constructed withal, and absolutely free from any trace of that enemy of our youth, Mr. Barlow.

In the upper classes the appointment of a sanitary officer should be continued, but such appointment should be made by ballot, as forming a peg on which to hang much useful information anent civic and municipal government and methods. Some reasoning may be expected from children in this division, and principles may be explained. Elementary physiology is useful here if employed for the purposes of explanation and illustration. Instruction in elementary civics and domestic economy should be interwoven, and the physical side of the environment kept clearly in view. Communicable diseases, their results to the individual and the community, and measures for their limitation and extinction by domestic or civic isolation and disinfection, may be explained in some detail, together with the common-sense rationale of health protection and preservation in the individual and in the community. In the larger centres regular demonstrations of proper methods of clothing and caring for infants may, with advantage, be given to the older girls by a trained nurse or other skilled person; artificial feeding being explained, and illustrated, whilst emphasizing its true position as an

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undesirable alternative. By the exercise of a little tact, it would generally be possible to borrow a live subject for purposes of demonstration. The attendance of mothers, and particularly of young mothers, should be encouraged.

In large schools a sanitary monitor of each sex should have the general supervision of all school sanitary matters outside the teaching rooms, reporting daily to the head teacher, or principal female assistant respectively. These reports should, after investigation, where necessary, be recorded for examination by the inspector.

All this may perhaps sound rather formidable, but there is good reason to believe that it and a good deal more might—and, as I have no reason to doubt, eventually will—be made to form a part of the educational administration of at least one Australian State, without much risk of hygienic overfeeding. In this I am supported by the opinion of educationists of wide experience, and by the keen interest and appreciation with which Tasmanian teachers have welcomed my own efforts in this direction. Time does not permit me to deal with the practical details of medical inspection, but it will be only through the co-operation and intelligent assistance of teachers that such procedure can be organised or conducted on economic or effective lines. That this co-operation and assistance will be forthcoming I have good reason to believe, so far at least as my own State is concerned. All measures of medical inspection and of hygienic school management, all measures in fact of School Hygiene, have for their object the enhancement of school efficiency. The leaders of medical and educational thought in the older world recognise this; and I am of opinion that the time has come when educationists and medical men in Australia should work manfully together for the physical and mental advancement of our race. Such co-operation is not confined to professional hygienists and to educational administrators: every practising medical man, and every teacher, has a definite interest in the question, and can afford useful assistance in the cause of common-sense hygienic education, and of common-sense school management.

I am painfully aware that, in setting forth to address this learned audience upon so wide a subject as that of School Hygiene, I have been unable to do more than touch upon a few of its more important aspects. The treatment has of necessity been sketchy, the chain of reasoning as presented lacks many of its links, and the suggestions for rectification of existing conditions have not been worked out to meet particular cases. Nevertheless, I present this paper with all its imperfections, in the hope that it may perchance serve to elicit the opinions of others who may have worked at or given thought to the subject, and to attract, in some degree, the attention of our profession as a whole to what is without doubt a matter of great national importance to Australia.

COMPULSORY NOTIFICATION OF PHTHISIS.

By T. Borthwick, M.D., Edin.

I propose to open the discussion on this subject by simply referring to the sections of the South Australian Health Act which deal with this disease, and to the administration of these sections as far as Adelaide is concerned, and the experience gained thereby.

Phthisis is termed Pulmonary Tuberculosis in the Health Act, the reason being partly to define the disease with greater accuracy and partly to avoid a term which conveyed a rather deadly significance to the public. It was felt

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that it would be more easy to convince the public of the curability of the disease under the one term than under the other at what was an early period in the campaign against consumption.'

Phthisis is not included under the term "infectious diseases," as defined by the Health Act. If it had been so included, patients would have been subject to the restrictive clauses applying to these diseases. This would have caused much hardship in many cases, and, in a new departure which might prove to be premature, it was incumbent to disarm opposition as far as possible.

Section 128 renders it compulsory on medical practitioners to report a case as soon as the fact becomes known to them. The head of the family, or nearest relative, is not required to report. Section 131 gives power to local boards to carry out disinfection, and section 132 gives power to remove a patient from a building used for the storage of milk, or the storage or manufacture of any article of human food. There is no other restriction imposed on the movement of persons suffering from phthisis.

It will be seen that the framers of the Health Act took into consideration the susceptibilities of patients, and also tried to obtain the confidence of the public. But there was no discretionary power given to the medical practitioner in the matter of reporting cases, and many of these looked on with passive disapprobation, fearing that the interests of their patients might suffer. In order to disarm this opposition, it was decided by the city and some other local boards to take no action in cases where the medical attendant, in notifying, undertook to see that all necessary precautions were carried out, and to report to the board any change of address or the death of the patient.

The number of notifications of cases, and of deaths, from phthisis, in Adelaide, since the Health Act came into operation are as follows :—

Year ending September 30th—	Number of Deaths.	Number of Cases.
1899-1900	70	72 } 188 { 94
1900-1901	67	116 } { 94
1901-1902	64	79
1902-1903	67	82
1903-1904	63	89

Cases coming into the city for treatment are not included, but it is impossible to make any other correction in regard to the numbers. I do not think any conclusions can be drawn from this table. What decline there is seems to be in the number of deaths rather than of cases, and it is possible that the success of the campaign against consumption will show itself first in this direction. The disparity in the number of cases in the first two years is probably accounted for by the hesitation of medical practitioners to notify cases during the first year, and the complete abandonment of that hesitation afterwards. By striking an average for these two years, we may arrive at the approximate number for each. So completely has the administration of the Health Act in the city satisfied the medical profession that it may be safely assumed that every case is now notified, and very rarely do medical practitioners request us not to take any action.

The routine action in Adelaide is practically the same as in a case of infectious disease. When a case is notified, the trained nurse attached to the staff of the Health Department visits the house and interviews, in the first place, some relative of the patient. She advises as to the precautions to be taken by the patient and attendants, and also as to the use of disinfectants, leaving printed instructions for further guidance.

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Disinfectants are supplied to the poor free of charge, and sputum is also examined gratis. I personally rarely visit cases (only for some special reason), partly because the nurse gets all the information I require, and partly because I think it advisable to come between the patient and the medical attendant as little as possible. The nurse keeps in touch with the patient during the course of the illness. At its termination, by recovery or by death, the contents of the room or rooms are disinfected under her supervision, and the rooms themselves are afterwards disinfected by a sanitary inspector without any charge being made. Insanitary conditions of the house are also attended to by him. It is found that the poorer patients frequently change their place of residence, and all houses so vacated are disinfected before they are again occupied. In order to minimise leakage, the city local board decided to pay to medical practitioners, in addition to the ordinary fee for notification of cases, a fee for notification of change of address or of the death of patients. When it is found that the patient has moved into another district the local board of the district is informed of the fact; and a few boards adjoining the city reciprocate this action. Experience has shown that it is especially in relation to phthisis that a woman has the advantage over a male inspector. At first she was looked upon with suspicion by patients and relatives, but, with the exercise of tact, this was soon overcome, and all classes now welcome her as a valuable ally. She is cheerfully admitted into the sick-room, and, by establishing friendly and confidential relations, succeeds in persuading the most obdurate patients to carry out instructions. The educational effect of the nurse's work on the patient's family and on the neighbors must not be overlooked. In addition to the appreciation of our methods by patients and relatives, there is a more general recognition in the shape of requests to disinfect offices where a person suffering from phthisis has been employed, and where the fact has been unknown to the health department owing to the patient having lived outside of the city. It is coming to be recognised that such patients need not be shunned in the event of proper precautions being adopted, and I do not know of any case where the material prospects of a patient have been injured as a result of notification of phthisis.

Notification of phthisis, to be of practical value, must be followed by other measures—disinfection, facilities for bacteriological examination of sputum, and the provision of sanatoria for isolation and treatment. I shall content myself with saying that the latter institutions have been fairly provided in this State; but experience has shown that there is a great difficulty in getting early cases—especially when the patients are bread-winners—to enter sanatoria.

To meet this difficulty, the establishment of a fund to assist the family to live during the absence of the bread-winner is a necessity.

A REVIEW OF SOME RECENT WORK ON THE ETIOLOGY OF DYSENTERY.

By Frank Tidswell, M.B., Ch.M., Syd., D.P.H., Camb., and James Froude Flashman, B.A., B.Sc., M.D., Ch.M., Syd.

I. Introduction.

Although the term "Dysentery" has been in use from a remote period, it does not seem possible at the present day to express its meaning with definite precision. Clinically, it is associated with the passage of blood and mucus, per anum, attended with pain; and, in this sense, embraces "several

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varieties of intestinal flux" (1). It is recognised that such a condition may result from the operation of mechanical and toxic agencies, or be a part or merely an event in the course of well-defined maladies (heart affections, Bright's disease, tuberculosis, pneumonia, &c.). But, apart from this symptomatic significance, the word is used also to denote a particular morbid entity—a specific disease—which finds a place in systematic nomenclature under the designation "Dysentery." It is with this latter connotation of the term that we propose to concern ourselves in this place.

To the epidemiologist dysentery formerly signified a disease of endemic prevalence in certain tropical and sub-tropical lands, but which appeared now and then under the guise of epidemics in more temperate regions; as such it was regarded as a specific disease. Modern views, however, have come to look upon this conception as of questionable validity. "We cannot say for certain," states Sir Patrick Manson (2), "whether there is but one disease having grades of severity, or a dozen specifically distinct diseases included under the term dysentery." B. Scheube expresses himself to the same effect. "It is a question that is still waiting to be solved," he says (3), "whether dysentery is a specific disease, or, as is largely assumed, whether it is attributable to a variety of causes; nor has it been settled whether tropical dysentery is etiologically identical with the dysentery of temperate climates."

It is to be observed also that clinicians nowadays are equally sceptical with respect to this matter. "In individual cases," writes Andrew Davidson (4), "dysentery often arises out of what seemed to be an attack of simple diarrhœa, so that no hard and fast line can be drawn between diarrhœa and dysentery." Hale White refers (5) to the possibility of confusing dysentery with ulcerative colitis, and even with simple colitis; and Pye Smith remarks (6) "whether cases of ulcerative colitis, with passage of blood and mucus . . . . . should be called dysentery is, from a clinical point of view, little more than a question of terms." L. Emmett Holt prefers to drop the term dysentery as applied to these conditions in children, and to group all such cases under the general head of ileo-colitis, which he considers is "to be regarded as a condition into which any case of gastro-enteric intoxication (gastro-enteritis) may develop" (7). Mott and Durham believe asylum colitis to be identical with the condition which, when occurring elsewhere, is called dysentery. "The disease we have had to deal with," they report (8), "agrees in its anatomical signs with the dysenteries described by observers in this and other cooler climates as well as those of warm regions; the range of variations in the clinical and anatomical conditions is the same in all, and we see no reason why they should not be grouped under the one title—dysentery."

It would thus appear that the idea of dysentery as a specific disease is far from being accurately delineated. One gathers from such statements as those cited that conditions referred to as colitis, whether sporadic or occurring as institutional outbreaks, as well as some forms of diarrhœa—including infantile diarrhœa—may perhaps be identical with the endemic or epidemic malady or maladies actually called dysentery. Yet, in spite of this inclusiveness, there is pretty generally displayed an undercurrent of thought to the effect that there is somewhere a line of cleavage separating a true specific dysentery from resemblant conditions. The suggestion is, that although present sub-divisions may need to be abandoned, there will come a time when a re-arrangement may be made on a satisfactory basis. It is in accordance with the spirit of the times that such a basis should be sought for in the realm of etiology.

The ordinary etiological circumstances mentioned in connection with dysentery—water-supply, food, exposure, chill, &c.—are not peculiar to it.

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They are to be regarded either as predisposing conditions, or as vehicles for the conveyance of the real causal agent: this latter being presumably of a microbic nature. In view of this hypothesis, numerous investigators have engaged in microbiological researches with the object of determining whether any particular micro-organisms are associated with the disease. The issue as it stands at present incriminates two kinds of parasites—amœbæ and bacteria—and upon both an abundance of attention has been bestowed. So much is this the case, so numerous are the reports and so conflicting in their advocacy of different views, that a clear conception of the actual state of affairs is by no means easy to attain. We have been obliged to attempt this task recently as a matter of official duty, and we have put together in this paper the information we were able to gather, in the hope that it might prove of service to others who have not the time or opportunity to collect it for themselves.

II. Amœbic Dysentery.

The existence of living protozoa (cercomonas, paramœcium) in the bowel excretions of human beings was demonstrated about half a century ago, and it is stated the occurrence of amœbæ therein was first recorded by Lambl in 1860. In his case the amœbæ were found in the stools of a child suffering from diarrhœa. In 1870 Cunningham reported their presence in the evacuations of cholera patients. In neither of these cases was any pathological significance attached to the amœbæ. But in 1875, Losch, as the result of a careful investigation of a case of chronic dysentery at St. Petersburg, suggested that the amœbæ found in the stools might have a causal relationship to the disease. Since that time this aspect of the question has been the subject of numerous investigations (9).

The occurrence of amœbæ in the stools of dysenteric patients is no longer a matter of dispute. They have been found in Egypt (Kartulis, Koch, Kruse, and Pasquale, and others); Russia (Losch, Massiutin); Germany (Nasse, Quincke & Roos, and others); Austria (Hlava, Kovacs, Epstein); Greece, (Kartulis); Italy and Sardinia (Calandrucia and others); the United States of America (Osler, Simon, Councilman & Laffeur, Howard & Hoover, Thayer, Stengel, and others); in Brazil (Lutz); in the Philippine Islands (Flexner, Musgrave, and Clegg); Australia (Isbister) (10), &c., &c. The instances given suffice to display a very wide-spread occurrence of the amœbæ in association with dysentery cases.

But whilst it is admitted that amœbæ do occur as stated, it is pointed out that they occur also in non-dysenteric conditions, *e.g.*, in cholera stools (Cunningham), in typhoid stools (Massiutin, Casagrandi & Barbagallo-Rasipardi), and in the stools of healthy persons (Cunningham, Schuberg, Kruse, and Pasquale, Gasser, and others). In view of the abundant occurrence of amœbæ in the surroundings of places where dysentery is common (Egypt, Philippine Islands), the presence of the amœbæ may be merely accidental.

It is further urged, however, that the amœbæ are not merely loose in the bowel, but penetrate the tissue, being found at the base and in the vicinity of dysenteric ulcers, and in the pus of liver abscesses associated with dysentery. This also is admitted, but the significance to be attached to their occupancy of these positions is a matter of dispute.

As regards the intestine, it has been suggested that the ulcerative lesions are caused by other agencies (*e.g.*, bacteria), and only secondarily invaded by the amœbæ, which themselves are not able to cause ulceration (Casagrandi and Barbagallo-Rasipardi, Janowski, and others). But it would appear that the amœbæ do really possess the power of setting up lesions of the intestine

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(*vide infra*), and, further, that although digestion in amœbæ is usually regarded as occurring in vacuoles, some of them at least can exert a solvent action on external materials.

Again it is stated that the liver pus does not always contain amœbæ, and very frequently does contain bacteria, *e.g.*, bacillus coli communis, streptococci. Some authors argue that the abscess is due to these bacteria (streptococci, Zancarol). On the other hand it is urged that the pus in some cases contains only amœbæ, and is bacteriologically sterile; and further that the peculiar character of the pus (few leucocytes, much debris and blood), and the absence of a definite abscess wall, is more consonant with its production by amœbæ than by bacteria.

The advocates of the pathogenicity of the amœbæ point to the production of dysentery by experiments on the lower animals. It appears that the usual laboratory test-animals—rabbits, guinea pigs, &c.—are insusceptible, and most of the experimental work has been done on dogs and cats. According to Kartulis, dogs naturally affected with dysentery in Egypt have amœbæ in their stools, and dogs have been several times experimentally infected by rectal injection of amœbæ-containing stools (Losch, Hlava, and others). Cats appear to be even more susceptible; at all events they have been more commonly used in experimental work. Successful results by the rectal injection of dysenteric stools or liver pus with these animals have been reported by many observers (Kartulis, Quincke & Roos, Kruse & Pasquale, Hlava, Vivaldi, and others). With respect to reported failures it is recognised that attempts are not always successful; the results may differ even with the same material.

Concerning this infection of cats it is urged that the intestine of these animals is very easily disturbed, and that a condition resembling dysentery can be produced by merely mechanical means, *e.g.*, injection of sand. Some authors assert that when dysentery follows the injection of the stools there are no amœbæ in the cat's evacuations (*e.g.*, Celli & Fiocca), but others report the presence of amœbæ therein (*e.g.*, Kruse & Pasquale). It is stated further that dysentery in cats is producible by the injection of stools in which the amœbæ have been killed by heating (Gasser, Celli & Fiocca), but this is denied (Kartulis); and also by the injection of amœbæ-free pus from a liver abscess (Zancarol). It is obvious, of course, that this experimental method is not free from the objection that the condition so produced may be due to toxic substances, or to bacteria; although, as regards the latter, it is stated that the bacteria associated with the amœbæ do not produce dysentery (Kruse & Pasquale, Harris). This objection is in course of removal by observations made, not with stools or pus, but with amœbæ isolated therefrom by cultures in hay infusion (Kartulis), and on solid media (Ogata, Schardinger, Muller, Celli & Fiocca, Schaudinn, Musgrave, and Clegg). Pure cultures have rarely been obtained; as a rule the amœbæ only grow when there are also present bacteria upon which they may feed. But it is possible to secure growths containing the amœbæ and only one kind of (non-pathogenic) bacterium; what has been rather paradoxically, called a "pure-mixed" growth. By the use of such cultures results have been obtained which indicate that there are in the intestine both harmful and harmless amœbæ, *i.e.*, the rectal injection of one kind produces dysentery, that of the other does not (Kruse & Pasquale). From his investigations in this direction, Schaudinn has come to recognise at least two species of amœbæ habitually parasitic in the human intestine (11). One of these—amœbæ coli of Losch, re-christened entamœba coli by Schaudinn—is harmless; the other—entamœba histolytica of Schaudinn—is pathogenic. The distinction between the two is made on the basis of their morphology and method of reproduction.

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Schaudinn believes that dysentery is produced by ingestion of the "spores" of entomœbæ histolytica, not by the naked (amœbæ) forms. The administration of these with food produced dysentery in cats. Quinck and Roos had previously reported that dysentery could be produced if encysted forms were given by mouth. The objection raised by Calandruccio, that he failed to produce dysentery in himself by swallowing amœbæ, including encysted forms—although amœbæ appeared in his stools—may perhaps be based upon the use of a harmless species. No doubt much of the previous work was vitiated in the same way. The observations of Musgrave and Clegg (12) in the Philippine Islands generally confirm Schaudinn's results. These investigators produced the intestinal lesions of dysentery in monkeys, by the administration of "pure-mixed" cultures by mouth, and hepatic abscess by injection of such cultures directly into the liver. They report that the feeding of dogs and cats was generally negative; but that a human being, who took the amœbæ in gelatine capsules, acquired dysentery. They express the view, however, that all amœbæ found in the intestine are, or can become, pathogenic; and even those found outside the body, *e.g.*, in water, straw infusion, &c., can be rendered harmful by passages through the liver of animals. The validity of this contention remains for future determination.

In the meantime it is reasonable to conclude from these various results that amœbæ, or at all events a particular species of them, can produce dysentery. But it is not believed, nor indeed is it contended, that dysentery is exclusively caused by amœbæ; the particular form of malady for which they are held responsible is that commonly referred to as "tropical dysentery"; but this term does not now imply that all the dysentery of the tropics is caused by amœbæ, nor that amœbic dysentery is restricted to the torrid zone. It is considered to be more prevalent in warm climates, and to have endemic centres in Egypt and in Greece; but it has been observed also in North America, and in more northerly parts of Europe (Russia, Germany, Austria, &c., *vide supra*). This form of dysentery is characterised clinically by an irregular course and a tendency to become chronic, and, pathologically, by the formation of undermined intestinal ulcers and a liability to hepatic abscess. But, in addition to this ulcerative form, there are described "catarrhal" "diphtheritic," and "gangrenous" varieties of dysentery in which amœbæ are not found. These three conditions, it is suggested, may be merely grades of one and the same disease: a disease which, whilst "endemic" or "sporadic" in some localities, is apt to assume at times epidemic prevalence even in territory which has previously seemed to be free from it; and which, in individual cases, usually runs a more or less acute course. It is to this malady, if to any, that the idea of dysentery in the sense of a specific infectious disease applies. Hence the observations on amœbic dysentery, that we have just considered, bring us nearer to the object of this paper only in so far as they enable us to separate off from a group of maladies labelled "dysentery" a particular class of cases dependent upon the agency of amœbæ (amœbic enteritis); just as, for other reasons, there have already been separated classes of cases dependent upon toxic or mechanical influences, or which are incidents of other diseases.

III. Bacterial Dysentery.

The features of the non-amœbic dysentery just referred to are sufficiently analogous to those of other diseases known to be caused by bacteria as to suggest the operation of these agents also in its case. For some time, however, the searches made did not result in the detection of bacteria other than those which habitually occupy the intestine. For this reason many observers were induced to conclude that dysentery did not depend upon the activity of

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any special micro-organism; other observers, however, having found a particular species predominating or permeating into the tissue, or possessed of particular virulence, have attached to it the onus of producing the disease. Of such species the micro-coccus of Durham, the streptococci of Zancarol and Petridis, the diplococcus of Silvestri, the streptothrix of Pottein, bacillus enteritidis sporogenes (Klein), bacillus pyocyaneus (Maggiora, Calmette, Kruse, and Pasquale), the bacillus of Ogata, and various unnamed bacteria (Orth, Ziegler, Krebs, Laveran, and others), need no more than mention in this place, since subsequent investigations have not endowed them with any special importance.

More interest attaches to the belief expressed by some that the disease is dependent upon nothing else than bacillus coli communis (Viellon & Jayle, Maggiora, Arnaud, Pottein, Alessandri, Escherich, Valugussa, Campbell, Galli-Valerio, and others). It is significant, however, that very often the species isolated is said to possess a much higher degree of virulence than ordinary bacillus coli communis, and quite commonly referred to as a variety of that species, *e.g.*, bacillus coli dysenteriæ of Celli. Chantemesse and Widal also isolated a particular bacillus of this kind. But the indications in this direction were not very definite until, in 1898, K. Shiga described as isolated by him from cases of dysentery in Japan a bacillus which, though resembling bacillus coli communis, was nevertheless distinct from that species. Whether or not any of the previously isolated coli "varieties" are identical with Shiga's bacillus, as some authors maintain, is a matter which it would be profitless to discuss in this place. We may therefore pass to the consideration of the evidence brought forward by Shiga in favor of his contention that the bacillus isolated by him is etiologically related to dysentery.

In a preliminary communication (13) Shiga reported the results of his bacteriological observations upon thirty-six cases of epidemic dysentery, and mentions the regular detection of a bacillus having certain specified characters. In particular the bacillus was agglutinated by the blood serum of persons sick with dysentery, and was not agglutinated by that of persons suffering from other diseases, nor by that of healthy persons. Other bacteria isolated were not agglutinated by the serum of dysentery cases. In a further paper (14), Shiga gives details concerning the morphological, cultural, and pathogenic characters of the bacillus, its toxins, the production of specific agglutinins by injection of killed cultures, &c. In a still later and longer paper (15) he reviews and extends his previous work; and, amongst other things, explicitly states the following as the points of evidence upon which his bacillus is to be regarded as the specific cause of dysentery:—

1. The bacillus is forthcoming in all cases of dysentery.
2. It is exclusively related to dysentery: is not found in other diseases nor in healthy persons.
3. In dysentery its occurrence keeps pace with the progress of the disease.
4. It occurs abundantly, or in the pure state, in the deeper layers of the intestinal mucous membrane: on the surface, and in ulcers, it is sparser and outnumbered by other bacteria.
5. The dysentery bacillus and its toxins cause hæmorrhages. In experimental animals there are developed hæmorrhages under the skin, serous, and mucuos membranes, just as occurs in severe cases, as seen *post mortem*, in man.
6. The bacillus is agglutinated exclusively by the blood serum of persons suffering from dysentery: not by that of persons suffering from other diseases, nor by that of healthy persons.

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7. The agglutinating power of the blood of dysentery patients is related to the stage of the disease. It describes a definite curve, which rises quickly at first, reaches its highest point at convalescence, and after that gradually declines.
8. The sub-cutaneous injection of killed cultures of the bacillus causes a marked inflammatory reaction in healthy persons, but no such reaction is produced by similar injection into dysentery convalescents.
9. The Pfeifer reaction is specially evident in convalescent cases.
10. The serum produced by immunisation with the bacillus has preventive and curative efficacy against dysentery.

The details with respect to these various points are such as to justify Shiga's contention that his bacillus is the specific cause of acute epidemic dysentery in Japan; and, indeed, the validity of this conclusion is no longer disputed.

Very soon it began to be reported that dysentery elsewhere than in Japan was caused by the same, or a very similar, bacillus. Simon Flexner (16), as the result of observations on dysentery in the Philippine Islands, reported that there also acute epidemic dysentery was of bacillary origin; and that the bacillus isolated by him was identical with that described by Shiga. R. P. Strong, Curry, Bowman, and others support Flexner's conclusion, as also does F. C. Craig, who, at the Presidio, San Francisco, examined soldiers invalided from the Philippines. Flexner further reports the occurrence of the bacillus in a case of dysentery from Porto Rico, West Indies; from cases occurring at Philadelphia, and at Morvin, N.C. He concludes that acute epidemic dysentery in the Philippines, in the West Indies, and in America is also due to Shiga's bacillus. In the meantime W. Kruse (17) had isolated from cases of dysentery at Laar, Germany, a very similar micro-organism. At first he regarded this as distinct, on the grounds of minor differences from the descriptions of Shiga and Flexner. But a later opportunity of making comparative observations with two specimens forwarded to him by Flexner led him to conclude that his own and one of Flexner's strain were identical; whilst the other Flexner strain (distinguished by Kruse as "Amerika") was distinct from these two. The distinctions were ably based upon agglutination re-actions with sera prepared for each species separately. Flexner (18), as the result of comparative observations, concluded that his own, Shiga's, and Kruse's bacilli were identical; and Shiga (19), who covered the same ground, agrees with Flexner. A commission, consisting of E. Pfuhl, Schmiedecke, Schuder, and Lentz, pronounced in favor of the identity of the bacilli of Shiga, Flexner, and Kruse, as well as of bacilli obtained from an epidemic of dysentery at Doberitz (20). Numerous other investigators, *e.g.*, Vedder and Duval (21) (Philadelphia, &c.), Park Collins and Goodwin (22) (New York), Park and Carey (23) (Tuckahoe, N.Y.), Jurgens (24) (West Germany), Rautenberg (25) (East Prussia), Raczynsky (26) (Galicia), P. T. Muller (27) (Sudstiermark), Spronck (28) (South Holland and Gelderland), Rosenthal (29) (Moscow), Vaillard and Dopter (30) (Vincennes), Leonard Rogers (31) (India), Aldo Castellani (32) (Ceylon), Morgenroth (33) (China), and Foulerton (34) (cases from South Africa, Florida, China, London, and Hertfordshire) report the isolation of bacilli which they identify with one or other of the above-mentioned strains.

As the outcome of these different observations it appeared that the bacillus isolated by Shiga in Japan was also forthcoming from dysentery cases in various other parts of the world; the extensive area covered indicated that this bacillus was to be regarded as the specific cause of acute epidemic dysentery, wherever occurring. According to some observers, it is the cause not only of acute epidemic dysentery, but also of asylum dysentery (Vedder and

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Duval (35), Eyre (36)), and of the summer diarrhœa of infants (Duval and Bassett (37), Litchfield and Hipsley (38)). This conclusion, however, was not universally accepted. It will have been noted that Kruse had separated one of Flexner's strains (" Amerika ") as distinct from his own and the other Flexner strain. It may be now further mentioned that Kruse, at the same time, drew a sharp distinction between the bacillus he had isolated from acute epidemic dysentery and certain resemblant bacilli he had isolated from asylum cases. By means of sera made separately for each, Kruse showed that three strains of asylum bacilli differed in their agglutination re-actions from the " true dysentery bacilli " (his own and Flexner's), and also from one another and from Flexner's " Amerika," although the latter had affinities with one of the asylum strains. To these asylum bacilli Kruse applies the name of pseudo-dysentery bacilli. He holds them responsible for the asylum dysenteries, which thus, in his view, are different from epidemic dysentery and also from one another, although exhibiting the same clinical and pathological features. Certain other observers also note variations from the strict Shiga or Kruse type, which lead them to refer to the bacilli isolated by them as " pseudo-dysentery " or " dysentery-like " bacilli (Lentz, Schmiedecke, Pfuhl) (39). As regards infantile diarrhœa, Martha Wollstein (40) states that the bacilli isolated by her from thirty-nine cases of summer diarrhœa agglutinated with serum prepared with the Flexner (Manila) bacillus, but not with that prepared with the Shiga bacillus ; and P. H. Hiss, jun., and F. F. Russell (41) report having isolated from a fatal case of infantile diarrhœa a bacillus which, though resemblant, was not quite identical with Shiga's bacillus. Further, Martini and Lentz, in a paper to be considered directly, take exception to the method of comparison—testing against the blood serum of patients—practised by most other observers. They quote instances which show that bacilli, otherwise alike, behave differently towards patients' serum, and bacilli otherwise unlike behave similarly towards such serum. Moreover, some of the strains of bacilli are agglutinated by normal human or animal sera, by sera of patients suffering from other forms of inflammation of the bowel (tuberculosis), and by sera of animals immunised against cholera and typhoid. It thus became apparent that the matter was not quite so simple as had been supposed : the notes of discord suggested the necessity for revision of the whole subject.

Towards this revision an important communication was made by Martini and Lentz (42). As already stated, these observers reject the test with patients' blood as being unreliable. For the purpose of differentiation they prefer to use the blood serum of a goat, specifically immunised against Shiga's bacillus, *i.e.*, against this type alone. The serum in question was such as to agglutinate the Shiga type bacillus in a dilution of 1 in 500. Martini and Lentz made use of this serum for the purpose of comparatively testing various strains of bacilli isolated from dysentery cases by various observers at various places. As the result of their investigation they report that, whilst some of the strains behaved as did the Shiga bacillus, there were others which failed to do so. They accordingly divide the bacilli tested by them into two groups : Group 1 comprised ten strains, which agglutinated with the Shiga serum in dilutions up to 1 in 400, viz., the Shiga bacillus, the Kruse bacillus, four strains isolated from acute epidemic dysentery at Doberitz, two strains isolated by Pfuhl from dysentery cases in China, a strain isolated by Flexner at Newhaven, U.S.A., and a strain isolated by Muller at Sudstiermark. The authors state that these strains also closely resembled one another in morphological and cultural characters. In Group 2 they placed all the other strains tested by them, including the strain isolated by Flexner in the Philippines, a strain isolated by Strong in the Philippines, and various " pseudo-dysentery " bacilli (Martini and Lentz, Schmiedecke, Lentz, Kruse, Pfuhl, and Deyche).

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Apart from the distinction into two groups, it is noteworthy that Martini and Lentz report that the Philippine bacilli of Flexner and of Strong are different from the Shiga bacillus: other observers having pronounced for their identity. It is also to be noted that Flexner's Newhaven bacillus is classed with Shiga: it, also, is different from Flexner's Philippine strain. As further evidence of these differences, Martini and Lentz report that the serum of a rabbit immunised with Flexner's Philippine bacillus markedly agglutinated this bacillus, but did not agglutinate the Shiga bacillus, nor Flexner's Newhaven bacillus: further, it had only a slight action on Strong's Philippine bacillus, which thus appeared to be different from Flexner's Philippine bacillus; that is to say, the bacilli of Group 2 are not all of the same species as are the bacilli of Group 1.

Martini and Lentz conclude their able and interesting paper by formally stating the following conclusions:—

1. The serum of dysentery convalescent patients is untrustworthy as a means of identifying the bacilli obtained from the intestine of dysentery cases.
2. This identification can only be made by the use of highly agglutinative serum, obtained by active immunisation with one or other strain.
3. Shiga's, Kruse's, Muller's Flexner's Newhaven (U.S.A.), Pfuhl's Chinese, and the Doberitz bacilli of 1901 are identical; and it is a striking circumstance that they were all obtained from cases occurring in epidemics in the north temperate zone.
4. Other bacilli of dysenteric conditions—for example Flexner's Manila, Strong's Manila, Deyche's Constantinople, and Kruse's asylum bacilli—are different from those above mentioned.

In another communication Dr. Lentz reports (43) further studies upon the same types of bacilli dealt with in the paper just considered. From certain indications he believed it might be possible to differentiate between the two groups by cultural characteristics, as well as by the agglutination tests. The differences exhibited by growths upon ordinary culture media were too slight to be of service, but the different actions of the bacilli on various sugars gave some hope of finding a medium suitable for diagonstic purposes. This idea Lentz tested by growing the various types upon litmus agar containing 1.3 per cent. of one or other of the following substances—maltose, dulcite, dextrin, fructose, inulin, and mannite. He found the last—mannite litmus agar—would serve the purpose. Upon this medium the bacilli of Group 1 ("true dysentery") exerted no action: but the bacilli of Group 2 turned it violet red or red (acid); in the case of two pseudo-dysentery species, blue (alkaline). That is to say, with this medium the bacilli could be grouped in exactly the same way as resulted from the application of the agglutination test with specific (Shiga) serum. The other media were not suitable, because all (in the case of dulcite and inulin), or some (in the case of maltose, dextrose, fructose) of the bacilli of Group 2 behaved as did the bacilli of Group 1. The mannite medium, however, sharply marked off the two groups from one another. It is to be noted that the Flexner and Strong bacilli are again separated from the bacilli of "true dysentery." Both the Philippine strains behave similarly on mannite litmus agar—turn it red; but maltose litmus agar and dextrose litmus agar are turned red with the Flexner strain, but are left unchanged by Strong's bacillus; so that here again Flexner's strain is shown to be different from that of Strong. Lentz recommends mannite litmus agar as a simple and reliable medium for the determination of the bacilli of dysentery (44).

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The applicability of the two tests just mentioned, viz., agglutination with a high-value specific serum and the mannite re-action, has been investigated by various observers, with the result of confirming the contentions of Martini and Lentz. By their use J. W. H. Eyre (45) was also able to recognise two groups: Group 1 containing the Shiga, Kruse, and Flexner Newhaven bacilli, to which Eyre added a strain isolated by himself from asylum cases; whilst Group 2 contained Strong's Philippine bacillus, another Philippine strain isolated by Harris, and a strain isolated by Duval from infantile diarrhœa. Essentially the grouping is the same as that made by Martini and Lentz. To their Shiga group of bacilli from acute epidemic dysentery Eyre adds an asylum strain isolated by himself. In Group 2 he notes that Duval's bacillus from infantile diarrhœa is different from the bacilli of Strong and Harris, obtained from acute epidemic dysentery in the Philippines. Charles Todd (46) similarly places Shiga's, Kruse's, and Eyre's bacilli in one group, and Flexner's Philippine and Duval's bacilli in another. F. P. Gay (47), Philip Eisenberg (48), Karl Leiner (49), and others, report corresponding results. W. H. Park, Katherine H. Collins, and Mary E. Goodwin, working with these tests in the research laboratory of the Health Department of New York, arrive at classifying the various strains of bacilli into three groups as follow (50):—

I. Bacilli of the Shiga type. Produce little or no indol: do not ferment mannite, maltose, or saccharose. Animals immunised with them furnish agglutinins very active against the bacilli of this group, but little active against those of the other two groups.

II. Bacilli which produce indol: ferment mannite, but not maltose nor saccharose. Animals immunised with them furnish agglutinins specially active for this type, but active also against the bacilli of Group 3.

III. Bacilli of the Flexner Manila type. Produce indol: ferment mannite, maltose, and saccharose. Animals immunised with them furnish agglutinins specially active for the type, but active also against the bacilli of Group 2 (and against bacillus coli communis).

The bacilli of the first type are regarded as the true dysentery bacilli; they are responsible for the most severe dysenteries. The bacilli of Groups 2 and 3, although widespread and associated with characteristically dysenteric conditions, are less specific of this malady because found also in cases lacking dysenteric symptoms. The authors christen the bacilli of these groups "para-dysentery bacilli": they are related to bacillus coli communis.

In addition to the indications afforded by the tests just considered, similar group relationships are manifested by re-actions of other kinds. For instance, Todd has reported (51) that the toxins of the Shiga and Eyre bacilli were neutralised by a serum prepared by means of Kruse bacilli. His attempts to obtain toxins from Flexner's Philippine and Duval's bacilli were unsuccessful. Eisenberg, working with a Shiga serum, found that Shiga bacilli absorbed all the agglutinins therefrom, rendering the serum inactive against Flexner's bacillus. But Flexner's bacillus could not so absorb all the agglutinins: after treatment with this strain the serum was still agglutinative towards Shiga bacilli. R. H. Firth (52) appears to have determined a similar differentiation on the basis of pathogenesis; but his original paper is not available to us, and the reference note at our disposal is not sufficiently detailed to enable us to correlate Firth's results with those of the other authors mentioned.

These different investigations all converge to one issue, namely, that associated with cases having the characters of dysentery there are bacilli which fall into one or other of at least two groups. They belong either to

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the group ("true dysentery") of which Shiga's bacillus is the type, or to another group ("para-dysentery") the members of which show variations amongst themselves. The groups are differentiated one from the other by the results of agglutination with specific sera, and by the reaction with mannite, as well as by other means.

The bacilli which have been isolated from acute epidemic dysentery have belonged sometimes to the "true dysentery" group (Shiga, Kruse, Flexner-Newhaven, Pfuhl, Muller, &c.), and sometimes to the "para-dysentery" group (Flexner, Strong, and Harris Philippine strains). The bacilli which have been isolated from cases of asylum dysentery have belonged sometimes to the "true dysentery" group (Eyre), and sometimes to the "para-dysentery" group (Kruse). The bacilli which have been isolated from infantile diarrhœa have rarely belonged to the "true dysentery" group (Cordes (53), (Duval & Schorer) (54), more usually they have been "para-dysentery" bacilli (Martha Wollstein, Karl Leiner, Howland (55), Park, Collins & Goodwin, Cordes, Duval & Schorer).

Evidently it is necessary to abandon the idea that all bacillary dysenteries are caused by Shiga's bacillus. No doubt this species is responsible for a large number of cases of dysentery. It may even be justifiable to regard these cases as constituting a specific disease which various authors have called "true dysentery." From this point of view it might be separated off from other forms of dysentery, just as amœbic dysentery has been separated; but the factors available for discrimination are much less manifest. There is no present evidence that the symptoms, course, or pathological anatomy differ from those of other bacillary dysenteries, nor can a distinction be made on the basis of epidemiological prevalence. The distinguishing feature is the isolation and identification of Shiga's bacillus; and it would appear that even this may fail to decide the matter. Wm. H. Park and H. W. Carey, in connection with epidemic dysentery at Tuckahoe, north of New York, report (56) the isolation of a bacillus belonging to the Shiga-Kruse group. Neither it nor a type Shiga, agglutinated with Flexner (Baltimore) serum. The Flexner (Baltimore) bacillus with which the serum was prepared differs from Shiga in that it produces indol and ferments mannite; and is said to be identical with Flexner's Manila and American strains. The bacillus isolated did not do these things: it appeared to be a true Shiga bacillus. Nevertheless the blood serum of patients at Tuckahoe was found to agglutinate the Flexner Baltimore bacillus. From these results the authors are inclined to believe that, in the Tuckahoe outbreak, the Flexner strain was associated with the Shiga bacillus, although the latter alone was isolated. (But, as regards the validity of inferences drawn from the agglutination re-actions with patients' serum, see the views of Martini and Lentz, mentioned above.) It is to be noted that Duval and Schorer, in each of five cases, isolated both Shiga's bacillus and a "para-dysentery" bacillus. In another communication Duval and F. P. Gay report the simultaneous isolation of bacilli of both types from the intestine of a man dead of dysentery, and twice from the sanious stools of women who recovered. How far these complications can be resolved remains for the future to determine.

In the meantime it must be confessed that the etiological investigations hitherto made with respect to bacillary dysenteries, although they have added considerably to our knowledge of the subject, have not yielded such results as to clear away the lack of definition referred to at the beginning of this paper. Even if it be conceded that the disease caused by Shiga's bacillus is exclusively to be regarded as "true dysentery," the concession must be made with the reservation that a precisely similar complaint apparently can be produced by other species.

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SECTION OF STATE MEDICINE AND MEDICAL ETHICS.

PRESIDENTIAL ADDRESS.

By G. A. Syme, M.S., F.R.C.S., Eng.

Allow me to begin this Address with a grateful acknowledgment of the honor conferred by the Executive of the Congress in asking me to preside over this Section. I appreciate the compliment very highly, and the more because I feel it to be undeserved.

I accepted the position, however, very reluctantly, and with much misgiving. It seemed the irony of fate to preside over a Section the utility of which I much doubted. The meeting together of our members, from widely separated and different parts of the Commonwealth and New Zealand, certainly affords opportunity for comparison of differences in methods in State Medicine, and, as the first President of the Congress observed, in his address in this city eighteen years ago, for "making common property what suggestions relating to medical ethics or legal status arise out of our colonial life." But will the discussion of such matters *coram populo* promote, what I venture to hold ought to be the main object of these Congresses, the advancement of our profession as a science, not only among ourselves, but in the eyes of the public? We shall see.

At the first Congress, a section of State Medicine was instituted. Dr. Whittell, its President, said very truly in his address "that the term was an indefinite phrase, meaningless in itself, but which from usage is understood to express the action and duty of the State in relation to the physical welfare of its subjects." He did not say what he meant by State, but confined his attention to the duty of the State, evidently meaning the Government, in providing for the removal and prevention of disease. The term "Public Health" is now more generally used when speaking of this particular aspect of the subject, and in succeeding Congresses the title of the section was changed to "Public Health, including State Medicine," and sometimes other subjects, as Psychology and Forensic Medicine. Now we have two separate sections, one of "Public Health, &c.," and our own, called "State Medicine and Medical Ethics." I confess I have been somewhat puzzled to know what "State Medicine," as distinct from "Public Health," is supposed exactly to include. I propose to regard it as covering the relations of the State, *i.e.*, the body politic, both generally as the community, and particularly as the Government, to the medical profession, and its duty in caring for disease.

Prof. Metchnikoff, in that intensely interesting book, "The Nature of Man," has pointed out, more clearly and recently than others, that most of man's troubles and difficulties arise from mal-adjustment to changed environment, arising in the course of evolution. The medical profession is no exception. In its relations with the State, many such mal-adjustments exist. Many, probably the majority, of them arise from popular ignorance regarding the evolution of medicine as a science, popular misconceptions as to its true aims, and inability to appreciate both its immense potentialities and the nature of its limitations. In some instances, these mal-adjustments have become so great as to produce almost a war between the profession and

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certain sections of the community. We constantly read in our journals, *e.g.*, of the "Battle of the Clubs." I venture to think that the profession, instead of fighting certain sections in the State, and especially fighting them with their own weapons, should try to educate them; endeavor to make every member of the community realise that medicine is not a trade, whose business is the selling of charms and specifics, but a progressive science. When its true aims and aspirations are thoroughly "understanded of the people," the profession will be appraised at its due worth, and most of the grievances of its members, I believe, will be set right.

Educated people generally do appreciate us. R. L. Stevenson said of the physician—"He is the flower (such as it is) of our civilisation; and when that stage of man is done with, and only remembered to be marvelled at in history, he will be thought to have shared as little as any in the defects of the period, and most notably exhibited the virtues of the race. Generosity he has, such as is possible to those who practice an art, never to those who drive a trade; discretion, tested by a hundred secrets; tact, tried in a thousand embarrassments; and, what is more important, Herculean cheerfulness and courage."

When such an appraisement becomes universal on the part of the public, and is universally deserved by the profession, there will not be many maladjustments to rectify. In the meantime they exist, and afford matter for discussion in this Section. What I fear is, that the trade aspects of the questions brought before us may occupy too much attention. I hope that our discussions may be of educational value, to the public as well as to ourselves.

Let us now look a little more in detail at some of these relations between the State and the Medical Profession.

In Great Britain, the relationship starts at the very commencement of a medical student's career. The State, as Government, through the powers entrusted to the General Medical Council, determines the course of study he is to pursue. The Government does not, however, assist very materially in directly providing for his medical education. In Australia, the position is reversed. The Governments of New South Wales, Victoria, and South Australia contribute very largely to the support of the University Medical Schools, but do not interfere with the course of study. The Government does not, in any State, come into direct relation with the profession until the possessor of a medical diploma wishes to be registered as legally qualified to practise. The requirements for registration vary greatly in the different States of the Commonwealth. It is highly desirable that they should be made uniform, and that the portals to the profession should be the same in every part of Australia. Something has been done in this direction in recent years by the amendments of the Medical Acts passed in New South Wales and South Australia. It is surely an anomaly that while Australians, to obtain a qualification in their own country, must pass through a five years' course at their own Universities, in Victoria, Western Australia, and Queensland, anyone who has passed through a regular course of medical study of not less than three years in a foreign school of medicine, and received a diploma, can be registered. In South Australia the course must be four years; in New South Wales only it must be five years. The Tasmanian Act, though passed in 1842, is very effective for preventing unqualified practice, and is the only Act that is. It expressly says, "No person shall practise as physician, surgeon, or apothecary, or prescribe or do any surgical act," unless certificated by the Board. Another serious defect in the Victorian Act is that, once a person's name is on the Register, it cannot be removed, although the body that gave him the diploma under which he registered may have taken it away for

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misconduct. The State Premiers, at their next conference, might well consider the advisability of making the various Medical Acts uniform. In any new legislation, it should be provided that qualifications that are not registerable in Great Britain should not be recognised in Australia. It has been suggested that medical registration should be a function of the Commonwealth Government, with a Federal Medical Council for Australia, similar to the General Medical Council of Great Britain. Owing to the great distances between the different centres of the Commonwealth, such an arrangement would probably prove expensive and inconvenient.

Whether the requirements for registration as medical practitioners should be materially altered is a matter for grave consideration. The fulfilment of those now in force in most countries ought to be a guarantee to the public of theoretical knowledge, but is not something more necessary—some more satisfactory guarantee of competence for actual practice? In that interesting, if morbid and exaggerated, book, "The Confessions of a Physician," the Russian author describes very graphically his despair on first entering into actual practice, and the pitfalls into which his "abstract bookish wisdom" led him, owing to want of practical experience. So great was his mental distress, that he threw up his practice and returned to St. Petersburg for further practical work, quoting Billroth—"That only a doctor without a drop of conscience can permit himself, without more ado, to make use of the rights which his diploma gives him."

Some years ago, the General Medical Council of Great Britain recognised that further requirements were necessary. The Council imposed a fifth year of study, recommended to the licensing bodies that it should be devoted entirely to clinical work, and provided that six months of it *may* be passed as a pupil to a registered practitioner. Is this enough? Judging from my own experience, I should say that it is not. I was foolish enough, when just graduated, to take a position as *locum tenens* for a practitioner who had been suddenly taken ill. He had a very large general practice, with numerous lodges. My experiences were not so tragic as those of the Russian physician referred to, but they were extremely disheartening. I was quite satisfied that, in spite of my legal status, I was *not* qualified to practise, nor did I attempt to do so till I had done six years' additional work, holding various subordinate hospital appointments, in which I could acquire practical knowledge, technical skill, and a feeling of confidence. It seems to me a question for very serious consideration whether practitioners should be registered unless they can show evidence of practical work, either as resident medical officer, or pupil at a hospital, or as assistant to a medical man in practice, subsequent to the taking of a diploma. The difficulty is to obtain such positions, as the Russian writer of the "Confessions" found to his bitter sorrow. Much could be done if every hospital in Australia appointed Resident Medical Officers annually, and reserved the positions for recent graduates of our own Universities. At the metropolitan hospitals, also, provision might be made for resident senior pupils, as at the Johns Hopkins' Hospital.

Medical Acts, all the world over, have been passed, not in the interests of the medical profession, but for the purpose of protecting the public, and enabling them to distinguish between those who are regarded by the State as properly qualified, and those who are not. A considerable number of medical practitioners thinks that Legislative action should be taken for the repression of unqualified practice altogether. Most of those who clamor for the legal restriction of quacks do so openly in the interests of the profession, as much as in the interests of the public. I venture to take a different view. Personally, I do not believe that quackery does the slightest harm to the medical profession, from a pecuniary point of view. But that is not the important

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question. What is requisite is that the public should be better informed on these matters, and more will be done by educating the people than by legal enactment. People must be made to realise both the limitations and possibilities of treatment, and must get rid of their antiquated and superstitious notions of the action of drugs, too many of which, I regret to say, are too often supported by some of our own profession. Much could be done by making public the real composition of quack preparations, and here legislation can be of service. The mysterious always appeals to the ignorant. Even the cultured can be attracted by artistic mysticism, as evidenced by the vogue of Maeterlinck, the so-called Shakespeare of Belgium. Take away the cloak of secrecy from quack medicines; let it be known that so-and-so's "Wizard Oil" is only turpentine, camphor, and ammonia; that somebody else's "Soothing Syrup" is composed of morphia, with essence of anise and balsam of tolu, and the occupation of their manufacturers would be, like Othello's, gone. Hence the angry commercial protest against recent legislation in New Zealand on the subject of patent and proprietary medicines. The New Zealand Parliament passed an Act, under which regulations were framed making it compulsory that the formula of all patent and proprietary medicines should be printed on the bottle, and that if any poison were contained in the preparation it must be labelled accordingly. The manufacturers of patent medicines, and their agents, raised such powerful protests, and brought such influence to bear, that the regulations were modified, to the extent that the formula, instead of being placed on the bottle, was to be lodged with the Health Department. The manufacturers still objected, and, as no further alterations in the regulations could be obtained, they decided not to send their medicines to New Zealand. Evidently they realise very clearly that, once the real nature and commercial value of their preparations became publicly known, they would have no demand at the extravagant prices at which they had hitherto been sold.

Having been registered, the legally qualified medical practitioner comes into relation with State authority in various ways. He is bound to attend a summons from a coroner to attend and give evidence at inquests, and to make *post-mortem* examinations. A coroner is invested by the State with very plenary powers, and the discretion with which he exercises them may sometimes be called in question, especially by the medical profession. It has been much debated, *e.g.*, whether he should instruct the nearest practitioner, or a pathologist, with special experience, to make *post-mortem* examinations. As a general rule, I think it is unquestionably best, both in the interests of the public and of the profession, that such examinations should be made by skilled pathologists. The coroner, however, should be very careful that, consistently with the safety of the public, the reputation and interests of general practitioners giving evidence, or connected with the case, are protected. The main object of the inquiry is to ascertain the mode of death, and for this the medical evidence is generally all-important. If the coroner, by his attitude to medical witnesses, practically closes their mouths, as happened recently in Melbourne, he may be unable to ascertain the exact cause and mode of death.

The Coroner's Acts in Australia have been copied from the English Act, without taking account of altered conditions. Thus, if the practitioner reside more than ten miles from the place where the inquest is held, he can only be paid 1s. a mile for every mile of such extra distance. In the scattered districts in the interior of Australia this arrrangement, probably fairly equitable in England, is very unfair. Cases have been brought under the notice of the Medical Defence Association of Victoria where men have had to travel long distances, involving absence from practice of two days and nights, with

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attendant expenses, for which the payment authorised by the Act was very inadequate. Even in England complaint is made as to the inadequacy of fees to medical witnesses, and some time ago a joint deputation from the British Medical Association and the Police Surgeons' Association memorialised the Home Secretary, asking that the fees for medical witnesses should be raised. The application was not granted. In almost all cases where the Government demands service from medical practitioners it pays for them very poorly. The fees for vaccination and notification of infectious disease are ridiculously small. In Victoria, the post of Government Vaccinator in country districts entails a good deal of badly paid work, inasmuch as the vaccinator has to arrange beforehand to visit certain distant townships on a fixed day. He then probably has only a very few cases to vaccinate at the ordinary fee, for which he has had to sacrifice perhaps a whole day's remunerative work. As it is not compulsory for men to take these appointments, a little combination among medical practitioners in a district would probably compel the Government to pay more adequately in special cases.

Let us next consider the duties and responsibilities of the State in caring for disease. Here we find the greatest diversity of procedure, not only in Australia, but all over the world. In all the States of the Commonwealth, the Government contributes largely to the support of public hospitals, and often provides some hospitals entirely. The amount contributed by the charitable public, and the degree to which the Government can interfere in the management, vary, not only in each State, but in each hospital. Here, in South Australia, I understand, the Government practically maintains all the hospitals, and, if it chooses, can exercise control over the management, as notoriously exemplified some years ago in the appointment of Drs. Leith Napier and Ramsay Smith to the Adelaide Hospital. In New South Wales, the Government has contributed very largely to both the building and maintenance of hospitals, and public subscriptions have not been at all proportionate. Last year, *e.g.*, in Sydney, the Royal Prince Alfred, Sydney, and Children's Hospitals cost £49,236 for maintenance, towards which the Government contributed £31,781, and the public subscribed £9,282. In addition, the Coast Hospital, entirely supported by Government, cost £20,130. It is probable, however, that the Government grant to hospitals in New South Wales will be much reduced in future. In Victoria, Government and public contribute about equally. The management is practically free from Government control.

When Prof. Senn, of Chicago, was visiting us a little time ago, he said to me—"You have far too many hospitals and charitable institutions in Australia, in proportion to your population." His observation was correct. According to Mr. Bruck, there was one hospital to every 12,299 people in Australia in 1899. Many of these institutions overlap in their work, and money is wasted in expenses of management and collection of subscriptions. Population is unsettled and shifting, and hospitals established in country towns, especially near goldfields, when they were thriving, are still maintained, though the population has left, and the hospitals are not really necessary. To quote Mr. Bruck again, the total number of beds available was 11,280, but the average number of in-patients a day was only 7,809. It is evident that some central board or authority is required, in each State, to control all the hospitals, and co-ordinate their functions. On this central board, and also on the boards of management of the individual hospitals, the medical profession should be represented. The executive officer of the Central Board, also, should preferably be a medical man.

In providing for the care of disease, it seems to me that it is the duty of the State, using the word in its widest sense, to furnish free accommodation

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and treatment for the destitute sick, but for no others. It is not necessary, however, to provide palatial buildings, luxurious comforts, and the attendance, in an honorary capacity, of the most eminent men in the profession. To a large extent it is the very excellence of the public hospitals that causes them to be abused.

If we glance "into the dark backward and abysm of time," we see that the present system of State care for the destitute sick has not always existed. In pre-Christian Rome, the sick poor were attended by the public medical officers, who were appointed for every district in the city. They were well paid, and also enjoyed many immunities. In ancient Arabia, and other Mohammedan countries, hospitals, with paid physicians, were established by various rulers. With the advent of Christianity, the care of the sick became a religious duty, and hospitals became religious institutions, attached to monasteries, or founded by priests. St. Bartholomew's, the oldest hospital in England, was founded by Rahere, first Prior of the convent of Augustinian canons, and in charge of a master, brothers, and sisters. St. Thomas' was founded by the canons of St. Mary Ovaries, and held by the Prior of Bermondsey. The monks and priests were the first physicians at these hospitals, and naturally gave their medical services gratuitously. It might be imagined that out of this custom grew the practice of honorary medical service, when physicians and surgeons came to be appointed to these hospitals. But it appears it was not so. The first physicians and surgeons at both St. Bartholomew's and St. Thomas' were paid. The immortal Harvey was a paid physician at St. Bartholomew's. So late as 1738, a writer, describing St. Thomas', says that the physicians and surgeons had salaries, "as well as perquisites considerably better than the salary."

When and how these positions became honorary I have not been able to ascertain. St. George's Hospital, founded in 1733, was the first in England to be established and supported entirely by voluntary subscriptions, and its first staff was elected by a meeting of the subscribers. It consisted of six physicians and three surgeons, all of whom "had declared their willingness to serve without fee or reward." They were, however, to be "permitted to take pupils," which possibly explains the willingness to serve without payment. The need of clinical teaching in the training of medical men became recognised very gradually, being first properly instituted in Leyden, in the 17th century. For some time before this, certainly in 1561, the physicians and surgeons at the great London hospitals, St. Bartholomew's, St. Thomas', and Guy's, took pupils or apprentices, who paid large fees, and were instructed at the hospitals. The position of physician and surgeon at these hospitals thus became very valuable and lucrative, both directly and indirectly, and was much sought after. The profession is apt to take credit for the large amount of gratuitous work done by its members, and rightly. But, so far as large clinical hospitals are concerned, there is a good deal of cant about the noble generosity of the honorary staff in giving its highly skilled services to the suffering poor gratuitously. To men who desire to become and remain leaders of the profession, positions on the honorary staff of a clinical hospital are almost indispensable, and are accordingly keenly competed for and much coveted.

Unfortunately, the custom of honorary service has spread from large clinical to nearly all hospitals, and the amount of gratuitous work done by the profession, in British communities, is very great. In my opinion, it should be very much restricted. At clinical hospitals, it is necessary to have a staff of eminent men as teachers, who should be adequately paid for teaching. All the beds in such hospitals should be used solely by destitute patients, who, by providing clinical material, give all they can. In all other hospitals

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the medical staff should be paid. Burdett, in his great work on hospitals, says—"We believe, as the result of prolonged investigation, an intimate knowledge of the facts, and great practical experience, that if the voluntary hospitals were to determine that every medical officer, both resident and non-resident, should henceforth be paid for his services, the cost per bed at every institution which thus asserted its independence would be considerably reduced. The medical profession has rendered a great deal too much eleemosynary service in the past." The ideal arrangement, to my mind, is such as obtains at the Johns Hopkins' Hospital, Baltimore. There the endowment is sufficient to provide handsome salaries for all the staff, who are also teachers in the Johns Hopkins' University. In no hospital in the world is better work done.

Besides hospitals for the really destitute, paying hospitals are required for the poorer middle class, who cannot afford ordinary private hospital charges, together with the fees of leading members of the profession. A great deal might be done by the Friendly Societies, as I suggested years ago in the leading columns of the *Australian Medical Journal*. They could establish hospitals for their members, where they would be attended by their own lodge surgeons. When required, the services of a paid consulting staff should be available, just as now lodge members obtain the services of specialists. This consulting staff might be composed of young men, not yet in the height of their fame, perhaps holding junior positions on the staff of a clinical hospital.

Most American hospitals have paying wards. When a hospital, like the Johns Hopkins', is founded and maintained entirely by private benevolence, and when its staff is paid, I see no objection to its having paying wards. Nor can there be much objection, under other conditions, if the paying patients are permitted to select their own medical attendants, whether on the staff of the hospital or not. But to have paying wards attached to a public hospital, subsidised by Government, and restrict the paying patients to the attendance of the staff, is very unfair alike to the patients, the outside profession, and those who keep private hospitals. I have had personal experience of the difficult situations such a condition creates, as a member of the staff of the St. Vincent's Hospital, Melbourne, when it used to take paying patients. I strongly opposed the practice, which, I am glad to say, was given up.

While on this subject, I would like to say a few words on a rather delicate question, viz., whether private hospitals ought to be conducted by medical men? I hold they ought not. It seems to me that the more all members of the profession keep to the "practising of an art," and away from "the driving of a trade," the better. Conducting a private hospital is purely a trade. Plenty of capable non-professional people are willing to carry it on properly, and to them it should be left. It is a trade which involves its driver in many petty annoyances and worries, which must distract a professional man, and interfere with his whole-hearted attention to his proper work, which is sufficiently exacting and anxious as it is. The more the medical profession presents its commercial side to the public, the more it loses in dignity and esteem, and the more difficult is it to induce the public to worthily appraise its higher aims. Further, a private hospital must be, more or less, a continual temptation to the medical conductor. It is so easy to make it a means of advertising, and it has to be kept filled.

Gratuitous attendance is another unsettled question, concerning which every member of the profession is a "law unto himself." It is desirable that some uniformity of practice should be established. It is a general custom not to charge fees to medical men, but Dr. Saundby says—"Medical practitioners have no right to the gratuitous services of their colleagues." To

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what relations of a medical man should gratuitous attendance be extended? I would say only to those actually and absolutely dependent on him, and living in his house. What about the families of deceased practitioners? There seems to be no general rule. Each case must be treated on its merits. I was once adversely criticised for charging the widow of a medical man. She was living with, and apparently dependent on, her son, who was in a good position, and I did not even know at the time that her husband, long dead, had been in the profession. It is customary to see medical students and nurses without fee; some extend the custom to dentists and chemists, but I don't see why, and don't think it ought to be done. In fact, it would be more satisfactory to everyone if gratuitous attendance were limited as much as possible. As Saundby say—"Medical practitioners should only forego their fees to those persons who are unable to pay, and should refuse to recognise any other grounds for the concession. The clergy, as a class, have no right to medical attendance without payment." Dr. De Styrap devotes several pages of his "Code of Medical Ethics" to justifying the position that "gratuitous attendance on ministers of religion and their families should no longer obtain." The profession, however, is not by any means unanimous on the question. Some do, and some do not, charge clergymen. My own practice, in the past, has been not to charge. I have lately come to the opinion that it is right to adopt Saundby's principle, and only remit fees to the clergy when they are obviously unable to pay.

If our scales of fees were more elastic, and the minimum fees less, it would be much easier to make it the rule to charge everyone. The rates adopted in the scales in use in New South Wales, Victoria, South Australia, and Queensland are very different, and it is desirable that they should be made more uniform. Thus, in Victoria, the minimum fee for a visit or advice at the house is 10s. 6d. In New South Wales, South Australia, and Queensland a minimum fee of 5s. is permitted, and in New South Wales, for what is called a second class visit in town, 2s. 6d. Discrepancies occur, also, in the fees for operations. Any scale of fees must be necessarily elastic, and absolute uniformity is not to be expected. The scale adopted in New South Wales appears to me to be satisfactory, and the other States might very well come into line with it, at all events as regards the minimum. It may be interesting to note that the first Medical Society in Victoria—the Port Phillip Medical Association—in September, 1846, drew up a scale of fees for first, second, and third class patients ranging from 10s. 6d. to 2s. 6d. I have no hesitation in saying that fees will have to be lowered, especially in Victoria, and particularly the ordinary fee for general work. As my friend and colleague, Dr. Howard, pointed out, in his presidential address to the Medical Society of Victoria, at the beginning of the year, disease is less prevalent, population is increasing slowly (2 per cent. in ten years, while the medical profession has increased 20 per cent. in the same time), and the number of patients attending hospitals has increased (probably 20, or even 30 per cent.). Under such conditions the incomes of the profession must suffer, and one remedy is a reduction of fees. Had fees been lower, and hospital accommodation not so lavish, many of those attending hospitals would have been paying fees to practitioners as private patients.

Private patients! Now we come to the relations of the profession to the individuals of the State, and of its members to each other—that is, to questions of medical ethics.

It is commonly held that medical like all other questions of ethics can be summed up by saying—"Whatsoever ye would that men should do to you, do ye even so to them." While it is true that all professional conduct should conform to this golden rule, society is so complex, and its members so varied,

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that different people may hold conflicting opinions as to "what men should do to them." Some generally recognised rules of conduct in professional relations are, therefore, desirable. Such rules, however, should be as general and as few as possible. Detailed complicated codes of medical ethics are, in my opinion, a mistake. The more stringent the code, the less likely is it to be carried out. The most ancient code of which we have any knowledge—that of Hamurabi, 1350 B.C.—was exceedingly drastic, and, if carried into effect, which it probably was not, would have almost prevented the practice of surgery. It is generally recognised that the more laws are minutely coded, as in Rome and France, the less is progress possible; while in England, where there is no code, but Equity Courts exist, most advance has been made. Law and ethics, like everything else, must be modified with changed conditions and the gradual evolution of society. As regards medical ethics, it is necessary that every member of the profession should be informed as to generally recognised customs, or conventions, in his relations with his patients and his fellow practitioners. Very often so called breaches of medical ethics are committed in ignorance. It would be well that medical students should receive instruction on the subject. It is also highly desirable that the laity should be more familiar than it is with the general principles of medical ethics. The public is too apt to think that what it calls medical "etiquette" is some mysterious system for the protection of the profession, with peculiar laws, differing altogether from the principles which regulate the conduct of ordinary people. It cannot be too strongly enforced that the ethical conventions of the medical profession are quite as much in the interests of the patient as the practitioner, and are simply intended to promote fair and honorable dealing.

Unfortunately it is to be feared that some members of the profession are to a certain extent responsible for the view of the public, and hold what I cannot regard but as mistaken opinions as to the relations of practitioner and patient. One of these is the idea that a medical practitioner has a kind of proprietary right to a patient. We all recognise that it is to the patient's interest that a medical man who has attended a patient for some time, and knows his personal idiosyncrasies, and the various affections he may have had, knows his "constitution," as it is popularly termed, should continue to attend, and, if further opinion is desired, should, when possible, meet the consultant, and give all information. Again, it is not to the patient's interest that he should be attended by more than one medical man for the same illness and at the same time, except in consultation. Accordingly it is a rule of medical ethics that a medical practitioner shall not see a patient, wittingly, who is under the actual care of another practitioner, except in consultation, or in a sudden emergency. But such a rule does not imply that, once a medical man has seen a patient, that individual becomes as it were his commercial property, and that neither the individual nor any member of his family must ever consult any other practitioner, except with him, or on his advice. I have here two letters, bearing on these points, from general practitioners in the country to a leading consulting physician in Melbourne. In one, complaint is made because the consultant had seen a patient, who had at some previous time been under the care of the country practitioner, in a local hospital, and given a different opinion without communicating with the man in the country. The other complaint is that the consultant had seen a patient, who had been previously under the care of the country practitioner, as a lodge patient, and had not only seen him, but requested him to further communicate with the consultant by letter. I will quote the actual words used by this practitioner:—"The principle is a very wrong one which allows patients to see consultants without the advice of their regular medical attendant." . . "I understand now that a consultant is a man who consults with the patient, and not with that patient's ordinary medical man." The writer further accuses metropolitan

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consultants generally of "seeking to get our patients." Another instance that came under my own observation recently may be given. A patient saw a consulting physician at his consulting rooms. After investigation, he advised her to go home, go to bed, and get a nurse. Finding she had some time previously seen a local practitioner, he arranged to meet him in consultation at her house, and leave the case in his hands. They met, but the local man was evidently annoyed, and demurred to going on with the case. He considered that his patient had no right to go to anyone unless he sent her, and that the physician had no right to see her and act as he had. I maintain that the attitude of these general practitioners is based on a misapprehension of the ethical rule previously mentioned. The first consideration must be the welfare of the patient. The general practitioner has no proprietary right in what he calls *his* patients. The patient has a perfect right to get any opinion he pleases. It may be that what he calls an "independent" opinion may not be so valuable as one obtained after a regular consultation with the previous attendant, who may give information considerably modifying the consultant's view of the case. On the other hand, a consultant may occasionally be unconsciously biased by what he is told.

While a patient has a perfect right to change his medical attendant, there are circumstances which debar certain practitioners from attendance. The ethical rule in such cases is put clearly and briefly by Saundby:—"No one is at liberty to take over the treatment of a case to which he has been called in consultation, or where he has acted as a substitute for the ordinary medical attendant." While preparing this address, my advice was asked about this very matter. *A* was away on a holiday, and arranged with a neighboring practitioner, *B*, to see patients for him. *A* had previously attended the household of *C*. The wife of *C* sent for *A*, and *A's* wife asked *B* to go as *A's* substitute. After *A* had resumed practice, *B* was asked by the lady to attend her. He seemed to think he would be justified in doing so, as *A* had never actually attended her. I advised him to explain the ethics of the case to the lady, and suggest that she should get *A*. He did so. She still wished *B* to attend, and he seemed to think he had no option but to do so. I think he had, and could have declined to attend, because he acted in the first instance as a substitute. The patient had the option of sending for *B* in the first instance, but did not do so. She sent for *A*, and but for *A's* introduction, *B* might never have been called in. Dr. Roderick Maclaren recently gave an address on the relations of medical men to each other, in which he remarks, "We are too chary of exercising our right to select our patients, which is just as definite as a patient's to select his medical attendant . . . . It happens to most men, when acting for another, to be asked to continue an attendance for themselves; to do so would be a breach of good faith towards the man for whom he acts, who has trusted him as a friend to take his work, or otherwise help him. A friend cannot honorably supplant him." In short, it is not a question of medical ethics, or the interests of the patient, but of general ethics, of what is fair and honorable between man and man. Indirectly, it may affect the patient. When *A* or *B* next go away, each will probably employ a paid *locum tenens*, in whom the patient may not have the same confidence as he has in either *A* or *B*.

Not only has a practitioner a right to select his patient, but also to select his consultant, or, rather, to refuse to meet a particular consultant. It is a right which should be seldom exercised; but I agree with Dr. Maclaren when he says, "It is not difficult to conceive of circumstances in which the request to meet some individual amounts to an insult, and it should be dealt with as any such occurrence would be in ordinary social life." It is generally accepted in this country that homœopaths should not be met in consultation. According to Saundby, it is the custom in England for consulting surgeons to meet

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homœopaths. He also raises the question whether the profession should not reconsider its attitude to homœopaths. He suggests that "boycotting for differences of opinion is unworthy of a profession based on natural science, in which conflicts of opinion are the inevitable condition of progress." I take it, however, that the objection to meeting homœopaths is based on ethical grounds —not on differences of opinion. It is because no man who regards medicine as a science, and has grasped its scientific principles, can honestly say he believes in the principles enunciated by Hahnemann. Huxley, in his Lectures on Evolution, said—"Scientific men get an awkward habit—no, I won't call it that, for it is a valuable habit—of believing nothing unless there is evidence for it; and they have a way of looking upon belief which is not based upon evidence, not only as illogical, but as immoral." Homœopathy is, as the Royal College of Surgeons of Ireland states in its by-laws, a deception. A practitioner can only assume the designation of homœopath for reasons which are, therefore, unprofessional, unethical, and immoral.

Professional secrecy may, at times, lead to ethical difficulties in practice. As a rule, the obligation of the ancient Hippocratic oath is still binding—"Whatever in connection with my professional practice, or not in connection with it, I see or hear in the life of men, which ought not to be spoken abroad, I will not divulge, as reckoning that all such should be kept secret." The law as to professional secrecy is most stringent in France, and there a medical man is in no difficulty as to his position. Under no circumstances, not even as a witness in a criminal case, can he reveal a professional secret. The only exception is when a crime is about to be committed, and when, by keeping silence, the medical man might make himself an accessory. When a crime has been committed, a medical man must not give information, if, in so doing, he reveals a professional secret, not even when an innocent person is wrongfully accused. In great Britain there is no hard and fast law. While the Hippocratic oath is binding in ordinary circumstances, a medical man can be compelled to reveal professional secrets in a court of law. It was laid down by Mr. Justice Hawkins, in the case of *Kitson* v. *Playfair*, that no medical practitioner may state facts which have come to his knowledge in the course of medical examination without the consent of the patient, except in a court of justice, and under the direction of the judge, who has to decide each case on its merits. He is supposed not to allow unnecessary disclosures, but to see that no material facts which can assist the court in arriving at the truth are withheld. In Australia, the law of evidence in regard to professional secrecy differs in different States. In New South Wales and Tasmania it is the same as in England—the medical man is not protected. In Victoria he must give evidence in criminal cases, but shall not in any others. Section 47 of the "Evidence Act" reads—"No physician or surgeon shall, without the consent of his patient, divulge in any civil suit or proceedings (unless the sanity of the patient be the matter in dispute) any information which he may have acquired in attending the patient, and which was necessary to enable him to prescribe or act for the patient." While this is the law, and notwithstanding the Hippocratic oath, occasions arise in which, I think, one is justified in taking all risks and communicating professional information concerning patients. This is especially so with regard to communicable disease. Some time ago a lady saw me on what she described as a delicate mission. A patient under my care was engaged to be married to a friend, who was an orphan. The lady believed this man to be dissolute, and suspected that he was being attended by me for a communicable disease. Would I tell her? I declined, on the ground of professional secrecy. I sent for the patient, and told him he must break off his engagement. He declined. I then said I must advise the girl's friends not to let her marry him. He objected to my doing so. However, I took the risk. I told the lady that, while I could not reveal the nature of the disease from which my patient

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suffered, I could tell her that he ought not to marry. Even in France, eminent authorities on legal medicine and ethics, as Tardieu and Garde, say that, in similar circumstances, "conscience must be heard above law."

I had intended to refer to some other matters, such as the relations of the profession to the public press, and also to the friendly societies. I fear, however, that I have overtaxed your patience already. I will, therefore, conclude by reminding you that, while the profession undoubtedly has many just grievances, there ought to be manifold compensations, at least to those of its members who "have a right spirit within them." Let me remind you also that much has been done to remedy these grievances by proper organisation, and more can yet be done. Our brethren in New South Wales have shown us the way, and it is to be hoped that the organisation of the whole profession, so satisfactorily achieved in New South Wales, may be extended throughout Australasia. It is to be hoped, further, that its aim will not be simply the selfish one of the material improvement of the profession, but will be primarily to promote and maintain the best traditions of the past, and to afford inspiration for scientific progress in the future. Let us not forget, amidst all our failures and disappointments, ethical and otherwise, that, in the eloquent words of Sir James Paget, our profession unites "those three qualities which have the greatest charm for pure and active minds—novelty, utility, charity," and that we can compete in the world, not for wealth and position, but "in the nobler ambition of being counted among the learned and the good, who strive to make the future better and happier than the past."

Now I am at last ended. You may probably say—"There needs no visitor come from distant State to tell us this, such poor obvious commonplace." I can only reply, like Hamlet—

> "Why, right; you are i' the right;
> And so, without more circumstance at all,
> I hold it fit, that we shake hands, and part."

FRIENDLY SOCIETIES IN NAPIER, NEW ZEALAND.

By T. C. Moore, M.D., Univ. Dublin, Napier, New Zealand.

I have been asked by Dr. A. A. Hamilton to give some information to the Congress with regard to club practice in New Zealand. I am afraid I can only speak for my own immediate district. For some years I have done very little of this class of work, only holding one lodge, of the Druid order, for which I am paid 16s. per member per annum, without medicine. None of the members of this lodge are in such circumstances that they could afford to pay private fees. They are charged, also, 5s. a mile over three miles if they live beyond the three-mile limit—for these mileage payments the lodge is not responsible—also the ordinary fees for confinements and anæsthetics. All difficult operations are performed in the public hospital, to which the lodge subscribes £5 a year. All the other town lodges are held on much the same terms. The Oddfellows, M.U., many of whom are in good positions, and unfitted to be in a benefit society, pay their doctors £1 a year for attendance, and are dear at the price, in my opinion. There is little competition for lodges here, and the medical men who attend Foresters' lodges have lately raised their terms to £1 a year. The other small lodges pay 15s. a year. There is a dispensary here, started by the friendly societies, where they procure their medicines. There are country lodges at the villages around, which have various agreements, the usual one being 5s. a visit, in addition to a retaining fee of £1 a year.

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HOSPITAL ABUSE.

By H. W. Bryant, L.R.C.P. and S., Edin., &c.

In the report of the Inspector of Charities of Victoria, ending June 30th, 1904, he states that "Greater care is now being exercised by some of the managing bodies to see that patients who are suitable cases for hospital treatment contribute according to their means."

Now there is no doubt that this is so, for I am emphatically informed that at the Melbourne Hospital wardsmen and nurses are specially instructed to see that both in-patients and out-patients should pay something towards the upkeep of the hospital; and the very people for whom the charity should be available are the very ones that this system presses upon most harshly.

The other type, who are only too willing to pay a small sum, flood the hospitals, and easily obtain admission and gratuitous medical attendance, to the great detriment of the poorer classes.

The inspector says "that from the declared particulars furnished by each applicant for treatment, the officials of the institution should be in a position to determine whether such applicant is a proper case for treatment, and too much care cannot be taken against imposition."

I could give you case after case of imposition on every hospital about Melbourne that has come to my knowledge during the last two years. Perhaps it would not be amiss to specify a few instances, for the benefit of the profession generally:—

A well-to-do tradesman, worth at least £5,000, sent his wife to the lodge doctor (A.N.A.) for a note to take his child to a hospital in order to have post-nasal growths removed. The doctor, on making inquiries, found that the lady had already been to the hospital, and the Resident, finding the people were on a lodge, refused to treat the child without a letter from the lodge doctor. The lodge doctor said to the lady, "Do you understand that this hospital is only intended for poor people?" and she replied, "Mrs. So-and-so, who is quite as well off as we are, had her child operated upon there, and I don't see why my child should not receive attention there also." These people were very indignant at being refused a letter, and finally got the child operated upon at that hospital by subterfuge and lying.

Dr. J. P. Ryan, in his presidential address to the Medical Society of Victoria, published January 20th, 1903, in the *Intercolonial Medical Journal*, drew attention to the number of hospitals, &c., in the State receiving Government and public aid, and I think it worthy to note that for the year ending June 30th, 1904, the Inspector of Charities for the State of Victoria gives the following list:—

Hospitals (special)	41
Convalescent homes	2
Hospitals and benevolent asylums	9
Benevolent asylums	6
Orphan asylums	7
Other asylums	3
Refuge and rescue homes	10
Ladies' benevolent asylums	70
Medical dispensaries	2
Philanthropic associations	14

All of these are receiving Government grants, besides public subscriptions and donations.

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Dr. J. P. Ryan also drew attention to the number of benefit societies in every town in the State, whose members obtain medicine and medical attendance for their families and themselves for a very small sum.

Dr. Barrett gave the result of his researches into this question some time back, and he came to the conclusion, from carefully worked out statistics, that our population was being pauperised. Our hospitals were being used by patients who were quite capable of paying for their medical attendance, but who would not do so when they could get it for nothing. He considered that there was no necessity to increase the hospital accommodation, which was quite large enough in comparison to the population, but rather to look more closely into the position of the patients applying for charity.

There is a general feeling at the present time amongst the profession that something should be done to try and stop the impositions that are being made upon our hospitals.

Many of us have been consulted by well-to-do people with regard to some operation, and when told that it would cost, say, £10, or £15, which they could easily afford, they have gone to one of the large hospitals and been operated upon by one of our leading surgeons for nothing. The same surgeon would probably charge from £30 to £50 for the same operation outside.

We have all been told of cases by the patients themselves where they have gone to the paying ward of one of the hospitals, and have, by paying a small sum for their maintenance, obtained gratuitous nursing and medical attendance. They have afterwards expressed the opinion that, having given a small sum to the funds of the hospital, they were under no further compliment or obligation to that institution or its staff, and they feel they have highly honored both by their patronage.

The general public are thus becoming more pauperised every year, and it has been the feeling of many medical men for some time past that some great effort must be made to bring matters to a crisis, in order that they may obtain a little fairer treatment for the future. The general practitioner knows that small operations, such as the removal of post-nasal growths, curettes, &c., are performed in the hospitals for patients who are too mean to pay for them privately. There is no doubt that such cases should not be attended at hospitals, unless the subscriber sending them in, or the general practitioner, can positively state by letter that they are destitute. This would save a lot of the dissatisfaction at present felt by general practitioners, and would also be a great saving of the voluntary contributions which are given in the belief that only the poor are benefited.

It is quite time the committees of the hospitals took more active means than are at present adopted to curtail their expenditure and improve their positions and efficiency.

By properly investigating into the positions of people who are attending at the hospitals, a great saving might be made, and the legitimate poor get their proper share of attention; but frequently we hear that the hospitals are overcrowded, and poor people have to be carted about from place to place before they are admitted. Then we hear that a member of Parliament is found by a policeman close to his home, suffering from a broken leg, &c., and instead of taking him to his home, and sending for a doctor and a nurse, or even taking him to a private hospital (of which there are a plentiful supply in all the suburbs of Melbourne), he is taken to a place where only the poor are supposed to be attended, and kept there until his leg has knitted.

I should not object so much if he were temporarily attended to, and then sent to his home or to a private hospital; but no, he is kept there until he is quite fit to be moving about again.

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Another common practice is much to be deplored, viz., the sending of crushed hands and other injuries occurring at big factories and Government workshops to the nearest hospital.

In these days of telephones, it is always easy to obtain a medical man at very short notice, and I consider that a first aid should be provided for at all big institutions, and paid for by a fund initiated for such purposes.

The medical man called in to such a case could then inquire into the circumstances of the individual, and, if poor, he could go to the hospital, but if in a position to pay for outside attendance, he ought to be made to do so.

This would save a lot of work at the hospitals, not to mention the expense in dressings, &c. All these people in Government employment receive a fair wage, and the most of them can afford to pay for medical attendance, and I consider if they are attended to at the time of the accident at the hospital, they can, after this, receive outside attention, and not, as at present, return for weeks for dressings, &c.

In the late President of the Medical Society of Victoria's address (G. F. Howard, B.A., M.D. & Ch.B., Melb.) he has given a very straight opinion, from a medical point of view, of the position of most of our hospitals. He has drawn attention to the abuse of gratuitous medical relief in the hospitals about Melbourne. He points out that the population has not increased of late years, but there has been a great increase in the number of hospital patients; and he thinks the cause of this to some extent is due to the system of exacting contributions from patients who say they are unable to pay ordinary fees. He considers, as we all do, that hospitals should only be used by the destitute classes. He quotes the following extracts from the Police Offences Act, 1890, section 41, subsection 3 :—

"Any person who shall commit any of the following offences shall be deemed a rogue and a vagabond within the meaning of this part, and be liable to the punishment next hereinafter specified."

"Any person imposing or endeavoring to impose upon any charitable institution or private individual by any false or fraudulent representations, either verbally or in writing, with a view to obtain money or any other benefit or advantage, shall be liable to imprisonment in any gaol with hard labor, for any time not exceeding two years."

This ought to be printed in very large letters in and about every one of our hospitals, and also on the back of all forms of admittance either to the in or out patient departments, and a few prosecutions would clear the atmosphere wonderfully.

Dr. Howard says that practically the whole of the classes from whom contributions are now being exacted ought to be in friendly societies, or similar institutions.

He summarises the objections to the pay system as follows :—

(1) Exclusion of deserving cases from the hospitals by paying patients.
(2) Tendency to pauperise the section who really know they are being treated gratuitously.
(3) Patients paying fancy they are paying full value for services rendered, when they are paying nothing for the medical attendance.
(4) It leads to the underselling of the outside medical practitioners, whose lot is assuredly hard enough at present.
(5) The conversion of a charitable institution into a trading institution.
(6) Unsatisfactory examination into circumstances.

At the present time everything is being done by hospital committees to pander to the pauperisation of the public.

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Paying wards in public hospitals must be done away with *in toto*.

Strict investigation into the circumstances of each patient attending the in or out patient department, and a fixed wage of the individual, or collectively of the family, should be settled upon.

In the *British Medical Journal* dated October 29th, 1904, a brief report of the proceedings of the hospitals committee contained references to a memorandum on the principles of hospital management and hospital letters that might be wisely applied to the management of our hospitals in Australia.

The first difference of importance was "that an investigating officer shall be appointed, whose duty shall be to inquire into the eligibility of all applicants."

The following certificate, which has to be signed by all subscribers to the Royal Victoria Hospital, Bournemouth, seems an admirable idea :—

"I certify, having satisfied myself as to the circumstances, that A.B., ..., aged ....., occupation ..... residing at ....., is a fit and proper person to receive the benefits of the charity."

The front of the letter also contains the two following statements :—

"The out-patient department is only intended for those who are too poor to pay for medical relief."

"In any case where the subscriber is in doubt as to the circumstances of the patient, please write across the letter 'For inquiry'"

On the back of the letter is the statement that—"Persons earning more than £1 per week, or members of a family, the collective incomes of which family at home exceed 30s. per week, are not admissible to the benefits of the out-patient department."

At this hospital in all doubtful cases a specially trained inquiry agent is employed to visit the home of the applicant and ascertain the circumstances of the case. The out-patient staff are also required to mark "For inquiry" the letter of any applicant who appears not to be a fit case for charity.

The following recommendation was finally drafted and approved by the annual representative meeting on July 26th :—

"That some means of investigation into the circumstances of the applicants for relief shall be employed in all medical charities, and, where possible, a special officer shall be appointed for this work."

These suggestions are the result of careful investigations in the mother country; but what about the results of investigations into the same subject in Australia?

The number receiving charitable relief for the year ending June, 1904, was 139,771; being 4,830 over the number assisted in the previous year.

Every year more medical practitioners are getting their diplomas, and our population seems at present to be at a standstill. The lodges are using all kinds of endeavors to obtain members from the middle and wealthy classes instead of keeping to the poor, and the result of all this means absolute slave-driving for the general practitioner, for a miserable remuneration.

The question then arises, is our population in a state of poverty? The answer is emphatically no. For Australia pays the highest wages in the world. What are we to do to stop this state of things? For I fear we have no one to help us but ourselves. In fact we seem to have an enormous opposition to any attempt to ameliorate our positions, and the first thing we must do in order to make a start is to unite.

Let us lay aside all minor differences and come together in one vast union, and thus meet our opponents on equal terms to start with.

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By union all our present abuses will be easily remedied, and numerous schemes might be adopted to benefit ourselves generally and the community as well.

Dr. J. P. Ryan has pointed out that half a century ago a public hospital was only used by those who were in much need of help, and it was avoided by all who were not more or less destitute; but—partly owing to the part-paying system adopted—this idea is passing away, and the profession is suffering very seriously for its good nature and foolishness in days gone by.

With reference to the discussion that has been raised lately about the representation of the medical staff of a hospital on its committee, it seems to have caused great anxiety in certain quarters of the community, and fears have been expressed that it would be a mistake.

This matter was thrashed out some time ago in the case of the Women's Hospital, with the result that the hospital has gained in efficiency, and an absence of the interminable quarrels that were once so characteristic of its management. It is well known that in almost all the hospitals of Great Britain the honorary staff is represented.

According to Mr. Butters, one of the committee of the Melbourne Hospital, it is impossible for a gentleman on the honorary staff to also occupy a position on the committee without making use of the latter position in order to gain advantages in the former. He drew attention, on the morning of the election, to Dr. Moore, who occupies this dual position, having used his beds for his own nominees. He speaks of ungenerous and uncalled-for remarks when they are applied against himself; but how does this statement of his compare with his action in stating certain so-called facts in a public paper on the morning of an election, when there is no chance of their being denied, justified, or replied to? Why men should be on a hospital committee who know absolutely nothing about the internal organisation of such a place, and who refuse to take the advice of those whose lives have been spent in studying such subjects, passeth all understanding. Nevertheless, all efforts at reform must be crushed with a stern hand; expert knowledge must not be allowed on hospital committees, and abuses must go on to the end of the chapter, keeping our profession down to its lowest point, using its services gratuitously, and even contemptuously, and curtailing its sources of income in the most barefaced manner.

Suggestions for consideration—

1. Union of the profession.
2. An investigating officer attached to each big institution, and one for, say, three or six of the smaller ones, with power to summon any people abusing the charities; their duties being to investigate into the circumstances of each case at the homes of such cases.
3. A wage limit to be fixed for a single individual and for families.
4. To do away with all part payments, and only use the hospitals for the destitute.
5. The adoption of dispensaries conducted on similar lines to those in Edinburgh, which would come in between the hospitals and the lodges.
6. The establishment of private hospitals on cheaper lines than at present, charging sums sufficient to pay for the up-keep of these hospitals, and within the range of expenditure of the working classes; any medical man who is desired by that patient attending on cheaper lines than ordinary. This would have to be guided by a wage limit also.

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A FEW STATISTICS FOR THE INFORMATION OF THE PROFESSION.

Number receiving charitable relief in Victoria—

For year 1895	115,346
1900	118,412
1901	127,037
1902	127,860
1903	134,941
1904	139,771
1905	—

POPULATION, NUMBER OF REGISTERED MEDICAL MEN, AND MEMBERS OF FRIENDLY SOCIETIES IN VICTORIA.

Year.	Population at End of Year.	Number of Registered Medical Men.	Number of Members of Friendly Societies.
1884	944,564	621	58,859
1894	1,182,290	1,051	80,604
1904	*1,207,605	1,265	†102,040

* On September 30th. † Figures for 1903.

DISCUSSION.

DR. G. T. HOWARD (Melbourne) said that it was a waste of time to discuss the question whether there really was hospital abuse. About one form of it, by people well able to pay ordinary medical fees, there was practically no difference of opinion. Impostors of this class ought to be prosecuted; but owing to the inadequate inquiry into circumstances they escape at present unscathed. The real difficulty is with the class not destitute and not able to pay ordinary medical fees. One way, apparently in the line of least resistance, the hospital managers thought, was to admit them and make them pay something. This innovation, plausible enough in its way, threatens to revolutionise the relations existing between the profession and the poorer classes of the community. The more of these contributing patients (who fondly imagine that they are paying for what they get, while really being treated gratuitously) there are in the hospital the worse it is for the destitute sick, for whom such institutions are intended. It must inevitably happen that deserving destitute are turned away while beds are occupied by contributing patients. This is the point to which attention must be persistently drawn. Most of them ought to be in friendly societies, or in provident dispensaries, like those of Sydney or Broken Hill. These latter institutions were on the right lines, but needed a peculiar combination of enthusiasm, perseverance, and tact and patience, very rare, and as a rule badly appreciated. Those of them needing hospital treatment should be in hospitals instituted and kept up by the benefit societies. The points the profession should insist upon were much closer inquiry into patients' circumstances by some extra hospital agency—preferably charity organisation society—and that public hospitals were for those absolutely unable to pay even the most modest fees.

In addition they ought to use every opportunity to impress these views on hospital managers, and, above all, they ought to organise until their social and political influence was much greater than at present.

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THE ETHICAL RELATIONS BETWEEN TOWN AND COUNTRY PRACTITIONERS.

By Frank Allwork, L.S.A., Lond., New Norfolk, Tasmania.

An experience of upwards of twenty years in "unopposed" country practice in Australasia, and of the many accompanying disadvantages arising therefrom, must be my excuse for calling the attention of my confreres to this subject.

I have purposely avoided the use of the term "consultant," because where my lot has been cast it must be admitted to be inapplicable, except when used in connection with one who is purely a specialist. It is well known that in the great majority of cases the medical men in colonial towns are, both of necessity and possibly also of choice, in every sense of the term, general practitioners. How few (comparatively) honorary surgeons to our public or charitable institutions will, in private practice, decline to attend midwifery cases, or honorary physicians refuse to operate on a surgical one. Now it is especially the relations between the country practitioner and these men that I wish to deal with; and let me make it quite clear that, with some important and disreputable exceptions, I am not charging them with, or even wishing to impute to them, dishonorable motives; but that there is a great want of thought, tact, and discretion often displayed by them when they are consulted by a patient from the country, to the detriment of the patient's own adviser, is a fact well known to, and sometimes bitterly experienced by, the lonely man in the country. Let us for a moment consider the position and surroundings of such an one, as compared with those of his more fortunate town brother. He is morally bound to attend all cases to which he may be summoned, under any and every condition as to distance, weather, hour, or financial means of the patient. His summons is often of the most vague description, especially as regards accidents. His opportunities for arriving at a diagnosis (which it is generally insisted shall be immediately given) are frequently limited to a single or perhaps two interviews, probably under the additional disadvantages of a candle or bad lamp in the middle of the night. For surgical or midwifery cases the nursing assistance is limited to the nearest neighbor, or, at most, a handy woman; the sanitary conditions, bedding accommodation, and culinary arrangements are generally deplorable. All his thinking has to be done under the most adverse circumstances, it may be at the termination of a long journey; nor has he the opportunity of discussing dubious points with a friendly neighbor. He must also be somewhat of a general specialist, and certainly possess a mind capable of adapting itself to peculiar circumstances and making shift with most primitive appliances. Added to all this, the very term "unopposed" means "unassisted"—even by a competent nurse. Finally, the patients of the town man employ him of their own free will—he is of their own choice. What an enormous preliminary advantage this is. On the other hand, the country man is employed *of necessity;* there is no one else to go to, and possibly some prejudice, personal or professional, is held against him; the friends, and it may be the patient himself, are prepared to differ from him in every possible way, even in some instances wilfully disregarding his instructions or advice; the case may present such features as to make a second opinion desirable, if not actually necessary. Out of consideration for the patient's slender means, or not wishing to entail a journey with a doubtful fee upon a professional neighbor, the practitioner hesitates to suggest this too long, when, if able to travel, he finds as his reward the patient has been taken to town to see Dr. So-and-so, who is well and favorably known to his first cousin. The diagnosis, line of treatment, and general opinion of the case may in every

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respect be the same; but the unfortunate employment of different terms conveys a very different meaning to the lay mind, and apparently reflects upon the judgment and skill of the man who has borne the heat and burden of the day by attending a case a distance from home at great personal physical and mental expense. Such and many similar circumstances prevent the customary letter, introducing the case and giving the previous history, from being sent. Yet how great are the possibilities in the hands of the second practitioner for preventing misunderstanding and mischief were he, before pronouncing himself very decidedly, to communicate with the original attendant, to whom the patient will, under the circumstances, probably sooner or later return. Would he not thus be acting more in the interests of the patient also? If a similar sequence of events occurred in town, it is scarcely to be conceived that some communication, either personal or by telephone, would not take place. Why not extend this courtesy to a colleague in the country, even at the cost of a little more time? Such instances are, doubtless, due to thoughtlessness. Want of tact and discretion in giving a positive diagnosis, without a previous history, in a promiscuous case from the country, are mistakes which sometimes score to the credit of the country man, but not till misunderstanding or mischief, which generally could be avoided, has been provoked.

One cannot but regret, also, the disposition sometimes shown by young medical officers of our hospitals to unthinkingly disparage the work of the country practitioner, without having the least conception of the difficulties of private practice in general and unopposed country practice in particular.

In conclusion, I would like to testify to the fact that these violations of ethics of which I speak are rather the exceptions than the rule, but are, in my opinion, and I believe, in the opinion of others, of sufficient frequency to justify attention being drawn to them, with the result, I hope, that those men who have the greater opportunities may be more liberal-minded towards those who are less favorably circumstanced, and, by more courteous communication, help to diminish their many disabilities. This might, with advantage, even be extended into a friendly discussion of cases of mutual interest, though such be only of an every-day character, especially when we take into consideration the fact that rural men are generally debarred from the possibilities of attending the meetings of any medical society. The effect of such action would be to not only raise these men in the esteem and gratitude of the "unopposed" practitioner, but to elevate the general standard of professional ethics.

THE TAKING OF PAYMENT FROM HOSPITAL PATIENTS.

By Dr. W. Moore.

The subject that I have chosen for my paper is one of great importance, but it has only become obtrusive in quite recent years. As yet the medical profession has devoted but little attention to it; and, so far as I am aware, very little has been written upon it.

Though of only recent origin, this evil, for such it must be regarded, has already become very wide-spread. In Victoria the practice is, I believe, universal; indeed, a former Treasurer commanded it as a condition of receiving the Government grant. But even before this the evil was wide-spread. In New South Wales and Queensland the practice is very prevalent, probably just as prevalent as in Victoria; and I presume that here in South Australia, and

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in the other States, it exists. It has grown to such an extent in England that recently the Hospitals Committee of the British Medical Association met a number of hospital managers, and discussed the matter with them.

I think at the Melbourne Hospital the practice of taking payment from patients is met with in about as bad a form as is possible; and I shall, therefore, devote a little time to describe its mode of origin, and its extent at the present time. Like many evil practices, it started in a very insidious way. Some years ago it was considered by the authorities that certain very undesirable people made use of the warmth and comfort of the out-patient department, and used it as a resting-place. It was thought that, by making all out-patients pay a shilling registration fee, these undesirables would be kept away, as they would prefer to keep their shillings to provide themselves with beer. It was soon found that even this shilling fee brought in a considerable sum of money, and so the principle of payment was extended, and all out-patients were asked to pay a certain sum per week for medicine and for treatment; and, of course, it is very easy to see how the principle was still further extended, and in-patients were next asked for payment.

Recently the Finance Committee of the Melbourne Hospital furnished a report on this matter. It stated—"In the Melbourne Hospital the compulsory contribution system has been found satisfactory. There is a special officer appointed to make the necessary inquiries into the circumstances of patients, and to collect their contributions with tact and humanity, and in no case to exact more than a reasonable amount in proportion to the patient's means."

During the year ending June 30th, 1904, 164 out-patients paid 5s. a week; also from out-patients contributions of 10s. to 20s. had been received, these sums amounting to £31 13s. Among in-patients, 168 paid from 10s. to 20s. a week, and as many as 95 paid over £1. It wound up with the following statement:—"In no case, apart from casualties, has a patient been able to contribute such sums as would have obtained suitable private medical attendance, or treatment in a private hospital; and the Finance Committee are of opinion that the system of requiring contributions from patients in proportion to their means is just and proper in order to provide for the large number of patients who, though not absolutely indigent, are unable to pay the ordinary medical fees."

For the year ending June 30th—	£	s.	d.
1901—In-patients' contributions amounted to ..	1,699	0	0
Out-patients' " " ..	1,216	4	4
1902—In-patients' " " ..	1,608	14	1
Out-patients' " " ..	1,144	6	3
1903—In-patients' " " ..	1,560	9	3
Out-patients' " " ..	1,074	4	6
1904—In-patients' " " ..	1,464	8	7
Out-patients' " " ..	1,198	11	0

It will thus be readily seen that not only are the lay managers of the Melbourne Hospital strongly in favor of taking payment from hospital patients, but that very considerable sums of money are obtained in this way.

In his report for the year ending June 30th, 1904, the Inspector of Charities in Victoria, who has been and is a strong and most persistent advocate of the practice of exacting payment from patients, says, "The amount obtained from patients under the form of admission ticket last year was £19,627, which was an increase of £1,211 over that received in the previous year.

"Greater care is now being exercised by some of the managing bodies to see that patients who are suitable cases for hospital treatment contribute according to their means.

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"When the new form of ticket was introduced a few years ago, I stated that, even under a lax administration, at least £20,000 per annum should be obtained from this source towards the maintenance of our medical charities. Although that amount was practically reached last year, yet the administration in regard to the ticket still continues to be lax in many of the hospitals.

"If the provisions of the ticket were carried out in their entirety, the revenue to hospitals from this source should reach £30,000 per annum at the very lowest."

It is thus seen that not only is the practice prevalent, but that it is strongly supported by hospital managers and officials.

It may very reasonably be asked—and I have heard the question asked by medical men—why should hospital patients not pay what they can afford? And I must admit that it is a question that cannot be answered in an off-hand way. At the same time I am satisfied that very good reasons can be given why public hospital patients should not be asked to pay.

In the first place the medical profession cannot too strongly insist on the fact that public hospitals are maintained for the treatment of the sick and injured poor; for the treatment of those who are so poor that, under the added misfortune of sickness or injury, they are dependent for the treatment necessary to bring them back to health again on their better-off fellow citizens. It is for the relief of such cases that physicians and surgeons give their services, not for the relief of those who can afford to pay for treatment. Last year, among other recommendations to the Council of the British Medical Association by its Hospitals Committee were these:—

"That poverty and sickness be the considerations for the admission of all patients for hospital treatment.

"That no charge for the treatment of any patient shall be made, though voluntary contributions may be accepted."

If money is asked for and given, then the patient not unnaturally thinks that he is paying, and paying adequately, for what he gets—not only for his medicine and his food and nursing, but for his medical treatment; and seeing that he is paying for it, there is no reason why he should not get it at the hospital as well as anywhere else. He, too, recommends his friends to go to the hospital and get the same excellent, but cheap, attendance. Thus the constituency of the hospital is greatly increased, and the increase is of the very class that should not go to hospital. Of course the reply is made that inquiries are always made into the circumstances of every patient. The report of the Finance Committee of the Melbourne Hospital, above referred to, says, "There is a special officer to make the necessary inquiries into the circumstances of patients, and to collect their contributions. It admits that this important investigation and the collecting of the money is handed over to a subordinate, and I maintain that, under such circumstances, any subordinate will be certain, in order to show his importance and to please his employers, to raise as much money as he possibly can. He will naturally tend to do two things that are wrong—(1) to take money from those who can pay him the larger sums, such as 5s., 10s., or more a week for out-patients, and 15s., 20s., or more a week from in-patients, without inquiring too closely into their ability to pay ordinary fees for medical treatment; and (2) to demand some payment, even though it be small, from people who cannot afford to pay at all.

That these two errors are freely committed, I am quite certain. Here are some proofs. A woman under my own care I found possessed £1,000 or £1,200 worth of property of her own; her husband had a fair suburban business, and money in the bank. The hospital knew of most of these facts, and yet the patient was taken in. Again, I have a cutting from a newspaper saying that Mr. G., whe died in the Melbourne Hospital, left £450 to be divided equally

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between the Melbourne and Alfred Hospitals. Only the other day a distinguished member of this Congress told me of a case where a colleague, who has a private hospital of his own, sent a patient to a public hospital to be operated on. Among papers in the pocket of this man, who said he could not pay for treatment, were deposit receipts for £1,400. Another case occurred at still another hospital, where the surgeon, after he had done the operation, learned that the patient's husband was very well off. He told the patient that she should give a donation to the hospital. This she refused to do, saying that her husband had no money, and, as a reason for his having no money, made the statement that he had recently bought the farm adjoining his own. I know of numbers of such cases, and I am sure that most of you know of them too.

But it may be asked, did not such cases occur when payment was not sought, and would it not occur now if payment were not demanded? They certainly did occur when payment was not sought from patients, but I think to a far less extent than now; and in those days there was a little more excuse than now. There were then practically no private hospitals, and the public hospitals were the only places properly equipped for dealing with cases requiring serious operations, and no doubt a certain number of such patients, who could have paid medical fees, did go into public hospitals. But the conditions are very different now, when better treatment can be given outside than in the large hospitals. And I am perfectly certain that numbers of people who would never dream of trying to get their medical treatment for nothing are quite content to go and pay £1 a week, although they could easily pay for nursing outside and for medical attendance as well. It is my opinion that many patients who pay for their treatment in private hospitals are less able to do so than are many whom we treat as objects of charity in our public hospitals. It frequently happens that patients sent to me by other medical men, who state that they are fit subjects for treatment in a public hospital, begin to make inquiries about the cost of having their treatment carried out in a private hospital; and a very considerable number of them eventually decide to be treated privately. Thus I am sure that numbers of patients who could afford to be treated privately in a nursing home, or in their own homes, find their way to a public institution, where, in return for the payment of a small weekly charge, they receive board, nursing, and medical attendance.

Now in regard to the second error—the demanding of payment from people who cannot afford to pay anything—I know it will be said that this is not done; but my colleagues and I have met with too many cases to admit of any doubt on the matter. We have been told by patients that they have not been to hospital for some weeks, when they should have been, because they had not the shilling to pay. Then it is quite fair to assume that for every such case that comes under the notice of a member of the staff there will be several that are not seen. Again, it has happened that an out-patient has been told that she should have plenty of milk and nourishing food, and she has asked in astonishment where she is to get them; and yet it has been found that she was paying 2s. 6d. a week to the hospital. These, perhaps, are extreme cases, but I also maintain most strongly that many of those who can apparently pay 5s., 10s., 15s., or 20s., or even more, suffer very great hardships in consequence of that payment being exacted. For instance, in one case a mother was paying 30s. a week for her child who had typhoid. She was earning her living and that of her children by teaching and in other ways. To enable her to do this she kept a servant; but through having to pay this sum to the hospital she had to dismiss the servant and do her housework as well as her other work, and in consequence her own health

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broke down. In another case a man's sick-pay from his lodge was taken whilst he was ill with typhoid. He went out very weak, lost his situation, all his money was gone, and he was stranded.

I am convinced that thus in the great majority of cases an injustice is done in demanding payment from patients at a public hospital, whether out or in patients. The great majority of those who pay cannot afford to pay. Even the smallest payment is a severe strain on their slender resources, and in great part defeats the ends sought to be attained by the charitable, and tends to frustrate the efforts of the members of the medical staff in their treatment. A small minority, but still in the aggregate a considerable number, could pay for medical treatment, and yet receives it as paupers by paying to the hospital a small sum to cover the expenses of medicine and nursing. These patients also in the case of in-patients exclude others who are fit subjects for charitable relief.

It is perhaps worth while briefly to inquire into the causes of the great development of the practice of exacting payment from hospital patients. No doubt hospital committees have found it increasingly difficult to obtain sufficient funds for the carrying on of their work. The great increase in the number of operations performed, the introduction of anti-toxins and other expensive drugs, &c., have tended to increase expenditure. At the same time the trend of legislation in Australia has undoubtedly had much to do with making it difficult for the hospitals to increase their revenues by private subscriptions. Legislation intended to benefit the laboring classes, who used the hospitals, at the expense of their employers, who for the most part maintain them, lessens the income of our hospitals. And the existence of a heavy income tax on larger incomes, whilst the smaller incomes are quite exempt, has the same effect. Then the straitened finances of our State Governments have caused them, instead of increasing the grant from Government to meet the increased expenditure, to actually in many cases lessen it. Mainly in consequence of this action of the Government the Brisbane Hospital is at the present moment in a serious difficulty, the committee has resigned, and the Government is considering the question of undertaking the management. At the same time, at any rate in the case of Victoria, the Government, through the Treasurer, has practically insisted on patients being made to pay what they can.

These seem to be the main causes, and this has to be borne in mind from the hospital managers' point of view. At the same time I am sure that the medical profession should strenously maintain the principle that no payment whatever should in any case be asked for, and even the taking of voluntary contributions from patients should not be in any way encouraged. If on such a matter the medical profession were agreed, I believe they could get their views adopted. In all hospital matters the medical profession, if certain of its own ground and unanimous in its opinion, could have its own way.

If no fees were asked for, and if great care were taken to exclude all patients who can pay, the number of patients seeking admission to the public hospitals would be greatly reduced, and the very poor would have a much better chance of being promptly attended to. The greatest objection to treating all patients absolutely free is that there still would be a certain number of patients who could not afford to pay for treatment outside; but who could, without great hardship, pay something for their maintenance. Now, I maintain that the number of these cases is very small, and that it is steadily diminishing, for the conveniences of private treatment are steadily increasing. It would be much better too that the few patients who could pay a little for their maintenance in hospital should be made a present of everything than that a pernicious principle should be allowed to prevail in the management of our hospitals.

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The profession should devote more time and thought to the management of our hospitals, and endeavor to see that they are managed on lines that would prove more satisfactory to ourselves and to the general public.

Not long ago Dr. Thomson, of Brisbane, speaking to me on this matter, said that four different kinds of hospitals were needed—

(1) Hospitals for infectious diseases, into which patients were compelled to go more for the safety of the community than for their own. These should undoubtedly be maintained out of taxation, municipal or State.

(2) Private hospitals for those who can pay.

(3) Public hospitals maintained by the subscriptions of the charitable, and here, unfortunately, by subsidies from the Government. These should be solely for the poor, and no payment should be taken.

(4) There still remains an important section not provided for: those people who cannot pay the recognised fees for nursing and for medical attendance. These should not be provided for in the same institution as the very poor, for the reasons stated. I think that individual effort on the part of medical men and others will easily settle this question; indeed, it is doing so now. If any collective effort is to be made it should be by friendly societies or some such bodies.

MEDICINE AND SOCIOLOGY.

By T. F. Macdonald, M.B., C.M., &c.

Mr. President and Gentlemen—Students of Sociology, more especially those who are at the same time active members of our profession, must have long ago become convinced of the necessity for a reclassification of the main divisions of medicine. Hitherto medicine has been divided into two main classes—curative medicine and preventive medicine. It seems to me imperative that a division must be added, namely, that of philosophic, social, or evolutionary medicine, in keeping with the deep social changes which are everywhere to be discerned in society at the present time.

The question before the medical profession is, what shall be its position when the social changes shall have become complete, and the present system has been altered? The necessity for a thorough study of Sociology must be apparent to everyone, and I propose that a Chair of Sociology be established in every medical school throughout the world, but more particularly in Australia. Every other science has been duly honored by medicine, and Sociology, the latest—but not the least—of the sciences, is entitled to every consideration at the hands of the medical profession. Perhaps, indeed, it is the most important of studies, and although one is loth to add to the curriculum of the already too heavily weighted medical student, at the same time the necessities of the present trend of things make it imperative that some definite step should be taken for the general study of Sociology.

As examples of our ignorance on the subject, I may mention that in 1894 a congress of evolutionists was held in Chicago, and the following questions were submitted for discussion:—

(1) Can the doctrine of evolution, in its sociological aspects, offer wise suggestions for the solution of the grave social problems of our time?

(2) What are the next steps society should take in keeping with the suggestions which evolutionists may offer?

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These questions were sent to all prominent evolutionists throughout the world, both in England and on the Continent. I had the privilege of reading some two hundred answers to those questions. In not one single instance was the great principle of mutual aid, so important to a complete conception of the true nature of social evolution, mentioned. So important did the matter appear to me that, at the International Medical Congress in Rome, that same year, I read a paper upon the subject, and drew the attention of the international medical world to the importance of a revision of the whole study of evolution. Professor Lombroso made a point of specially congratulating me upon the happy ideas in my paper, and agreed with me upon the necessity of the medical profession giving more attention to the study of the science of Sociology.

The Malthusian doctrine is responsible for the Darwinian conception of evolution, which, put in a nutshell, means the survival of the fittest according to the law of struggle for existence. Huxley emphasized this law in his famous Cambridge manifesto, and argued that society, in its process of evolution, was fighting nature all the way. Herbert Spencer has elaborated the theory of the struggle for existence in his famous Synthetic Philosophy, covering the whole range of human thought. The deductions from Spencer's philosophy and from the teachings of Huxley go to show that in society, according to this theory of evolution, there must be a perpetual bloodthirsty struggle among the units of society in the first instance, and between combinations of these units in the second, and the larger combinations as represented by societies, nations, and empires. According to this law, national war, competition, and struggle must for ever exist.

The fallacy in the Darwinian conception of evolution is that of half truth being asked to do duty for whole truth. While no one can deny the existence of a natural law of self-preservation among animals which entitles them to every consideration as individuals (and the principle of individuality is undoubtedly one of the first principles of social evolution), at the same time there is another principle of equal if not of greater importance to humanity. It has been established more particularly by Prince Kropotkin, in his views which were elaborated in a course of seven years' exposition in the nineteenth century, namely, the law of *mutual aid* among animals, primitive peoples, medieval nations, and amongst ourselves. Kropotkin has shown that the animals which have survived best in the struggle for existence are the associated animals. He gives excellent illustrations of the mutual aid principles expressed in the development of ants, termites, bees, and birds. He shows how the sparrow has survived the eagle, although the eagle is a more powerful individualistic animal; how cockatoos, by a process of mutual aid, rob orchards and cornfields. They post sentinels who give warning of any danger. The flock stand by each other, and by this means they strengthen the individual chances of survival, and so far as being able to survive as a species, they can all the better survive as individuals. Kropotkin further pointed out that Darwin's statement that the struggle for life is more severe between units of the same species is a fallacy. Even Darwin himself never brought one single instance to prove the statement that the struggle was most severe among members of the species. In all other departments of his work Darwin is famed for his elaborate illustrations of any statements he makes, and this is the one notable instance of his failing to do so.

The mutual-aid principle, however, is well exemplified in the quadrupeds. Beavers exist in societies on terms of complete autonomy. It is simply marvellous how the combinations among the beavers fulfil the function of abstract government in ruling bodies. The beavers by mutual aid and com-

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mon action build great dams for common benefit, and mathematicians have been puzzled to account for the accuracy of their engineering. These dams are built of logs, clay, &c., which are obtained by the beavers swimming up streams, gnawing through some tree which might be overhanging the stream, floating it down, and placing it by common effort. Initiative is a great principle among the beavers, as among all the associated animals. Should an ant find a tit-bit too large for itself to carry, it is not long in calling its mates to its assistance. In the same way, when the beaver finds a tree in a good position for falling into the stream, he calls his brethren to the common work of felling the tree, gnawing off its branches, floating it down stream, and putting it in place. Monkeys are well known to live in great tribes, and it is by the principle of mutual aid which they have practised that they derive their intelligence and survival against strong individualistic heavily armed animals. Against combinations of monkeys, the lion, the tiger, the bear, and the eagle, or any other strong individualistic animal, has no chance whatever. It has been described how monkeys in desiring to cross a small river have formed themselves into a living chain by clasping each other and swinging until the last unit of the chain can catch a grip of a tree on the opposite side of the stream, thus forming a natural living suspension bridge over which the whole tribe of monkeys may pass. The reverse action of the chain then takes place, the first unit letting go his hold and swinging towards the other side of the river. A more beautiful illustration of the principle of mutual aid it would be hard to find in the whole range of animal knowledge. The mutual-aid principle manifests itself in its most powerful form among primitive peoples. Man may indeed be said to have attained his position at the head of the animate world because of his social nature and his deep knowledge of the principle of mutual aid and social affairs.

The two principles of self-preservation and mutual aid, then, play their part as a dual number in the development of humanity. They might be called the centripetal and centrifugal forces of society. In its evolution society swings either along the principle of individuality to its extremes, or along the principle of association or mutual aid. By experience society finds its mistakes, when the pendulum swings backwards and forwards, according to what might be called the law of social oscillation. A true conception of the nature of social evolution, then, is impossible without taking into account the full bearing of the mutual-aid factor in social development; but, with the exception of a few ardent students of Sociology, the mutual-aid factor is never taken into account, and we are brought face to face with the impossible problem of attempting to solve social questions upon the struggle-for-existence theory, and we find our efforts in social reform at variance with natural laws.

The mutual-aid factor supplies a long-felt want to humanitarian thinkers, and society has nothing to fear in its evolution if the full force of the mutual-aid theory is taken into account.

Another question which is not understood by thinkers of the present time is the great question of the *unemployed*, and it behoves medical men who look to the future of their profession, as well as to the future of society, to take into account this great question. It seems to me that the unemployed must be looked upon as a permanent feature of the present system of society, comparable in this respect to the criminal, the sick, the indigent aged, and the criminal child. Whenever society makes the discovery of any permanent feature of this nature, it is not long in setting to work to build a permanent institution around it. Hence we find that gaols were invented to meet the criminal question, hospitals were evolved to deal with the sick, reformatories for naughty children, and benevolent asylums for old people in poverty.

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At present, society perhaps does not consider that the unemployed question forms a permanent feature in the present social system. I do not propose to enter into an argument on the subject here, but will content myself with appealing to the general intelligence of medical men that such is the case, and that it behoves society to set to work, and as quickly as possible, to build up permanent institutions for the relief, if not solution, of the unemployed question.

I have thought it possible to devise a scheme of social settlements which would embrace workshops of every description, and great tracts of land for agricultural purposes: these settlements to be controlled by enormous associations, which would elect executive committees. The unemployed would be received freely to these settlements. They would be given freedom of choice as to which trade or work they would preferably take up—agricultural or manual trades, such as carpentry, weaving, paper-making, and pursuits of that nature. By this means the unemployed would be quickly regenerated. The profits arising from any work which they would perform would be divided on the co-operative principle, so as to avoid all difficulties about trade union rates of wages. A permanent reservoir, then, of healthy normal labor would thus be built up, and numbers of really useful citizens could always be on hand as occasion arose for their absorption into society. Should there be no immediate demand for such labor, one thing is certain: these regenerated units of society can always be converted into soldiers and sailors, and no one can deny that at the present time Australia is in very great need of such assistance. Drill would be enforced upon all inmates of these settlements, and one of the finest armies in the world could gradually be built up, if required, by means of institutions of this sort.

I instance these two important questions more as an argument in favor of the necessity for establishing Chairs of Sociology at our universities and schools than to ask the medical profession to take up any special or definite action in regard to them.

I would like to submit the following resolution to the Congress, if my colleagues are in agreement with me on this matter, viz.:—That this Conference expresses its belief that it would be to the advantage of the medical profession if Chairs of Sociology were established in all the teaching schools of Australia.

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APPENDIX TO STATE MEDICINE, ETC.

THE ORGANISATION OF THE AUSTRALIAN ARMY MEDICAL CORPS ON PEACE FOOTING, WITH ITS EXPANSION TO WAR REQUIREMENTS.

By Surgeon-General W. D. C. Williams, C.B.

Mr. President, Ladies and Gentlemen—A proposition was submitted to me by Lieutenant-Colonel Eames, C.B., as to the desirability of requesting that a Naval and Military Section might be included in the Medical Congress we are now assisting at, and this officer voiced the opinion of a number of the officers of the Australian Army Medical Corps in New South Wales.

The President of the Congress was approached on the matter, and although the movement was not taken in hand early enough to form a distinct section, permission was graciously given to ventilate the question at a separate meeting.

This is the first time in the history of the Naval and Military Medical Services of Australia when distinct attention has been given to this most important branch of the services, and I venture to express a hope that in future Congress gatherings it will form a section of its own, and engage that attention which those conversant with the many and various phases it presents will most readily admit it deserves.

Gentlemen, the honor has fallen on myself to lead the way with a paper, at what I may hope to be really the inauguration of a Naval and Military Medical Service Section, and deeply proud am I of that honorable distinction.

With the brief time at disposal every endeavor will be made to clearly and concisely lay before you the present organisation of the Australian Army Medical Services.

The Australian Army Medical Corps organisation provides for three main conditions, viz :—

1. Departmental—at Headquarters.
2. Field Force Troops in each State on peace and war establishments.
3. Garrison Troops in each State on peace and war establishments.

1. The departmental administration is vested in the Director-General Medical Services, who is responsible to the Military Board for all matters connected with the general efficiency, training, medical and surgical field equipment, transport, &c., of the Medical Services.

2. The composition of the Australian Army Medical Services is as follows:—

1. Permanent Army Medical Corps, Nucleus of.
2. Militia Army Medical Corps—Officers' active list and unattached.
3. Volunteer Army Medical Corps—Officers only.
4. Army Nursing Service.

3. The Permanent Army Medical Corps do not at present exist in all States. They carry out the medical duties in connection with permanent troops, and act as an instructional cadre in medical duties generally for all arms.

4. In each military district a principal medical officer is appointed, and in the State of New South Wales there is a staff officer medical service in addition.

5. The Militia and Volunteer Army Medical Corps is organised as follows:—

1. Officers attached to regiments and corps.
2. Mounted bearer companies.
3. Infantry bearer companies.
4. Field hospitals.
5. Garrison companies.

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Certain officers of the Australian Army Medical Corps, Militia, and Volunteer are attached for duty with special regiments and corps. These officers, together with the regimental stretcher-bearers, &c., constitute the regimental medical service.

6. The allotment and designation of mounted bearer companies, infantry bearer companies, and field hospitals for the field force and garrison troops respectively, are as follows :—

1.—FIELD FORCE.

No. 1. Mounted bearer company, New South Wales.
No. 2. Mounted bearer company, New South Wales.
No. 3. Mounted bearer company, Victoria.
No. 4. Mounted bearer company, Victoria.
No. 5. Mounted bearer company, Queensland.
No. 6. Mounted bearer company, South Australia.
No. 1. Infantry bearer company, New South Wales.
No. 2. Infantry bearer company, Victoria.
No. 3. Infantry bearer company, half Queensland and half South Australia.
No. 1. Field hospital, New South Wales.
No. 2. Field hospital, New South Wales.
No. 3. Field hospital, Victoria.
No. 4. Field hospital, Victoria.
No. 5. Field hospital, Queensland.
No. 6. Field hospital, South Australia.

2.—GARRISON TROOPS.

New South Wales garrison company.
Victorian garrison company.
Queensland garrison company.
South Australian garrison company.
Western Australian garrison company.
Tasmanian garrison company.

The medical units of the field force are found by the States of New South Wales, Victoria, Queensland, and South Australia.

The units have a similar allotment in New South Wales and Victoria, and those of Queensland and South Australia also correspond as regards distribution and numbers.

The garrison companies of the Army Medical Corps, organised for duty with garrison troops at the fortifications and with district reserves, vary in strength in each State, in accordance with the number of forts and the distribution of garrison troops.

7. The Australian Army Nursing Service is a voluntary body, formed to supply a nursing service under an organised military system, for duty at base hospitals and stationary field hospitals in times of war.

8. The establishment of the Australian Army Nursing Service will be, until further orders :—New South Wales and Victoria, one lady superintendent, one matron, and twenty-four nursing sisters in each State ; Queensland and South Australia, one lady superintendent, one matron, and fourteen nursing sisters in each State ; Western Australia and Tasmania, one lady superintendent, one matron, and ten nursing sisters ; total, six lady superintendents, six matrons, and ninety-six nursing sisters.

Provision has not, as yet, been made on the Estimates for the nursing service of Western Australia and Tasmania.

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9. It is a nucleus of a military nursing service. The nursing sisters undergo annually a course of instruction in the special organisation and duties which are absolutely essential for the conduct of military nursing.

This Australian Army Nursing Service, I am proud to say, is extremely popular, and the keenest interest in the work is shown by all.

10. Reserve of officers. A scheme has been formulated, but has not yet received official sanction, whereby the creation of a reserve of officers could be at once proceeded with.

Briefly, the reserve would provide staffs of consultant physicians and surgeons for duty at base and stationary field hospitals, and, from the junior members, additional *personnel* for bearer companies or field hospitals, or even hospital trains if required.

The scheme would not cause any dislocation of the working of the Government or civil hospitals in military districts. Much additional accommodation would inevitably be needed, to meet the requirements of sick and wounded sent down from the front, in whatever State or States that might be, entailing occupation of suitable buildings in hospital vicinity, or the erection of tents in hospital or adjacent grounds. This would naturally fall on the military authorities to carry out, and if in time of peace not only were the *personnel* for hospital duty duly allotted, the routine of official duty drawn up and requisitions prepared for all medical stores and equipment, there would not be any hitch when the pinch came; many valuable lives would be saved; and that useless expenditure of money avoided which hurried and hysterical organisation invariably brings about.

I feel certain, gentlemen, when the reserve system receives official approval that it will commend itself to a great number of the profession, who, although not now holding commissions in the Australian Army Medical Corps, would only be too glad to render patriotic service in time of need, and give their services on the lines indicated. The want of some such reserve, properly organised in peace time, was severely realised by the Imperial authorities when the strain on the *personnel* of the then existing medical services began to be felt.

11. The question of bringing into line the voluntary aid societies of the several States as organised bodies, to leaven in with the medical services in time of emergency, is one that requires early and intent consideration; allusion will be made to this again later on.

12. The establishments of units of the Australian Army Medical Services, both for peace and war, viz., mounted bearer companies, infantry bearer companies, or field hospitals, are organised for service with the brigades to which they are attached—whether light horse or infantry—and form part and parcel of those brigades, and would take the field with them in the first line of transport.

These medical units are capable of immediate sub-division, both as regards *personnel*, equipment, and transport, into two half units, should it be necessary from tactical considerations to subdivide the brigade to which any medical unit may belong.

The establishments, peace and war, of the several units of the Australian Army Medical Corps for the field force are given in Appendix No. 1.

13. There are several differences in the organisation of the medical services of the Imperial Forces and those of the Commonwealth. Prior to the new organisation of the Royal Army Medical Corps, which came into effect in the early part of this year, the main points of difference were :—

1. That the Imperial Service did not provide for any peace establishments of units—only a war establishment—with certain small additions of *personnel* when serving abroad. I have always held that a peace unit, complete in itself, gives every opportunity for

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a high-class and efficient training, as it is possible to carry out exercises, duties, and routine on a reduced scale, precisely as they would be on a war footing.

2. The next departure in the Commonwealth service is that whereas, in the Imperial Forces, the Royal Army Medical Corps are dependent on the Army Service Corps for their ambulance wagons, cart transport, horses, harness, and drivers, in the Australian Medical Services everything connected with its transport is part of the corps, and each unit has its own distinct allotment. It is thus self-contained, a dual command is avoided, higher efficiency is attained—as even all drivers, in addition to their special qualification, are trained stretcher-bearers and certified in "first aid," instead of being drivers pure and simple, and belonging to another corps, devoid of medical traditions. When the drivers form part of the medical unit, they are on the spot when wanted, and officers and non-commissioned officers know them, and they know their superiors; the *esprit* of comradeship exists, and a unit with a high standard ensues.

Whatever success may have attended the units of the New South Wales Army Medical Corps in the late South African war, I attribute in no small measure to the fact that its transport was self-contained, and a part of the corps.

Another point in which the Commonwealth service has struck out in a new line is the establishment of ambulance depots in the various States for the accommodation of the whole of the ordnance stores, medical and surgical field equipment, ambulance wagons, transport wagons and carts, harness and saddlery, required for regimental services, bearer companies—mounted and infantry—and field hospitals both for the field and garrison forces, in accordance with the allotment of troops in the general scheme of organisation per States.

By this means every officer throughout the medical services of the Commonwealth, whether attached to a regiment or medical unit, will be able—as soon as the remaining depots are constructed, and the supply of equipment and stores now on indent or for local contract is to hand—to draw his material from the compartment in which it is stored, available at a few minutes' notice, simply by signing a requisition, which, in printed form, tabulates his stores, and then join the regiment or brigade to which he may be allotted, fully equipped.

This system applies as equally to bearer companies and field hospitals as it does to regimental equipment.

Another departure is that in every fort throughout our defences casualty-rooms have been fitted up, replete with stretchers, surgical dressings, and a few drugs; and each casualty-room is equipped on similar lines. All the medical officer attached for duty at any fort has to do is to walk in and find all to hand, either by day or night.

14. The organisation which has so lately come into force in the Imperial Service does away with the separate units of bearer company and field hospital, and a combined unit has resulted, known as a field ambulance for use with infantry, and a cavalry field ambulance for mounted troops. This is the first attempt in the Imperial Service to provide a special unit for cavalry: up to now the bearer companies and field hospitals—as used for infantry—have had to do their best with cavalry troops, and a want of mobility has always been sorely felt.

These field ambulances are divided into sections, each capable of acting separately—the field ambulance into three sections, and the cavalry field ambulance into two sections.

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The ambulances are made up of a bearer division, corresponding to the old bearer company, a tent division for 150 patients to the previous field hospital, and transport details—which are still drawn from the Army Service Corps.

The *personnel* in these combined units has been increased. The details of the establishment of a field ambulance and a cavalry field ambulance are given in Appendix No. 2.

It will be noticed that in completing the *personnel* that a proportion of the officers required may be civilian surgeons specially employed, and a number of the men to be specially enlisted.

The most important factor in medical services is that of training, which, to be of any value, must be thorough. No detail can be spared, no point missed.

Our officers come into the service with high professional attainments, and many of them in special line of practice, and are given every opportunity to make themselves proficient in the military side of their service, both by courses of instruction and camps of continuous training.

Other ranks have similar facilities afforded them.

The training of men who are required to render first aid in the field, often under heavy fire, the conduct of the transport of wounded by the stretcher-bearers and ambulances, the multitude of duties which are part of the every-day work of hospital orderlies and attendants in field and other hospitals, is, in my opinion, the one point we should ever have in view. Nothing can be more lamentable to conceive—worse than lamentable, almost criminal—than that sick or wounded should be handed over to the care of attendants who, by want of training and experience, do infinite mischief. In the words of the dying King Arthur, "A little thing may hurt a wounded man."

The Defence Act provided that all officers, irrespective of what branch of the service they may belong to, must qualify for promotion up to and including the rank of lieutenant-colonel, and it is therefore incumbent for medical officers to submit themselves for examination in those subjects connected with the military conduct of a medical service as have been duly defined in the syllabus of examination for each rank.

Officers joining the Australian Army Medical Corps did so with the rank of lieutenant, and served three years in that position; but the Military Board have lately decided that they shall in future enter the service with the rank of captain on their first commission, to be provisional, and confirmed on passing the requisite examination of the rank within a specified time.

It is maintained that this will do much to popularise the medical service amongst the profession, and hold out an inducement for applications for commissions in the several States.

15. This brings us now to that most vital point—how can we expand our medical services from a peace to a war footing? In this we have to deal not only with conditions which do not exist in peace—the formation of hospitals at the base, on lines of communication, and in fortress areas, hospital trains, advanced depots of medical and surgical stores, and may be hospital ships, also with items of equipment and transport—but with the very difficult question of *personnel*, both officers and men. Officers have much to learn in the military conduct of their duty, and men cannot be trained in a week or a month. The training of a first-class hospital orderly cannot be attained in a year.

It would be beyond the power of any country to keep a highly trained and completely equipped medical service up to war requirements on its military estimates, but it is within any nation's power to have its medical units so organised that necessary expansion from peace to war can rapidly and efficiently be carried out. But this must entail previous organisation, which, when set in motion, will proceed without friction, delay, or useless expenditure.

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A highly-trained nucleus is essential; given this, expansion through means of a reserve of officers, and the welding into the service the *personnel* and equipment of an efficient Red Cross Society would be easily effected, and should give the most splendid results.

Let us look forward to the establishment of the Red Cross Society of Australia at no distant date.

In July last the new Red Cross Society of the United Kingdom was inaugurated at Buckingham Palace by their Majesties the King and Queen. This new Red Cross Society is not limited to the United Kingdom, but is to be an Empire movement. The object of the society will be to provide volunteer aid in time of war, on the lines laid down in the Geneva Convention. In times of peace the services of such a society could be used towards alleviating the suffering brought about by floods, fire, or any national calamity.

In time of war the society would come under naval and military control, and would provide equipment and a trained *personnel* to work side by side with army medical services.

Her Majesty in the opening proposed that—"This new organisation should be based upon membership association, and the members and associates of the society shall be recruited from all classes throughout the Empire.

"The society shall be entirely voluntary, and while in touch with the War Office and Admiralty, the society shall be organised and act wholly independently of those departments in time of peace, but naturally, in time of war, it must be under naval and military control.

"I therefore now appeal to all the women of the Empire to assist me in carrying out this great scheme, which is essentially a woman's work, and which is the only one and only way in which we can assist our brave and gallant army and navy to perform their arduous duties in time of war."

That a Red Cross Society, as indicated by the speech of Her Majesty, is not only possible, but from every aspect most desirable for our Commonwealth, seems to go without comment, and I trust in the near future that opportunity will be seized to place this magnificent movement of philanthropy on a pedestal to which all Australia will be able to look with pride and admiration.

The first step would be the formation of a central organisation: centres to be established in each State on similar lines, and a harmonious bringing into the fold of the various civil aid societies now existing amongst us.

The magnificent work carried out by the Red Cross Society of Japan during the war which has just been concluded by the signing of peace, has been an object lesson to the world, providing, as it has done, a thoroughly organised system, both as regards *personnel*, equipment, general and medical stores for a Red Cross hospital, relief detachments, rest stations, depots of supply, and transport columns for the vast armies of Japan which have been operating in the East for the past eighteen months.

It has a reserve of 3,000 trained nurses ready for immediate service.

The Japanese society was only incorporated in 1902, so that within a couple of years it has had to submit its organisation to the most severe strain that it could be called upon to perform, and it has stood every test in the most splendid manner. At the present time the society's income stands at £250,000, and the members number over a million.

I quote the above facts from that most valuable and interesting work "Under the Care of the Japanese War Office," by Miss Ethel McCaul, R.R.C., who received the sanction of Her Majesty the Queen and the permission of the Japanese Government to inspect and report on the work of the Red Cross Society of Japan.

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With such an example before us, does it not behove Australia to make some move in a similar direction, and establish a society which would be of the highest national benefit to our navy and army in time of war, and which would at once allow of that expansion of the medical services without which disaster would be inevitable, and untold sufferings the dire result.

16. Having already exceeded my time limit, I will now, Mr. Chairman and gentlemen, bring this paper to a close, with much regret that it has been so sketchy, but with the hope that it may afford some insight into what our Medical Service is, and what we should look forward to for the attainment of the high efficiency it demands, and its expansion, which would be such an urgent necessity in time of any national emergency.

Appendix No. 1.

AUSTRALIAN ARMY MEDICAL CORPS.

The Australian Army Medical Corps will be organised for administrative purposes into companies as follows:—

I. Mounted companies consisting of—

One mounted bearer company and half field hospital for service with Light Horse Brigades (Tables XIX. and XXIII.).

II. Infantry companies consisting of—

One infantry bearer company and one field hospital for service with Infantry Brigades (Tables XXI. and XXIII.).

III. Garrison companies of varying establishments to suit local requirements.

Table XIX.—Peace Establishment of a Mounted Bearer Company for Service with Brigade of Light Horse.

Ranks.	Personnel.						Horses.			Vehicles.		
	Officers.	Warrant Officers.	Staff Sergeants and Sergeants.	Buglers.	Rank and File.	Total.	Riding.	Draught.	Total.	Carts, Transport.	Wagons, Ambulance.	Total.
Medical Details.												
Captain or Lieutenant	1	—	—	—	—	1	1	—	1	—	—	—
Staff Sergeant	—	—	1	—	—	2	1	—	1	—	—	—
Sergeant	—	—	1	—	—		1	—	1	—	—	—
Bugler	—	—	—	1	—	1	1	—	1	—	—	—
Corporal	—	—	—	—	1	16	16*	—	—	—	—	—
Privates	—	—	—	—	13			—	—	—	—	—
Private as cook	—	—	—	—	1			—	16	—	—	—
Private as batman	—	—	—	—	1			—	—	—	—	—
Total medical details	1	—	2	1	16	20	20	—	20	—	—	—
Transport Details.												
Drivers, ambulance wagons	—	—	—	—	4	5	—	8	8	—	2	2
Driver, transport cart	—	—	—	—	1		—	2	2	1	—	1
Total transport details.	—	—	—	—	5	5	—	10	10	1	2	3
Total mounted bearer company	1	—	2	1	21	25	20	10	30	1	2	3

* Includes 1 pack horse.

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APPENDIX No. 1—*continued.*

PEACE ESTABLISHMENT OF A MOUNTED BEARER COMPANY, &c.—*continued.*

NOTE.—To bring up to war strength there will be required :—

Ranks.	Medical Details.	Transport Details.	Total.
Captain or lieutenant	1	—	1
Warrant officer	1	—	1
Sergeant	1	—	1
Rank and file	16	6	22
Total	19	—	25

WAR ESTABLISHMENT OF A MOUNTED BEARER COMPANY FOR SERVICE WITH BRIGADE OF LIGHT HORSE.

Ranks.	Personnel.						Horses.			Vehicles.			
	Officers.	Warrant Officers.	Staff Sergeants and Sergeants.	Buglers.	Rank and File.	Total.	Riding.	Draught.	Total	Carts, Transport.	Carts, Water.	Wagons, Ambulance.	Total.
Medical Details.													
Captain	1	—	—	—	—	2	1	—	1	—	—	—	—
Lieutenant	1	—	—	—	—		1	—	1	—	—	—	—
Warrant officer	—	1	—	—	—	1	1	—	1	—	—	—	—
Staff sergeant	—	—	1	—	—	3	1	—	1	—	—	—	—
Sergeants	—	—	2	—	—		2	—	2	—	—	—	—
Buglers	—	—	—	1	—	1	1	—	1	—	—	—	—
Corporals	—	—	—	—	2	32	32*	—	—	—	—	—	—
Corporal as cook	—	—	—	—	1			—	—	—	—	—	—
Privates	—	—	—	—	26			—	32	—	—	—	—
Private as cook	—	—	—	—	1			—	—	—	—	—	—
Privates as batmen	—	—	—	—	2			—	—	—	—	—	—
Total medical details	2	1	3	1	32	39	39	—	39	—	—	—	—
Transport Details.													
Drivers, ambulance wagons	—	—	—	—	8	11	—	16	16	—	—	4	4
Drivers, transport carts	—	—	—	—	2		—	4	4	2	—	—	2
Driver, water cart	—	—	—	—	1		—	1	1	—	1	—	1
Total transport details	—	—	—	—	11	11	—	21	21	2	1	4	7
Total mounted bearer company	2	1	3	1	43	50	39	21	60	2	1	4	7

* Includes 2 pack horses.

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APPENDIX No. 1—*continued.*

PEACE ESTABLISHMENT OF A BEARER COMPANY FOR SERVICE WITH INFANTRY BRIGADE.

Rank.	Personnel.							Horses.			Vehicles.			
											Wagons.		Carts.	
	Officers.	Warrant Officers.	Staff Sergeants and Sergeants.	Artificers.	Buglers.	Rank and File.	Total.	Riding.	Draught.	Total.	Ambulance.	Transport.	Water.	Total.
Medical Details.														
Major	1	—	—	—	—	—	3	1	—	1	—	—	—	—
Captain	1	—	—	—	—	—		1	—	1	—	—	—	—
Lieutenant	1	—	—	—	—	—		1	—	1	—	—	—	—
Warrant Officer	—	1	—	—	—	—	1	1	—	1	—	—	—	—
Quartermaster-sergeant	—	—	1	—	—	—	3	—	—	—	—	—	—	—
Compounder	—	—	1	—	—	—		—	—	—	—	—	—	—
Sergeant	—	—	1	—	—	—		—	—	—	—	—	—	—
Bugler	—	—	—	—	1	—	1	—	—	—	—	—	—	—
Corporals	—	—	—	—	—	4	1	—	—	—	—	—	—	—
Privates	—	—	—	—	—	16	26	—	—	—	—	—	—	—
Private as cook	—	—	—	—	—	1		—	—	—	—	—	—	—
Privates as wagon orderlies	—	—	—	—	—	2		—	—	—	—	—	—	—
Privates as batmen	—	—	—	—	—	3		—	—	—	—	—	—	—
Total medical details	3	1	3	—	1	26	34	4	—	4	—	—	—	—
Transport Details.														
Sergeants	—	—	1	—	—	—	1	1	—	1	—	—	—	—
Farrier-sergeant	—	—	—	1	—	—	2	—	—	—	—	—	—	—
Collarmaker-sergeant	—	—	—	1	—	—		—	—	—	—	—	—	—
Drivers, ambulance wagons	—	—	—	—	—	8	13	—	16	16	4	—	—	4
Drivers, transport wagons	—	—	—	—	—	4		—	8	8	—	4	—	4
Driver, water cart	—	—	—	—	—	1		—	2	2	—	—	1	1
Total transport details	—	—	1	2	—	13	16	1	26	27	4	4	1	9
Total bearer company	3	1	4	2	1	39	50	5	26	31	4	4	1	9

NOTE.—To bring up to war strength there will be required :—

Ranks.	Medical Details.	Transport Details.	Total.
Officer	1	—	1
Staff sergeants and sergeants	2	1	3
Rank and file	29	17	46
Total	32	18	50

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APPENDIX No. 1—*continued.*

WAR ESTABLISHMENT OF A BEARER COMPANY FOR SERVICE WITH INFANTRY BRIGADE.

Ranks.	Personnel.							Horses.			Vehicles.			
											Wagons.		Carts.	
	Officers.	Warrant Officers.	Staff Sergeants and Sergeants.	Artificers.	Buglers.	Rank and File.	Total.	Riding.	Draught.	Total.	Ambulance.	Transport.	Water.	Total.
Medical Details.														
Major	1	—	—	—	—	—	4	1	—	1	—	—	—	—
Captains	2	—	—	—	—	—		2	—	2	—	—	—	—
Lieutenant	1	—	—	—	—	—		1	—	1	—	—	—	—
Warrant officer	—	1	—	—	—	—	1	1	—	1	—	—	—	—
Quartermaster-sergeant	—	—	1	—	—	—	5	—	—	—	—	—	—	—
Compounder	—	—	1	—	—	—		—	—	—	—	—	—	—
Sergeants	—	—	3	—	—	—		—	—	—	—	—	—	—
Bugler	—	—	—	—	1	—	1	—	—	—	—	—	—	—
Corporals	—	—	—	—	—	2	55	—	—	—	—	—	—	—
Corporals as wagon orderlies	—	—	—	—	—	5		—	—	—	—	—	—	—
Corporal as cook	—	—	—	—	—	1		—	—	—	—	—	—	—
Privates	—	—	—	—	—	35		—	—	—	—	—	—	—
Private as cook	—	—	—	—	—	1		—	—	—	—	—	—	—
Privates as wagon orderlies	—	—	—	—	—	5		—	—	—	—	—	—	—
Privates as batmen	—	—	—	—	—	4		—	—	—	—	—	—	—
Privates as supernumeraries	—	—	—	—	—	2		—	—	—	—	—	—	—
Total medical details	4	1	5	—	1	55	66	5	—	5	—	—	—	—
Transport Details.														
Sergeants	—	—	2	—	—	—	2	2	—	2	—	—	—	—
Farrier-sergeant	—	—	—	1	—	—	2	—	—	—	—	—	—	—
Collarmaker-sergeant	—	—	—	1	—	—		—	—	—	—	—	—	—
Corporal	—	—	—	—	—	1	30	—	—	—	—	—	—	—
Drivers, ambulance wagons	—	—	—	—	—	20		—	40	40	10	—	—	10
Drivers, transport wagons	—	—	—	—	—	6		—	12	12	—	6	—	6
Driver, water cart	—	—	—	—	—	1		—	2	2	—	—	1	1
Drivers as supernumeraries	—	—	—	—	—	2		—	—	—	—	—	—	—
Total transport details	—	—	2	2	—	30	34	2	54	56	10	6	1	17
Total bearer company	4	1	7	2	1	85	100	7	54	61	10	6	1	17

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APPENDIX No. 1—*continued.*

PEACE ESTABLISHMENT OF A FIELD HOSPITAL OF 100 BEDS.

Ranks.	Personnel.							Horses.			Vehicles.		
	Officers.	Warrant Officers.	Staff Sergeants and Sergeants.	Artificers.	Buglers.	Rank and File.	Total.	Riding.	Draught.	Total.	Wagons, Transport.	Carts, Water.	Total.
Medical Details.													
Major	1	—	—	—	—	—	3	1	—	1	—	—	—
Captain	1	—	—	—	—	—		1	—	1	—	—	—
Lieutenant	1	—	—	—	—	—		1	—	1	—	—	—
Chief wardmaster	—	1	—	—	—	—	1	1	—	1	—	—	—
Steward*	—	—	1	—	—	—	2	—	—	—	—	—	—
Compounder	—	—	1	—	—	—		—	—	—	—	—	—
Corporal as clerk	—	—	—	—	—	1	15	—	—	—	—	—	—
Corporal as cook	—	—	—	—	—	1		—	—	—	—	—	—
Private as cook	—	—	—	—	—	1		—	—	—	—	—	—
Privates as ward orderlies	—	—	—	—	—	8		—	—	—	—	—	—
Private as washerman	—	—	—	—	—	1		—	—	—	—	—	—
Privates as batmen	3	—	—	—	—	3		—	—	—	—	—	—
Total medical details	—	1	2	—	—	15	21	4	—	4	—	—	—
Transport Details.													
Sergeant	—	—	1	—	—	—	1	1	—	1	—	—	—
Corporal	—	—	—	—	—	1	8	1	—	1	—	—	—
Drivers, transport wagons	—	—	—	—	—	6		—	12	12	6	—	6
Driver, water cart	—	—	—	—	—	1		—	1	1	—	1	1
Total transport details	—	—	1	—	—	8	9	2	13	15	6	1	7
Total field hospital	3	1	3	—	—	23	30	6	13	19	6	1	7

NOTE.—To bring up to war strength there will be required :—

Ranks.	Medical Details.	Transport Details.	Total.
Officers	2	—	2
Staff sergeants and sergeants	5	1	6
Artificer	—	1	1
Rank and file	15	6	21
Total	22	8	30

* Also perform duties of quartermaster-sergeant.

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APPENDIX No. 1—*continued.*

War Establishment of a Field Hospital of 100 Beds.

Ranks.	Personnel.							Horses.			Vehicles.		
	Officers.	Warrant Officers.	Staff Sergeants and Sergeants.	Artificers.	Buglers.	Rank and File.	Total	Riding.	Draught.	Total.	Wagons, Transport.	Carts, Water.	Total.
Medical Details.													
Major	1	—	—	—	—	—		1	—	1	—	—	—
Captains	2	—	—	—	—	—		2	—	2	—	—	—
Lieutenant	1	—	—	—	—	—	5	1	—	1	—	—	—
Quartermaster	1	—	—	—	—	—		1	—	1	—	—	—
Chief wardmaster	—	1	—	—	—	—	1	1	—	1	—	—	—
Assistant wardmaster	—	—	1	—	—	—		—	—	—	—	—	—
Steward	—	—	1	—	—	—		—	—	—	—	—	—
Compounders	—	—	2	—	—	—	7	—	—	—	—	—	—
Cook	—	—	1	—	—	—		—	—	—	—	—	—
Pack storekeeper	—	—	1	—	—	—		—	—	—	—	—	—
Supernumerary	—	—	1	—	—	—		—	—	—	—	—	—
Corporal as steward	—	—	—	—	—	1		—	—	—	—	—	—
Corporal as cook	—	—	—	—	—	1		—	—	—	—	—	—
Corporal as clerk	—	—	—	—	—	1		—	—	—	—	—	—
Corporal as supernumerary	—	—	—	—	—	1		—	—	—	—	—	—
Privates as war orderlies	—	—	—	—	—	14		—	—	—	—	—	—
Private as cook	—	—	—	—	—	1	30	—	—	—	—	—	—
Private as pack storekeeper	—	—	—	—	—	1		—	—	—	—	—	—
Private as messenger	—	—	—	—	—	1		—	—	—	—	—	—
Privates as washermen	—	—	—	—	—	2		—	—	—	—	—	—
Privates as supernumeraries	—	—	—	—	—	2		—	—	—	—	—	—
Privates as batmen	—	—	—	—	—	5		—	—	—	—	—	—
Total medical details	5	1	7	—	—	30	43	6	—	6	—	—	—
Transport Details.													
Staff sergeant	—	—	1	—	—	—	2	1	—	1	—	—	—
Sergeant	—	—	1	—	—	—		1	—	1	—	—	—
Shoeing smith	—	—	—	1	—	—	1	—	—	—	—	—	—
Corporals	—	—	—	—	—	2		—	—	—	—	—	—
Drivers, transport wagons	—	—	—	—	—	10	14	—	20	20	10	—	10
Driver, water cart	—	—	—	—	—	1		—	2	2	—	1	1
Driver as supernumerary	—	—	—	—	—	1		—	—	—	—	—	—
Total transport details	—	—	2	1	—	14	17	2	22	24	10	1	11
Total field hospital	5	1	9	1	—	44	60	8	22	30	10	1	11

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APPENDIX No. 2.

(A)—A Field Ambulance (Three Sections)—(1) Personnel and Horses.

Detail.	Personnel.						Horses.		
	Officers.	Warrant Officers.	Staff Sergeant and Sergeants.	Buglers.	Rank and File.	Total.	Riding.	Draught.	Total.
Detail of Field Ambulance—Bearer Division.									
Captains or subalterns	3	—	—	—	—	—	3	—	3
Sergeants	—	—	3	—	—	—	—	—	—
Buglers	—	—	—	3	—	129	—	—	—
Corporals	—	—	—	—	117*a*	—	—	—	—
Tent Division (150 *Patients*).									
Lieutenant-Colonel	*b*1	—	—	—	—	—	1	—	1
Majors	2	—	—	—	—	—	2	—	2
Captains or subalterns	3	—	—	—	—	—	3	—	3
Quartermaster	1	—	—	—	—	—	1	—	1
Sergeant-Major	—	1	—	—	—	—	—	—	—
Sergeants: Nursing duties	—	—	2	—	—	—	—	—	—
Sergeants: Stewards	—	—	2	—	—	—	—	—	—
Sergeants: Compounders	—	—	3	—	—	63	—	—	—
Sergeants: Clerks	—	—	3	—	—	—	—	—	—
Corporals: Cooks	—	—	—	—	3	—	—	—	—
Corporals: Pack storekeepers	—	—	—	—	3	—	—	—	—
Privates: Nursing duties	—	—	—	—	21	—	—	—	—
Privates: Clerks	—	—	—	—	3	—	—	—	—
Privates: Cooks	—	—	—	—	3	—	—	—	—
Privates: Washermen	—	—	—	—	3	—	—	—	—
Privates: Supernumeraries	—	—	—	—	9	—	—	—	—
Total	10*c*	1	13	3	165	192	11	—	11
Attached—Transport Details (*A.S.C.*).									
Sergeants	—	—	3	—	—	—	3	—	3
Drivers: For vehicles	—	—	—	—	41	60	—	82	82
Drivers: For spare draught horses	—	—	—	—	3	—	—	6	6
Drivers: Spare (10 per cent.)	—	—	—	—	3	—	—	—	—
Batmen	—	—	—	—	10	—	—	—	—
Total field ambulance (including attached)	10	—	16	3	222	252	14	88	102

Details left at the base (not included in above tables).

Private (storeman) *d*	—	—	—	—	1	—	—	—	—
First re-inforcement *e*	—	—	—	—	16	17	—	—	—

a Of these 90 may be specially enlisted men. *b* Commands the unit. *c* Of these 3 may be civilian surgeons specially enlisted. *d* Will mobilise with the unit. *e* Will mobilise with the unit for service abroad.

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APPENDIX No. 2.—*continued.*

(2)—TRANSPORT.

Detail.	Vehicles.	Drivers (A.S.C.).	Draught Horses.
Carts, water (1 per section)	3	3	6
Wagons, ambulance	10	20	40
Wagons, G.S. for baggage, stores, and supplies (3 per section)	9	18	36
Drivers, for spare draught horses	—	3	6
Drivers, spare	—	3	—
Total	22	47	88

NOTE.—Two field ambulances will be included in an infantry division, and one ambulance will be included in the corps troops of an army corps.

(B)—A CAVALRY FIELD AMBULANCE (TWO SECTIONS)—(1) PERSONNEL AND HORSES.

Detail.	Personnel.						Horses.		
	Officers.	Warrant Officers.	Staff Sergeants and Sergeants.	Buglers.	Rank and File.	Total.	Riding.	Draught.	Total.
Details of Field Ambulance—Bearer Division.									
Captains or subalterns	2	—	—	—	—	42	2	—	2
Buglers	—	—	—	2	—		—	—	—
Corporals	—	—	—	—	4		—	—	—
Privates	—	—	—	—	34*a*		—	—	—
Tent Division (50 *Patients*).									
Lieutenant-Colonel or majors	2*b*	—	—	—	—	34	2	—	2
Captains or subalterns	2*c*	—	—	—	—		2	—	2
Sergeant-Major	—	1	—	—	—		1	—	1
Sergeants: Nursing duties	—	—	2	—	—		—	—	—
Sergeants: Stewards	—	—	2	—	—		—	—	—
Sergeants: Compounders	—	—	2	—	—		—	—	—
Sergeants: Supernumerary	—	—	1	—	—		—	—	—
Corporals: Cooks	—	—	—	—	2		—	—	—
Corporals: Pack storekeepers	—	—	—	—	2		—	—	—
Privates: Clerks	—	—	—	—	2		—	—	—
Privates: Nursing duties	—	—	—	—	10		—	—	—
Privates: Cooks	—	—	—	—	2		—	—	—
Privates: Washermen	—	—	—	—	2		—	—	—
Privates: Supernumeraries	—	—	—	—	2		—	—	—
Total	6	1	7	2	60	76	7	—	7
Attached—Transport Details (*A.S.C.*).									
Sergeants	—	—	2	—	—	44	2	—	2
Drivers: For vehicles	—	—	—	—	32		—	64	64
Drivers: For spare draught horses	—	—	—	—	2		—	4	4
Drivers: Spare, 10 per cent.	—	—	—	—	2		—	—	—
Batmen	—	—	—	—	6		—	—	—
Total field ambulance, including attached	6	1	9	2	102	120	9	68	77
Details left at the base (not included in above totals).									
Private (storeman) *d*	—	—	—	—	1	—	—	—	—
First re-inforcement *e*	—	—	—	—	6	7	—	—	—

a Of these 18 may be specially enlisted men. *b* The senior commands. *c* May be civil surgeons specially enlisted. *d* Will mobilise with the unit. *e* Will mobilise with the unit for service abroad.

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APPENDIX No. 2—*continued.*

(2)—TRANSPORT.

Detail.	Vehicles.	Drivers, A.S.C.	Draught Horses.
Carts, water (1 per section)	2	2	4
Wagons — Ambulance — six-horsed (3 per section)	6	18	36
Wagons — Ambulance — two-horsed, light (2 per section)	4	4	8
Wagons — G. S. for baggage, stores, and supplies (2 per sec.)	4	8	16
Drivers, for spare horses (draught)	—	2	4
Drivers, spare	—	2	—
Total	16	36	68

NOTE.—A cavalry field ambulance will be included in a cavalry brigade.

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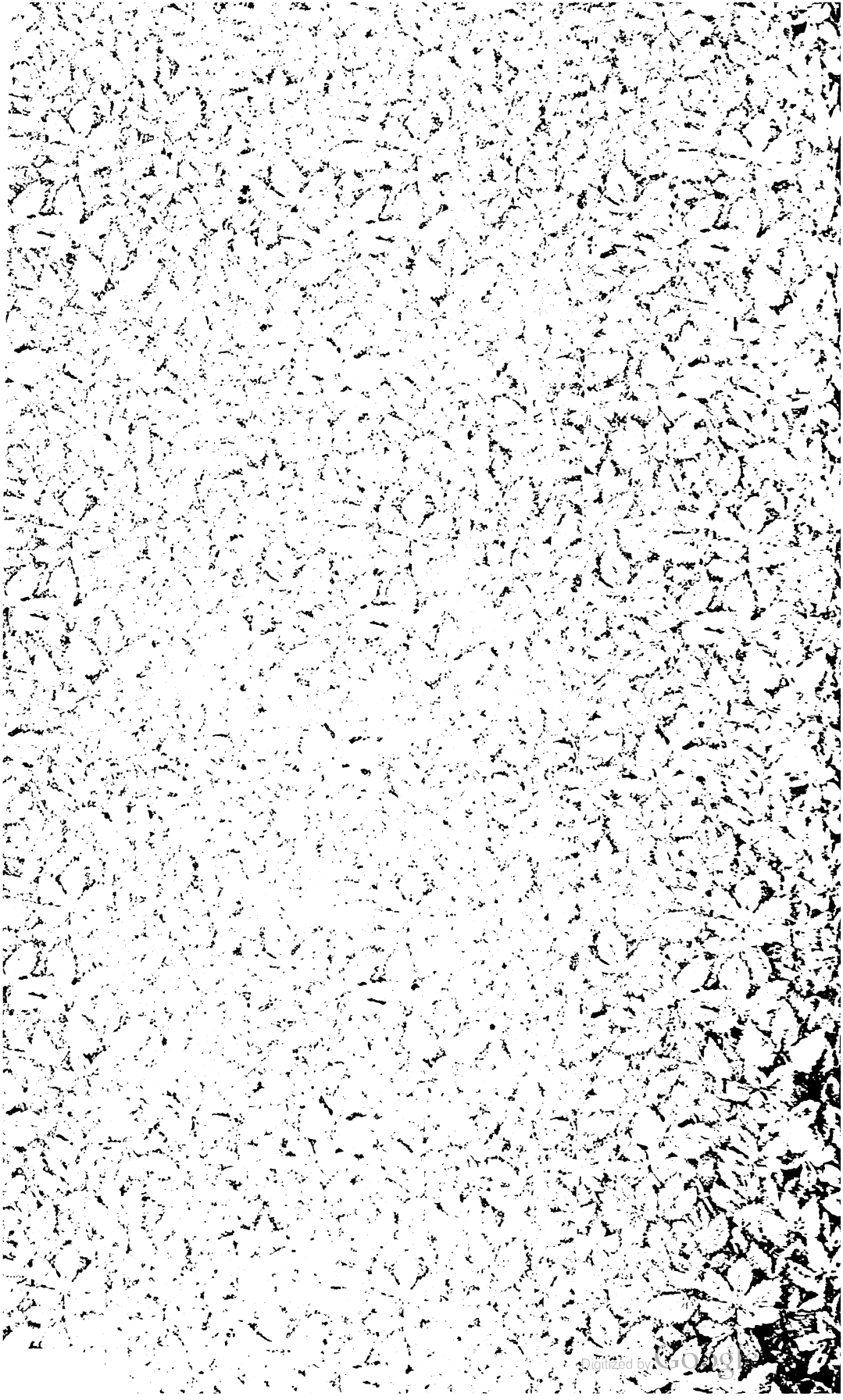
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Frauenleben und -leiden am Äquator und auf dem Polareise.

Von

F. v. Winckel,

München.

Seit das Deutsche Reich einen großen Kolonialbesitz erworben hat, hat es auch die Pflicht übernommen, sich zunächst mit den zahlreichen Völkerschaften, die diese Kolonien bewohnen, genau bekannt zu machen. Wir stehen jetzt erst in den Anfängen dieser Kenntnisse und das, was wir bisher von denselben erworben haben, ist herzlich wenig. Dabei ist das uns bereits Bekannte auch nur zum kleinsten Teil durch Ärzte, zum größten Teil dagegen durch Reisende, Missionare, Kaufleute, Farmer und Offiziere erworben und recht häufig, namentlich was ärztliche Angaben betrifft, auf Mißverständnissen beruhend. Immerhin bildet es eine Basis, von der die weitere Forschung ausgehen muß; jedes Erforschen ist nur eine Stufe zu etwas Höherem. Es ist nun interessant zu beobachten, wie die vorhin erwähnten Laien bei ihren Wahrnehmungen sich oft auch eingehend mit dem weiblichen Teil jener Völkerschaften beschäftigt haben und uns in ihren Schriften eine Menge von Tatsachen, die zur allgemeinen Gynäkologie gehören, mitgeteilt haben. Ich denke dabei namentlich an die Publikationen des Grafen Joachim Pfeil, Studien und Beobachtungen aus der Südsee, Braunschweig 1899, welche sich auf die Kanaken beziehen, ein Volk, welches noch heutzutage in der Steinzeit lebt. Mir schweben ferner vor die Wanderungen und Forschungen im Nordhinterlande von Kamerun, die Franz Hutter, ein baye-

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rischer Artilleriehauptmann, bei einem hochkultivierten Negervolke 1891—1893 machte und 1902 publizierte. Mit diesen beiden Schriftstellern und ihren Beobachtungen an am Äquator lebenden Völkern möchte ich dann die Beobachtungen von Schliephake (1885), v. Nordenskjöld (1886), Nelson (1899), Nansen (1903) und Amundsen (1907) an den Frauen der arktischen Zone vergleichen, da die letzteren, die Eskimos, zum größten Teil auch noch in der Steinzeit leben und die Bedingungen für ihre Existenz und ihr Fortkommen so außerordentlich von denen jener äquatorialen Völker verschieden sind, daß man a priori die Einwirkung des Klimas, des Lichtes, der Ernährung und Kleidung, der Beschäftigung, der Bewegung zu erkennen vermuten könnte, und jedenfalls gewaltige Verschiedenheiten zwischen jenen und diesen in jeder Beziehung für sehr wahrscheinlich halten könnte, was aber, wie wir bald erkennen werden, keineswegs den Tatsachen entspricht.

Beginnen wir zunächst mit dem Werke von Franz Hutter, welches von Forschern wie Wißmann als ein ganz ausgezeichnetes anerkannt worden ist, so hat derselbe, obwohl er auch kein Arzt, sondern ein Laie ist, eine Menge trefflicher sozialer, anthropologischer und ethnographischer Wahrnehmungen in den Jahren 1891—1893 in jenen Ländern gemacht.

Es liegt auf der Hand, daß bei dem Verkehr mit wildfremden Völkern die Heilkunde immer eine große Rolle spielen muß, und daß diese gerade in Krankheiten bei dem Fremden Hilfe und Rettung suchen. So wurde denn auch Hutter bei einer großen Ruhrepidemie im Balidorf im Jahre 1892 Tag für Tag zu solchen Kranken geholt und ist von Haus zu Haus mit Opium und Dowerschen Pulvern, mit Milch und Liebigs Fleischextrakt und mit Karbolsäure zur Desinfektion gewandert. Ja, noch mehr, eines Tages erschien ein hochschwangeres Weib in seiner Hütte mit der Bitte, er solle ihr bei ihrer Niederkunft helfen. Darauf hatte er sich zu Hause nun doch nicht vorbereitet und so sagte er ihr denn auch, daß er davon nichts verstünde. Aber schon seine Gegenwart beruhigte jene und so bat sie ihn, gleichwohl in seinem Hause bleiben zu dürfen. Nachdem ihr dieses gestattet, ging die Geschichte vor sich unter seiner passiven Assistenz, was, wie er selbst meinte, wohl das beste war.

Ehe wir aber feststellen, was Hutter in anthropologischer, ethnographischer, ärztlicher und gynäkologischer Beziehung uns Neues gebracht hat und wir seine Beobachtungen an der Negerin mit denjenigen des Grafen Pfeil an der Kanakin vergleichen, müssen wir einige geschichtliche Daten vorausschicken: Am 14. Juli 1884 hißte Dr. Gustav Nachtigal als Reichskommissär unter dem Donner der

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Kanonen der „Möwe“ am Kamerunflusse die deutsche Flagge und stellte damit dieses Gebiet unter Schutz und Oberhoheit des deutschen Kaisers. Nachdem der Kaufmann Robert Flegel 1879, 1882—1885 verschiedene Reisen auf dem Benuë gemacht hatte, wurden von Dr. Eugen Zintgraff 1886, 1887—1889 und 1891—1893 drei Expeditionen in das Nordhinterland von Kamerun unternommen, deren erste 1886 die Inangriffnahme der Erforschungen der unbekannten Hinterländer Nordkameruns begann, deren letzte aber durch die siegreichen Batut- und Bandeng-Aufstände fast vernichtet wurde. Infolgedessen schickte das Auswärtige Amt den Hauptmann Hutter mit Waffen und Munition zu seiner Hilfe. Dieser traf im Juni auf der Barombistation mit Dr. Zintgraff zusammen. Hier wurde der Plan zu einem neuen Vordringen nach der Station Baliburg entworfen, von der Zintgraff vertrieben worden war. Dieser Zug gelang ihnen und sie trafen am 25. August 1891 wieder in Baliburg ein, so daß Hutter, nach Einrichtung der Station von Anfang des Jahres 1892 an, seine wissenschaftlichen Beobachtungen daselbst anstellen konnte. Leider aber wurden diese bereits am 1. Januar 1893 auf Befehl des Auswärtigen Amtes wieder abgebrochen und der Rückmarsch zur Küste angetreten.

In dem ganzen Werk von Hutter ist nun bloß vom Nordhinterland von Kamerun, vom Monga-Maloba bis zum Benuë die Rede.

Hutter macht mit Recht darauf aufmerksam, daß gründliche und richtige Einblicke in ethnographische, kulturelle, soziale und sprachliche Verhältnisse nur durch monate- und jahrelange Arbeiten auf einer Station zu erwerben seien. Auf dem Marsche käme naturgemäß nur die geographische Forschung, insbesondere die Wegeaufnahme zustande, das übrige werde höchstens auf Grund von Erzählungen und Ausfragen der Eingeborenen ermittelt, ohne es durch persönliche Beobachtungen und Vergleichungen auf seinen Wert und Unwert prüfen zu können. Er unterscheidet weiter die hinter der Küste gelegene Waldlandschaft mit ihren hohen Temperaturen, ihrem feuchten Boden, ihren schlechten Wegen durch den Urwald und sein Dunkel von der höher gelegenen Graslandschaft, wo der Urwald geendet hat, mannshohes Gras, ein harter Boden, eine dichtere, höher kultiviertere und gesündere Bevölkerung sich befindet, in deren Mitte die Station Baliburg liegt. Bei Schilderung derselben wendet er sich zunächst zur Besprechung und Widerlegung zahlreicher falscher Vorstellungen über den Körper und Geist des Negers, die meist in der Überhebung der weißen Rasse ihren bewußten oder unbewußten Grund hätten. Der Neger sei durchaus nicht so häßlich, wie allgemein angenommen werde, sein Körper sei unverhüllt und lasse sich mit seinen schönen Formen ebenso wie mit seinen

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brechen leicht übersehen und Hutter sagt, er stehe nicht an, geradezu zu behaupten: der vorurteilsfreie Vergleich eines geistig und körperlich gut entwickelten Vertreters aus dem Volke eines weißen (kaukasischen) Stammes und eines geistig und körperlich gut entwickelten Vertreters eines der Hochlandstämme im Hinterland von Kamerunfalle nicht zum Nachteile des Letzteren aus. Vergleiche er aber minderwertige Repräsentanten der beiden Farben, so komme er zu demselben Ergebnis, wovon er nur die Küstenbevölkerung, die Dualla, ausnehme.

Je weiter Hutter ins Innere vordrang, um so mehr wuchs die Bevölkerungsdichtigkeit — auf 100 km 10000, auf den nächsten 70km 20000 — und auch die eigene, nicht dem Weißen abgelernte und abgeschaute Kultur.

In anthropologischer Hinsicht bemerkt Hutter als auffallend die geringe Breite der mittleren Körperpartie um Hüften und Becken, namentlich beim weiblichen Geschlechte, so daß man, hinter einer Anzahl Neger gehend, auf den ersten Blick die beiden Geschlechter nicht zu unterscheiden vermöge.

Die bei den Männern oft so stark entwickelte Nackenmuskulatur sei ebenso wie das häufig übermäßige Hervortreten des Unterleibs bei Weibern und Kindern und die damit vielfach verbundene starke Krümmung der Wirbelsäule, eine Folge frühzeitiger, starker Arbeit und schweren Tragens.

Die Ausdünstung des Waldlandnegers sei ihm, wenn derselbe nicht schwitze, nicht aufgefallen; wenn er aber schwitze, so sei der Geruch ein widerlich süßlicher; übrigens sei der Schweiß auch bei den Weißen bekanntlich nicht wohlriechend; ferner sei der Neger im Wald- und Grasland sehr reinlich: wo immer nur ein Bächlein riesle, ein Tümpel sich finde, da bade und plätschere groß und klein fleißig und mehrmals des Tages, während bei uns doch die Wasserscheu in breiten Schichten der Bevölkerung herrsche.

Im Gegensatz zu den Negern fand Graf Pfeil bei den Kanaken völligen Wassermangel, da auf den kleinen Inseln des Bismarckarchipels überhaupt keine Quellen sind und die Bewohner das wenige Wasser, welches sie benötigen, dadurch erhalten, daß sie in der Nähe des Strandes Löcher in den Sand graben, in die langsam ein wenig Seewasser einsickert. Es wird dadurch filtriert und seines Salzgehaltes beraubt, behält aber trotzdem einen so unangenehmen Geschmack, daß es für Europäer völlig ungenießbar ist. Hieraus erklärt er auch die große Unsauberkeit der Kanakin und ihr geringes Reinigungsbedürfnis, womit übrigens ihr öfters von ihm erwähntes Baden in der See nicht recht übereinstimmt.

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Die Ansiedelungen im westafrikanischen Urwalde, wozu natürlich auch die Pflanzungen, die Farmen, gerechnet werden müßten, seien nach Hutter so recht deutlich ein Gegenbeweis gegen die gedankenlose Mär von der Faulheit des Negers; sie widerlegten aufs gründlichste die Redensart, daß in den Tropen den Leuten alles einfach in den Mund wachse. Die Ansiedler hätten von jeher schwer arbeiten müssen und müßten es auch jetzt noch, zumal mit ihren primitiven Messern und Äxten.

Als Beweis der Reinlichkeit der Neger schildert Hutter auch die Anlage und Benutzung ihrer Aborte. An verschiedenen Stellen seien lange Schneusen in den Wald gehauen, an deren Enden lange Baumstämme auf dem Boden oder auf niedrigen hölzernen Gabeln lägen; dahinter befänden sich meist tiefe Gruben, also genau wie die Latrinenanlagen bei unseren Truppenbiwakplätzen. Aber die Art und Weise der Benutzung habe ihn, als er sie das erstemal gesehen, in einen minutenlangen Lachanfall versetzt. Sie fände nämlich nicht in der bei uns gebräuchlichen Art statt, sondern Männlein und Weiblein durcheinander stiegen rücklings vorsichtig und bedächtig hinauf, um dann, glücklich oben angelangt, die Hockstellung mit tiefer Kniebeuge einzunehmen. Wie Spatzen auf einem Telegraphendraht habe ihn so ein Gruppenbild angemutet. Übrigens sei in jedem Graslandgehöfte ein großer Lehmtopf eingegraben, der, mit Gras bedeckt, von Zeit zu Zeit in den Bach oder sonstwohin entleert werde.

Als auffallend bezeichnet Hutter die Scheu des Negers, dem Weißen beim Essen zuzusehen; wolle man sich von der oft so lästigen Neugier der Eingeborenen befreien, so brauche man nur Anstalten zum Essen zu treffen: sofort ziehe sich alles zurück. Auch würden nach den Mahlzeiten sorgfältig die Zähne gestochert und sehr häufig noch des weiteren durch Reiben mit Holzstückchen gesäubert.

Die Tätowierung kommt bei den Weibern der Banyangs ebenso wie bei den Männern, aber ausnahmslos in ornamentaler Art vor; in gleicher Weise fanden sie Graf Pfeil und Meyer und Parkinson bei den Kanakinnen. Aber während die Kanakin ihre Zähne meist gründlich schwärzt und es besonders modern bei derselben ist, die der rechten Seite des Ober- und der linken Seite des Unterkiefers zu schwärzen und die anderen weiß zu lassen und umgekehrt, fand Hutter bei den Negerinnen keine Spur von Färbung, dagegen wurden ihnen die oberen Mittelzähne ausgebrochen und vielfach die beiden unteren Mittelzähne zugespitzt.

Was Nachtigal von den Baghirmis sagt, hebt Hutter auch von den Balinegern hervor: daß sie psychisch und intellektuell aus-

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gezeichnet veranlagt seien, und daß kein Gedanke an irgendwelche körperliche oder geistige Minderwertigkeit oder Vernachlässigung seitens der Natur aufkommen könne.

Es komme bei den Negern Sklaverei vor, aber die Form derselben sei milde, das gehe schon aus der Sitte hervor, die Sklaven in eigenen Dörfern wohnen zu lassen. Auch hier habe die Hausklaverei ein patriarchalisches Verhältnis angenommen. Man dürfe überhaupt die Bewohner eines Landes, in dem die Eltern ihre Kinder zum Verkauf als Sklaven anböten, wie Hutter selbst es einige Male im Waldlande erlebte, nicht mit unserem bisweilen übertriebenen Gefühlsmaßstabe messen. Sklavenjagden gäbe es nicht, nur Einzelverkauf und Wegfangen einzelner, sowie Verwendung kriegsgefangener Männer und Weiber; wobei aber zu bemerken sei, daß die Kriege nicht zu diesem Zweck geführt würden.

Übrigens herrsche im Kriege Mordgier und Beutelust, Weiber und Vieh würden weggeschleppt und Hutter beobachtete, wie auf dem Marktplatze eines erstürmten Dorfes zwei Balikrieger sich um ein Weib rauften, der eine zog sie am Arme, der andere an ihren Beinen. Dabei war dem einen durch einen furchtbaren Hieb das ganze Gesicht zerfleischt, der andere aber hielt sich mit der einen Hand die aus einer breiten Bauchwunde hervorquellenden Eingeweide, ihre Beute aber ließen sie nicht fahren.

Menschenfresser sind die Balineger nicht, während die Kanaken in der Tat noch das Menschenfleisch mitsamt ihren Frauen als eine große Delikatesse betrachten.

Die tödlichste Beleidigung, die der Bali seinem Gegner zufügen könne, sei die Drohung, daß er die Geschlechtsteile seiner Eltern verstümmeln werde, offenbar damit letztere unfähig gemacht würden, nochmals einen solchen Menschen hervorzubringen.

Noch eine den Negern überhaupt aufoktroyierte Eigenschaft bespricht Hutter, und das ist der ihm angeblich innewohnende übermäßige Geschlechtstrieb. Bei den ihm bekannt gewordenen Stämmen sei derselbe jedenfalls nicht vorhanden gewesen. Auch hierin dürften wir uns nicht über den Schwarzen höherstehend dünken, jener sei eben, wie bei uns auch, individuell verschieden.

Unter den vielen Momenten, die dem Europäer in den Tropen besonders gefährlich seien, deren Opfer meistens dem sogenannten tückischen Klima aufgebürdet würden, nennt Hutter besonders den übermäßigen Alkohol- und Geschlechtsgenuß. Er sei, sagt Hutter, kein Mäßigkeitsvereinler und ein guter Bayer, aber gerade das schwere bayerische Exportbier halte er für das tödlichste Gift unter den Tropen.

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Ein mäßiger Genuß von Alkohol sei als anregendes Mittel in den südlichen Breiten bisweilen nützlich, sogar notwendig.

Von jeher war das Klima der westafrikanischen Küste berüchtigt und gefürchtet wegen des so oft tödlichen Fiebers, der Malaria. Die Gefahr desselben, meint Hutter, werde allerdings überschätzt, da hauptsächlich die ungesunde Lebensweise des Europäers die Schuld an derselben trage, dann aber auch die Unvorsichtigkeit, längere Zeit sich ohne Kopfbedeckung den senkrechten Strahlen der Äquatorialsonne auszusetzen. — Eine ebenfalls in den westafrikanischen Tropen recht häufig vorkommende Krankheit ist die Ruhr, die stets bedenklich und ernst zu nehmen sei, auch weil sich nicht selten ein Fieberanfall dazu gesellt. Sie herrsche auch unter den Negern nicht selten epidemisch, und 1892 fielen ihr im Februar und März vom Balistamme unter 6000 Köpfen nicht weniger als 600 Leute zum Opfer.

In dritter Reihe sind nach Zahl und Häufigkeit ihres Auftretens im Innern Kameruns Hautkrankheiten mannigfacher Art, fast die erste Stelle einnehmend, von denen Hutter auch oft in qualvoller Weise heimgesucht wurde. Auch bei den Kanaken kommen Malaria, Dysenterie, Hautaffektionen, wie wir durch Graf Pfeils Werk erfahren, recht häufig vor.

Wenn Hutter beobachtet zu haben glaubte, daß bei beiden Geschlechtern der Neger Nabelbrüche sich häufig fänden, so werden sie ihm wohl wegen der mangelnden Bekleidung häufiger aufgefallen sein, da der Europäer auch recht oft daran leidet, sie aber verdeckt trägt.

Mißbildungen des ganzen Körpers wie Höcker, Verwachsungen u. dgl. sah Hutter beim Neger nie, wohl aber öfter Elephantiasis scroti mehrmals bis zur Größe eines Kürbis; merkwürdig, daß er dieselbe nicht auch an den weiblichen Genitalien beobachtete. Einigemal fand er bei beiden Geschlechtern stets zwischen der großen und der zweiten Zehe eine sechste Zehe, aber ohne Nagelglied. — Krebs fand Hutter einige Male im Banyangland bei den Frauen an den Brüsten, bei den Männern an der Nase. Daß Geschlechtskrankheiten existierten, schloß Hutter aus der Anwendung pflanzlicher Mittel gegen dieselben, jedoch sollen sie nur leichteren Grades sein, wahrscheinlich Gonorrhöe, wogegen die Banyangs einen Absud der Blätter des Waldbaumes, genannt Ndakwa, verwendeten.

Gegen Erkältung wird Pfeffer gekaut und gegessen. Gegen Unterleibsbeschwerden wird heißes Palmöl innerlich und äußerlich gebraucht und außerdem werden Klistiere in einer nachher zu schildernden Weise gesetzt.

Bei der Ruhr suchen die Neger durch Tanz, Geschrei und Lärm-

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machen mit allen möglichen Instrumenten die „Krankheit in den Busch zu jagen“.

Palmwein, Palmöl und Kolanuß spielen bei verschiedenen Krankheiten eine große Rolle, außerdem aber auch Umschnürung und Abschnürung des erkrankten Gliedes; dann ferner Bähungen mit dem Mist der Haustiere.

Skarifikationen werden an der linken Seite gegen Milzvergrößerung ausgeführt und Schröpfen mit Aufsetzen von kleinen Antilopenhörnchen; die Blutung wird durch Auflegen von Mist gestillt.

Die allgemein übliche Beschneidung der Knaben wird erst im Alter von 10—12 Jahren vorgenommen und die Wunde gleichfalls mit Mist verbunden.

Von einer Kaste von Ärzten oder Medizinmännern existiert bei den Balis nichts, wohl aber eine große Menge abergläubischer Gebräuche, die den bei uns noch oft angewandten Sympathiemitteln in vieler Beziehung gleichen.

Die Hauptfeinde des Menschen aus der Tierwelt sind nicht die Leoparden, Elefanten, Krokodile und Schlangen, sondern die Ratten, Ameisen, Sandflöhe und Fliegen. Namentlich die Sandflöhe bohren sich heimtückisch unter die Nägel der Zehen, erzeugen dort Geschwüre und machen den Menschen oft für Wochen vollkommen marschunfähig. Sie sollen, ein Gastgeschenk Amerikas, erst Anfang der siebziger Jahre durch ein Schiff an der afrikanischen Westküste eingeschleppt worden sein.

Nach dieser mehr allgemeinen Einleitung wenden wir uns nun zu den uns hier am meisten interessierenden Beobachtungen Hutters an der Wald- und Graslandnegerin und begleiten diese von der frühesten Kindheit bis zum Tode, indem wir auch hier die Angaben Hutters mit denen des Grafen Pfeil über die Kanakin vergleichen.

Eine Kindererziehung in unserem Sinne gibt es bei der Negerin nicht. Die meisten Mütter stillen wohl ihre Kinder selbst, gewöhnlich fast ein Jahr; bei den Kanaken werden die Kinder gewöhnlich bis sie laufen können gestillt, ja manchmal, wenn keine neue Schwangerschaft eintritt, bis zum vierten Jahre. Nicht selten wird neben der Mutterbrust noch andere Nahrung gegeben; so beobachtete Hutter einmal, wie eine in seiner Nähe hockende, völlig nackte Negerin ihr Kind fütterte, indem sie ihm bald die Brust reichte, bald im Munde zerkauten Mais in seinen Mund spuckte. Diese ekelhafte Sitte des Vorkauens ist nicht bloß bei den Negerinnen beobachtet, sondern kam vor nicht langer Zeit auch noch bei deutschen Ammen öfters vor. Graf Pfeil sah im Bismarckarchipel, wie ein Kanake ein etwa einjähriges Kind mit großen Mengen junger Kokosnuß fütterte, die

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es gierig verschlang; das Kind verzehrte fast eine ganze Kokosnuß, ohne die mindeste Verdauungsstörung dadurch zu bekommen. Bei Unterleibsbeschwerden der Kinder werden ihnen von der Negerin Klistiere gesetzt, indem sie ein ungefähr 20cm langes, hornähnliches Holzrohr mit dem spitzen Ende in den After einführen, oben Wasser hineinschütten und nun, den Mund in den Trichter pressend, statt einer Spritze aus Leibeskräften das Wasser in den Darm blasen; ebenso werden auch Erwachsenen Klistiere gegeben.

Zur Arbeit werden die Säuglinge, die noch nicht laufen können, mitgenommen, indem sie rittlings auf der Hüfte der Mutter oder im Rücken auf einem um die Stirn der Mutter und unter dem kleinen Allerwertesten führenden Bande getragen, sich mit ihren Händchen an Arm, Schultern und Brust anklammern. Die Kanakin schleppt ihre kleinen Kinder auch in reitender Stellung auf ihrer Hüfte mit; hier fixiert durch ein in Form eines Schals um sie geschlagenes, auf der entgegengesetzten Schulter geknüpftes Pandānusblatt. Graf Pfeil bemerkt dabei, daß, solange die Kanakenkinder klein seien, sie mitunter ganz niedlich wären, obwohl sie den Negerkindern in keiner Weise zur Seite gestellt werden könnten.

Bei den Balinegern überwiegen die Langschädel; doch bezweifelt Hutter, ob dies von Geburt aus das gewöhnliche sei und nicht bloß zustande komme, weil es bei den Negerinnen allgemeine Sitte sei, den kleinen Kindern durch Streichen und steten Druck eine langgestreckte, eiförmige Kopfgestalt zu geben.

Der Eintritt der Pubertät wird bei der Negerin festlich begangen. Sie muß sich um diese Zeit in ihre Behausung zurückziehen. Dann wird ihr Menstrualblut in einer Schale aufgefangen, die ganze Verwandtschaft sammelt sich im Gehöft der glücklichen Eltern und besichtigt in Abwesenheit des jungen Mädchens die herumgezeigten Beweise.

Bei der Beschneidung der Knaben findet dagegen keine Festlichkeit statt, ebensowenig bei der Geburt eines Kindes und dessen Namengebung.

Die Ehe trägt den monogamischen Charakter; daher gibt es nur eine unter gewissen Zeremonien geheiratete Frau als legitime Gattin. Die Ehe zwischen Blutsverwandten auf- und absteigender Linie und zwischen Geschwistern ist verboten, gleiche Bestimmungen gelten nach Graf Pfeil auch bei den Kanaken. Dem Manne der Negerin ist der Verkehr mit Sklavinnen gestattet, in der Zeit, während sich seine Frau der Kohabitation enthält, z. B. während der Regel, während eines Teils der Schwangerschaft und solange sie ihr Kind stillt. Nicht gestattet ist dem Manne der Verkehr mit andern verheirateten

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Frauen oder anderen als Sklavenmädchen. Während aber beim Weibe der Ehebruch schwer, oft mit dem Tode bestraft wird, muß der Verführer nur eine schwere Buße zahlen. Das Verhalten der Kanaken ihren Frauen gegenüber hat Graf Pfeil nicht ganz klar dargestellt. Denn wenn er sagt: Ehescheidungen sind bei ihnen nicht häufig, Ehebruch wird als Unart betrachtet und durch eine Geldbuße bestraft — so steht damit in Widerspruch seine spätere Angabe: Eheliche Untreue kommt im allgemeinen selten vor, weil sie am Weibe mit Todesstrafe geahndet werden darf und die noch spätere: Die Weiber in Neumecklenburg hätten das Recht, ihre Gunst vor und nach der Heirat beliebig zu verschenken, in grellem Widerspruch. Vielweiberei ist nach Graf Pfeil bei den Kanaken durchaus die Regel, doch findet man selten mehr als zwei und kaum je mehr als sechs Weiber im Besitz eines Mannes. Die Weiber selbst sind durchaus nicht gegen, sondern für dieselbe, weil ihnen dadurch Teilnehmerinnen ihrer Arbeit in Garten und Feld erwachsen. Wie tief im übrigen die Kanakin unter der Negerin steht, geht aus der Angabe des Grafen Pfeil hervor, daß es die Miteinwohner eines Hauses gar nicht als lästig empfänden, Zeugen des geschlechtlichen Umganges von Ehepaaren zu sein, während Hutter mehrfach betont, daß der Geschlechtsakt von den Negern nie in der Öffentlichkeit vollzogen werde.

Die mosaische Forderung der Unberührtheit des Weibes kennt der Wald- und Graslandneger nicht; er bevorzugt bei der Wahl seiner Gattin sogar ein Mädchen, das bereits Kinder in die Ehe mitbringt, und urteilt von einer Maid ohne solche Aussteuer „Die scheine nicht liebenswert und fruchtbar zu sein, sonst hätte sie wohl schon einen Liebhaber gefunden", ganz wie die oberbayrischen Bauern.

Hutter fand bei den Graslandnegerinnen, im Gegensatz zu dem federnden, elastischen Gang der Männer, einen steifen unschönen Gang; dagegen die Gelenkigkeit der Füße und namentlich der Zehen, welche geradezu als Finger benutzt würden, um Gegenstände vom Boden aufzuheben, sehr überraschend, ebenso wie die schlanken Hüften an den meist schön gebauten Körpern der Weiber.

Trotz dieser schmalen Hüften solle das Gebären leicht und gut vonstatten gehen. Über das Verhalten bei der Niederkunft teilt Hutter mit, daß bei derselben dem eignen Manne der Zutritt zu der Hütte der Kreißenden untersagt sei. In dem einen, oben schon erwähnten Falle, den Hutter in seiner Wohnung beobachtete, war die Geburt in etwas mehr als drei Stunden in hockender Stellung beendet. Kind und Nachgeburt gingen gut ab. Die Nabelschnur wurde von der Kreißenden selbst, etwa 25 cm vom Kinde entfernt, mit den Nägeln abgezwickt und durchgerissen, eine Unterbindung derselben

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fand nicht statt. Gegen Abend kehrte die Mutter mit ihrem Neugeborenen wieder ins Dorf zurück. — Auch hat Hutter nicht selten gehört, daß Weiber, die in den Farmen von den Geburtswehen überrascht werden, ganz allein gebären und an dem gleichen Tage mit dem Kinde den oft stundenlangen Rückweg nach Hause antraten, eine Beobachtung, die man auch bei höher kultivierten Frauen, den Europäerinnen, wenn sie auf einem Wege, einer Fahrt oder bei der Arbeit auf dem Felde von der Geburt überrascht werden, häufig machen kann. Die hockende Stellung und das Abreißen der Nabelschnur soll bei den Negerinnen ganz allgemein gebräuchlich sein. — Anders ist das Verhalten der Kanakin bei ihrer Entbindung: Fühlt dieselbe den Tag ihrer Entbindung herannahen, so begibt sie sich an den Meeresstrand und wirft sich, belastet mit einem Stein, den sie in beiden Händen trägt, in die Brandungswelle. Diese ist mitunter so stark, daß ein Entgegenstemmen und Aufrechtstehen unmöglich ist; das Weib wird niedergeworfen, steht aber mutig auf, um sich von neuem der Brandung entgegenzuwerfen. Natürlich ist es unmöglich, dieses Spiel lange auszuhalten, ein- bis zweimalige Wiederholung genügt. Sie glauben sich damit eine leichte Entbindung und dem Kinde Wohlbefinden gesichert zu haben. Darauf zieht sich die Kreißende in die Hütte zurück, wohin ein bis zwei Freundinnen sie begleiten; während der Niederkunft nimmt sie eine kniende Stellung ein. Wie, nach Hutters Angaben, Mißbildungen des Körpers weder im Wald- noch in Grasland bei den Negern gefunden werden, offenbar weil daselbst mißgebildete Kinder gleich nach der Geburt getötet werden, so behauptet auch Graf Pfeil von den Kanakinnen, daß, da man niemals mißgestaltete Kinder bei ihnen zu sehen bekomme, dies auf die von den Müttern oft ausgeführte heimliche Tötung des Kindes zurückzuführen sei, indem sie durch Zuhalten seines Mundes dasselbe erstickten, oder so lange auf sein Herz drückten, bis es zu schlagen aufhöre.

Auch in bezug auf die Fruchtbarkeit scheinen sich die Kanakin und Negerin sehr nahe zu stehen. Vier bis fünf Kinder seien bei der Kanakin schon sehr viel, drei sei die gewöhnliche Zahl — nach Pfeil; und Hutter bemerkt ebenfalls, daß die Fruchtbarkeit der schwarzen Rasse nicht so bedeutend sei, als man gewöhnlich annehme. Hutter fand als Durchschnittsstärke der Familie im engeren Sinne im Waldland 5—6 Köpfe, 2 Eltern und 3—4 Kinder, und ebenso im Grasland, wo die Bevölkerung ihm viel dichter und kultivierter erschien; der Unterschied zwischen diesen beiden Völkern und den viel höher kultivierten in bezug auf diesen Punkt ist also außerordentlich gering.

Das Verhalten der Brüste der Negerin schildert Hutter wie

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folgt: Im eigentlichen Grasland sitzen die Brüste der Weiber gewöhnlich auf schmaler Grundfläche auf, ragen weit hervor und endigen in zapfenförmiger Brustwarze. Man kann sie also im allgemeinen als stark, wenigstens als voll bezeichnen, die Form ist häufig konisch. Rasch senken sie sich aber und werden häufig bald hängend; bei jungen Mädchen aber sind sie fast stets straff und fest und man bekommt nicht selten bei solchen klassisch schöne, herb jungfräuliche Formen zu sehen. Bei den Bali-njong sah Hutter ein Weib, dessen rechte Brust tadellos geformt war, während die linke ganz schlapp weit herabhing. Aber die hängende Form in der Ausdehnung, wie sie im Waldlande häufig zu sehen, beobachtete Hutter bei den sehnigeren und mageren Graslandnegerinnen nie. Bei einer Waldlandnegerin fand er beide Brüste so schlaff, daß die linke bis zur Scham, die rechte bis über den Nabel herabhing, jede Brust eine lange Hautfalte und unten daran kugelförmig noch aufgetrieben mit auffallend langer Warze. Bei dem frühzeitigen Herabsinken der Brüste ist offenbar der Mangel der Kleidung und wohl auch das lange Stillen der Kinder schuld. In Mabesse sah Hutter ein Weib mit ganz zerfressenen Brüsten: die eine bot eine große eiternde Fläche dar, die andere hing „in Fetzen“ herunter; ob hier Karzinom oder Mastitis vorlag, ist aus der Beschreibung nicht mit Sicherheit zu entnehmen.

Wie in Deutschland zur Zeit der Minnesänger, so herrscht bei den Negern jener Gegenden noch jetzt eine Sitte, wonach der Häuptling eines Dorfes für den angekommenen Weißen außer Ziegen, Schafen, Schweinen, Hühnern, Palmwein und Durrabier auch junge Frauen und Mädchen seines Haushaltes als lebende Gastgeschenke sendet. Hierin haben die Waldstämme ein ziemlich weites Gewissen, indem das Oberhaupt der Familie oder der Ehemann nicht eben selten aus den Reizen seiner weiblichen Angehörigen durch Anbieten derselben zur Benutzung Kapital zu schlagen sucht und auch im Graslande ist die Sitte allgemein üblich, daß der Häuptling aus seinem Haushalte junge Weiber, die er als seine Töchter bezeichnet, was übrigens nicht wörtlich zu nehmen ist, zur geschlechtlichen Benutzung übersendet.

Wie wenig die Kleidung der Negerin als Verhüllung betrachtet wird, erfuhren Hutter und Zintgraff in drolliger Weise. Sie hatten nämlich zur würdigen Feier eines Tanzes zwei Stationsfrauen ein Paar weiße, lange Frauenhemden geschenkt; nun stolzierten dieselben damit an und als sie sich zu jener Füßen zum Palmweineinschenken niederkauerten, hoben sie in gänzlicher Verkennung ihres Zweckes die Hemden hoch bis über die Hüften, damit sie ja nicht schmutzig würden. Die jungen Mädchen und Frauen gehen bis nach der Geburt des ersten Kindes vollkommen nackt; höchstens daß sie bisweilen eine dünne

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Schnur um die Hüften tragen. Nachher haben sie an dieser Schnur handbreite kleine Schürzchen vorn und rückwärts. An Stelle der Bekleidung ist das fast ausnahmslos geübte Einreiben des Körpers mit Rotholz gebräuchlich. Ein Schamgefühl ist den Negern nicht angeboren, sondern wird ihnen höchstens anerzogen. Gibt man den Leuten Zeug zur Verhüllung der Scham, so verstehen sie nicht warum: der oder die eine hält es sich an den Kopf, sie drapieren es anderswo, weil es ihnen da besser gefalle, oder lassen es vielleicht am bezeichneten Platze, nur weil es dem Geschmack zufällig entspricht.

Worin besteht nun die tägliche Beschäftigung der Weiber? Hutter spricht von einer Arbeitsteilung zwischen den beiden Geschlechtern. Die Feldarbeit obliege fast ausschließlich den Weibern; beim Hausbau bestände ihre Arbeit lediglich im Wasserherbeischleppen. Dagegen werde das Korbflechten, die Anfertigung von Taschen, Stricken und Mützen, das Spinnen des Fadens mit der Spindel nur von den Männern besorgt, während die älteren Weiber die Anfertigung stärkerer Fäden ausführten. Das Herbeischleppen der Lebensmittel aus den Farmen, sowie von Feuerholz, die Zubereitung der Speisen und die Kinderpflege sei selbstverständlich auch Sache der Weiber. Das Fleisch werde in Wasser gesotten, oder in Palmöl gekocht, ebenso die Pflanzennahrung. Vielfach würden aus den Knollenfrüchten allein, oder auch mit Mais, Maismehl, Erdnüssen und Bananenmehl zusammengeknetet Klöße geformt, die ihre rundliche Form durch Reiben an den Bäuchen der die Küche besorgenden Weiber erhielten und in Palmöl gesotten würden. Aus der heißen Öltunke würden sie dann mit gespreizten Fingern ganz oder in Stückchen, die noch rasch mundgerecht geknetet würden, herausgefischt — jedenfalls eine recht appetitliche Bereitung.

Bei den Negern nehmen beide Geschlechter ihre Mahlzeiten in hockender Stellung gemeinsam ein, bei den Kanaken speisen dieselben nicht gemeinschaftlich, sondern die Männer zuerst und die Frauen bekommen nachher den Rest.

Im Behandeln der Haare sind Negerin und Kanakin wieder beinahe einander gleich. Bei der Kanakin werden dieselben entweder künstlich gekräuselt, oder auch ganz abgeschabt, so daß der Kopf kahl erscheint; an den Genitalien werden die Schamhaare meist von den Mädchen und Frauen selbst entfernt. Die Banyangweiber gefallen sich in den tollsten Haartrachten. Wollte man, sagt Hutter, sie alle beschreiben, so gäbe es ein eigenes Buch und ein noch dickeres mit Zeichnungen derselben, denn mit Worten könne man diese Figuren nicht wiedergeben — so im Waldlande. Bei den Balis rasieren sich die Weiber den Kopf meist ganz kahl, oder es bleibt in der Mitte ein

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von der Stirn nach rückwärts in den Nacken sich ziehender länglicher Haarwulst. Zur Zeit nach der Regel und nach Geburten rasieren sich die Weiber die Schamhaare, auch die Augenbrauen und die Haare in den Achselhöhlen; die abgetragenen Haare werden aber nicht weggeworfen, sondern in einem Blatte verbrannt. Das Rasieren selbst üben meist alte Frauen mit kleinen Messern aus, indem sie zuerst die betreffende Körperstelle mit Rotholz einreiben, mit Wasser befeuchten, dann die kleinen Messerchen mit ihren Schnittflächen aneinanderreibend schärfen und nun die Haare abschaben. — Beim Tragen von Lasten werden die schweren Körbe meist auf dem Kopf getragen: zur Verminderung des Druckes legen sich die Weiber kranzförmig aus Blättern geflochtene Polster zwischen Kopf und Korb, ganz wie bei uns die Bäuerinnen einen flachen Kranz aus Zeug auflegen.

Dem jüngsten wie dem ältesten Weibe ein schier unzertrennlicher Begleiter ist die Tabakspfeife. Hutter sagt, er habe nicht so leicht ununterbrochene Raucher gesehen, wie im Hinterlande von Nordkamerun. Alles rauche daselbst vom Kinde bis zum Großvater und zur Großmutter. Das Schnupfen komme dagegen im Hochlande nur in Bamesson noch häufig vor, sonst weit weniger im Waldlande. Auch bei den Kanaken rauchen beide Geschlechter aus kurzen tönernen Pfeifen leidenschaftlich und beginnen schon in sehr frühen Jahren. Graf Pfeil sah, wie ein Kanake einem Säugling sogar die Pfeife zwischen die Lippen steckte. Schnupfen und Kauen von Tabak will er dagegen niemals beobachtet haben.

Eine Hauptfreude ist der Negerin und Kanakin der Tanz. Bei den Kanaken tanzen Männer und Knaben allein, die Weiber aber mit den Mädchen zusammen. Die Negerin tanzt viel graziöser als die Kanakin, zum Teil aber auch sehr ausgelassen. Hutter beschreibt z. B. folgenden Tanz, den Nachtigal auch bei den Baghirmis beobachtete: Zwei Tänzerinnen wirbeln, sich fortgesetzt drehend und mit den Händen über dem Kopfe zusammenklatschend, aufeinander los und der Gipfel der Kunst besteht darin, in der letzten Drehung mit den Gesäßen aufeinanderzuprallen. Glückt der Zusammenstoß, so federn die elastischen Puffer oft derart, daß das Gleichgewicht oft bedenklich verloren geht, natürlich zum größten Gaudium der Zuschauer.

Grobsinnliches Gepräge zeigen zwei weitere Tänze. Die Weiber stehen in einer Reihe und wiegen sich tanzend auf der Stelle. Eine verläßt ihren Platz und kauert sich der Reihe nach vor jeder der andern nieder, die im Tanz fortfahren, während die Kauernde sie mit der Hand über den Bauch und namentlich an den Genitalien streichelt. Ab und zu neigt sich die also Geliebkoste mit dem Oberkörper nieder

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und reicht der Huldigenden die Brust, woran diese saugt. Derselbe Vorgang wird von jeder einzelnen mit allen wiederholt.

Der zweite Tanz ist so: Aus dem Halbkreis tritt eins der Weiber hervor und wiegt sich ein Bein vorwärtsgestellt auf die Hüften gestemmt im gleichen Takt wie die übrigen. Eine zweite folgt und Brust an Brust mit der Solotänzerin schiebt sie eins ihrer Beine zwischen die der letzteren. Zusammen ahmen sie nun die Bewegungen der Kohabitation nach. Der übrige Tanzkreis begleitet diese Bewegungen mit stets rascher werdendem Gesang, zuletzt wird von allen das Wort ntchakeni ausgestoßen, beide Tänzerinnen treten zurück und werden durch ein anderes Paar abgelöst. Übrigens tanzen auch bei den großen Festen der Neger beide Geschlechter stets getrennt.

Bei ihren Tänzen singen die Frauen bald laut, bald leise, in der Regel obszöne Lieder, zum Teil eintönig, hier und da aber mit einem gewissen Wohlklang und Rhythmus.

Die Negerinnen berauschen sich zuweilen im Palmwein, die Kanakin soll sich durch übermäßiges Betelkauen öfter einen tiefen Rausch verschaffen.

Endlich ist der Negerin wie der Kanakin auch ein bestimmtes Verhalten beim Tode ihres Mannes vorgeschrieben. Beginnt nämlich bei einem Neger der Todeskampf, dann rennen die männlichen Angehörigen bald vor das Haus hinaus, bald umtanzen sie das Sterbelager, schreien und spektakeln mit allen möglichen Musikinstrumenten, besonders mit der Rassel — 10—20 hohlen halbrunden an einem Ring befestigten Eisenstäbchen — um die bösen Geister zu verscheuchen; dabei beginnen die Weiber in der Hütte um das Lager kauernd Klagegesänge in eigenartigen bald einförmigen, bald gellenden Tönen. Diese Totenklagen werden auch nach dem Tode mit Unterbrechungen fortgesetzt. Der Leichnam wird in Stoffe bänderartig eingehüllt, die Beisetzung erfolgt in ausgestreckter Lage und stets in dem zu Lebzeiten bewohnten Hause. In Bamesson wird die Leiche meist unter der Schwelle eingegraben. Viel härter sind noch die Vorschriften für die Kanakin: Ehe der Leichnam ihres Mannes in ein Kanoe gelegt wird, verbleibt er mehrere Tage in dem bisherigen Wohnhause. Hier haben ihm seine Frauen Gesellschaft zu leisten und niemand darf währenddessen das Haus betreten. Den Frauen wird ihre Nahrung hineingereicht. Nur die hervorragenden reichen Kanaken werden in offenen Kanoes der Verwesung ausgesetzt, die weniger bedeutenden werden in ihren eigenen Häusern begraben. Der Erdboden wird aufgewühlt und der Körper langausgestreckt hineingelegt. Die Frauen müssen nun das Haus weiter bewohnen und ihr Hauswesen auf dem frischen Grabe des Verstorbenen weiterführen. Die Weiber

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färben bei jedem Trauerfalle ihre Gesichter schwarz, die Männer nur, wenn der Verstorbene ein Mann war. — Übrigens kommen auch Verbrennungen von Leichen in Neumecklenburg vor und ferner Versenkungen von Leichen in die See, indem dieselben mit einer aus Kokospalmenblättern geflochtenen Matte umhüllt und mit einem Steine beschwert werden. Frauen sollen daselbst stets ihre letzte Ruhestätte in dieser Weise finden.

So ist die Stellung der Frau bei den Kanaken denn doch immer viel niedriger wie bei den Negern: die Kanakin kann vom Mann verschenkt, verkauft, getötet werden, nur ihre materielle Stellung ist insofern als sie Alleinbesitzerin alles des, von ihr in die Ehe gebrachten Heiratsgutes bleibt und alles dessen, was sie sich während der Ehe zu erwerben vermag, besser wie bei der Negerin ja sogar bei der Europäerin. Durch diese ihre materielle Unabhängigkeit vom Manne und weil sie in der Ehe der fleißigere, mithin wohlhabendere Teil ist, ist das Entwürdigende ihrer sonstigen Stellung mehr ausgeglichen und sie steht doch dem anderen Geschlecht ziemlich selbständig gegenüber.

Auch die Negerin ist physisch und sozial tiefer stehend, als ihr Mann, für den sie zu arbeiten und dem sie Kinder zu gebären hat; aber drückend empfindet sie diese Stellung nicht. Ältere Neger, namentlich Häuptlinge, erweisen ihren alten Müttern oft eine geradezu liebevolle Verehrung. Solche Matronen sind dann bei den geheimsten Palavern zugegen und haben geradezu Sitz und Stimme im Rat und nicht zu unterschätzenden Einfluß. Wenn diese Vertrauungsstellung die Hauptfrau auch selten einnimmt, so kommt es doch auch vor. — Jedenfalls ist das Weib dem Neger zum mindesten ein wertvolles Eigentum und die Anwesenheit der Weiber in einem Dorfe gibt dem Weißen das beruhigende Gefühl, daß augenblicklich wenigstens keine Feindseligkeit geplant ist.

Man darf dabei aber auch nicht vergessen, daß die Kanaken noch auf der allerniedrigsten Kulturstufe stehen und heute noch in der Steinzeit leben, d. h. ihre Waffen und Geräte aus Muscheln und Steinen herstellen und weder Bronze noch Eisen kennen, während der Neger den Speer und das Gewehr führt, Hämmer und Äxte und Messer kennt und Eisen und Messing.

Wenn nun auch weder von Dr. Hutter, noch vom Grafen Pfeil in den von ihnen durchforschten Gegenden besondere sexuelle Anomalien wie Elephantiasis der weiblichen Genitalien, Lageveränderungen derselben, ferner namentlich die Existenz von größeren Geschwülsten wie Eierstocksgewächse und Tumoren des Uterus gesehen oder erwähnt worden sind, so ist es darum doch nicht wahrscheinlich, daß dieselben bei jenen Völkerschaften überhaupt nicht oder auch nur viel seltener,

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wie bei den höher kultivierten Völkern vorkämen. Es ist ja allerdings z. B. vom Gebärmutterkrebs längere Zeit behauptet worden, daß er bei den Negerinnen viel seltener, wie bei der weißen Frau vorkäme, aber mit Unrecht, denn sowohl durch Middleton Michel und J. S. Billings ist schon vor $1^1/_2$ Jahrzehnten festgestellt worden, daß die Mortalität an Krebs bei den Negerinnen ebensogroß wie bei den Weißen ist und mit den übrigen genannten Anomalien wird es wohl ebenso sein. Allerdings gibt es eine ausgezeichnete ärztliche Schrift über die akuten Infektionskrankheiten bei den Negern der äquatorialen Küsten Westafrikas von Dr. A. Plehn (Virchows Archiv Bd. 174 Splh.). Diese stützt sich auf Beobachtungen an 15000 Eingeborenen aus Kamerun und seiner Umgebung und hat festgestellt, daß die Neger der Bantufamilien verschiedenen Krankheitseinflüssen sich ziemlich übereinstimmend und vielfach anders, als die kaukasische und gelbe Rasse (Chinesen, Malayen) verhalten. So fehlen bei jenen z. B. Tuberkulose, Skrofulose und Rhachitis fast ganz; Syphilis trat vollkommen zurück. Während die auf Madagaskar lebenden malayischen Hovas von der Syphilis völlig durchseucht sind, haben die madagassischen Neger trotz reichlicher Infektionsgelegenheit nicht von der Syphilis zu leiden. Bösartige Neubildungen, Lepra sind sehr selten. Puerperalerkrankungen und Erysipel sind ebenfalls selten. Gonorrhöe ist außerordentlich verbreitet, soll aber sehr schnell heilen (?!). Charakteristisch für die Pathologie des äquatorialen Westafrikas, besonders des Kamerungebietes sei also das Zurücktreten der chronischen Krankheiten hinter den akuten. Und hierin liegt allerdings ein gewaltiger Unterschied zwischen diesen Völkerschaften und denen in Grönland und am magnetischen Nordpol lebenden, wie wir später erfahren werden.

Wenn wir uns nun vom fernen Süden nach dem äußersten Norden zu den Bewohnern des Polareises wenden und uns bei den Eskimos nach der Stellung, den Arbeiten und Pflichten des Weibes umsehen, so könnte es manchem meiner Leser erscheinen, als ob dieser Übergang zu unvermittelt geschehe, als ob zwischen der Kanakin, der Kamerunnegerin und der Frau des Eskimos wohl gar keine Verbindung bestände, als ob deren Existenzen viel zu verschieden seien, um überhaupt einen Vergleich miteinander zu gestatten. Dem ist aber durchaus nicht so. Es ist ein besonderes Verdienst von Fritjof Nansen, nachgewiesen zu haben, daß bei den grönländischen Eskimos Mythen existieren, deren Wiege im fernen Innern Asiens zu suchen ist, ja solche Mythen gefunden zu haben, die unzweifelhaft Grönland, Südafrika und die Fidschi-Inseln verbinden. Auch hat Nansen (l. c. p. 257 ff.) konstatiert, daß die Möglichkeit, daß solche Mythen an

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mehreren Stellen zugleich entstanden sein könnten, hier ausgeschlossen sein müßte, weil sie zu viele und zu charakteristische Übereinstimmungspunkte besäßen. Daraus schließt er also, daß der Verkehr zwischen den alten Norwegern und Eingeborenen Grönlands durchaus nicht so oberflächlich sein konnte, wie man gewöhnlich annehme.

Schicken wir aber, ehe wir auf den gynäkologischen Teil dieser Skizzen eingehen, einige Bemerkungen über die Eskimos im ganzen voraus, so rechnet Nansen sie (l. c. p. 16) zu den mittelgroßen Völkern und erwähnt von den Frauen, daß bei ihnen die schmalen Hüften und die auffallend kleinen Hände und Füße zu sehen seien; die ersteren erklärt er dadurch, daß die Eskimofrauen sogenannte runde, die Europäerinnen aber flache, breite Becken haben. Dasselbe sagt Hutter von den Kamerunnegerinnen, an denen er die schlanken Hüften und den meist schön gebauten Körper bewunderte. Was die Dichtigkeit der Bevölkerung betrifft, so zählte Nansen 1889 5614 Personen, darunter 2591 Männer, also 3025 Weiber, also auf einen Mann kamen 1,16 Frauen; dagegen fand Kapitän Holm (Nansen l. c. p. 104) in Imarsivik auf der Ostküste Grönlands auf 21 Einwohner nur 5 Männer. Im ganzen hat aber die Bevölkerung in den letzten Jahrzehnten in jenen Gegenden sehr abgenommen (von 30000 auf der Westküste vor $1^1/_2$ Jahrhunderten auf 10117 i. J. 1889). Denn während die in das Land übergesiedelten Europäer, die sich ja im Grunde von den Eskimos ernähren, sich oft Reichtum erwerben und im Überflusse leben, verarmen die Eskimos.

Die Gesetze, auf denen der grönländische Heidenstaat basiert, sind dabei nach Möglichkeit in der Praxis durchgeführter Sozialismus und in dieser Beziehung christlicher, als irgendein christlicher Staat.

Bei der Geburt eines Sohnes jubeln die Eltern, bei derjenigen einer Tochter sind sie unzufrieden. Aber die Eskimos hängen mit außergewöhnlicher Liebe an ihren Kindern und tun alles, was sie ihnen an den Augen absehen können. Sie werden auch sehr lange gestillt; 3—4 Jahre sind nicht ungewöhnlich, Nansen (l. c. p. 128) erlebte sogar 10—12 Jahre. Zuweilen versucht sogar eine Frau, die noch nicht geboren hat, ein ihr geschenktes Kind anzulegen, da erzählt Amundsen (l. c. p. 240) eine reizende Geschichte, wie eine solche dem Säugling rasch einen Schluck Wasser in den Mund aus dem ihren einfließen läßt. Bei der Gelegenheit erfahren wir auch, daß dieselbe Eskimofrau als Lutschbeutel dem Säugling ein einfaches Stück Speck in den Mund steckt, durch das ein langer Stecken gebohrt ist, damit jenes Stück nicht verschluckt werden kann. Jede Züchtigung der Kinder wird vermieden, deshalb sind denn auch die Kinder unter sich sehr verträglich, nie zänkisch. Die Mutter lebt mit den Kindern

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stets in denselben Räumen und trägt sie draußen auf ihrem Rücken im Amaut mit zur Arbeit. Bis das Kind volle zwei Jahre alt ist, wird es in diesem Sack nackt auf dem nackten Rücken der Frau getragen und Amundsen (l. c. p. 240) sah, wie Säuglinge beim Herausnehmen aus dem warmen Platz mehrere Minuten lang bei einer Temperatur von bis —50° Celsius splitternackt gehalten wurden, ohne daß es ihnen im geringsten schadete! Schon frühzeitig werden die Kinder an die Arbeit gewöhnt. Ein kleiner Knirps von 5—6 Jahren kann in einem Tage große Bündel Dorsche vom Angeln nach Hause bringen (Amundsen l. c. p. 245), kleine Bogenschützen vom Netschjillistamme bildet Amundsen auf p. 283 ab. Die Mädchen werden gleichfalls früh an ihren Beruf gewöhnt, sie lernen nähen und müssen der Mutter im Hause zur Hand gehen. Wenn die Eskimos sich begegnen, so begrüßen sie sich nicht nach unserer Art durch Küssen, sondern pressen die Nasen aufeinander und schnupfen sich zu. Mit den Eskimos teilen auch die Bewohner der Marquesas-Inseln (10° südl. vom Äquator und 14° westl. Länge) nach Langsdorff die Sitte, daß sie bei der Begrüßung die Nasen gegeneinander drücken (Stoll, Das Geschlechtsleben, l. c. p. 232).

Bei den Eskimos erklärt Amundsen dieses aus den unappetitlichen Beschäftigungen, die sie mit dem Munde ausführen; so bildet er eine Eskimofrau ab, die ihrem Manne mit den Zähnen den Stiefel an der Ferse abzieht (l. c. p. 243), wobei es sie gar nicht genieren soll, daß sie den Mund voll von dem Schmutz der Nässe bekommt, in die der Mann den Tag über getreten ist. Aber noch verblüffender wirkt es auf den Europäer, sagt Nansen (l. c. p. 110), wenn wir die Eskimofrauen ein Fell aus der stinkendenden Urintonne, die vor ihrer Türe steht, nehmen, hineinbeißen und dann zu bearbeiten anfangen sehen. Für sie ist der Mund die dritte Hand. Daher haben die alten Weiber dort oben auch auffallend kurze abgestumpfte Vorderzähne.

Merkwürdig ist die Rolle, die der Urin im Leben des Eskimoweibes spielt. Zuerst wird er zum Waschen der Haut benutzt: sie lieben den Geruch, der ihnen von alledem anhaftet, nennen ihn jungfräulich und halten ihn für ein wirksames Zaubermittel zum Anlocken der Männer; dann benutzen sie den Urin um das Haar geschmeidig und glänzend zu machen und, damit es sich in einen straff und aufrecht stehenden Knoten recht fest zusammendrehen läßt, baden sie es vor dem Frisieren in Urin; außerdem wird er zum Erweichen der Seehundfelle gebraucht. Hinsichtlich des Waschens mit Urin bemerkt Nansen (l. c. p. 242), daß dies ein uralter Gebrauch zu sein scheine. Man fände ihn schon in den heiligen Büchern der Parsen erwähnt. So stehe in der Vendidad 8, 13, daß die Leichenträger sich mit Urin

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nicht von Männern oder Weibern, sondern von kleinen Tieren oder Zugtieren waschen sollen. Das würde wohl eine religiöse Vorschrift sein und nicht wie bei den Eskimos sich aus dem Mangel an warmem Wasser erklären lassen.

Bei den Eskimomädchen tritt während der arktischen Nacht 4 Monate lang die Menstruation nicht ein und obwohl sie meist mit 14 Jahren heiraten und auch konzipieren, bekommen sie die Menses doch zuweilen erst mit dem 19.—20. Jahr (Cook, Newyork Journ. of gynecol. and obstetr. 1894 Febr.—April). v. Haven fand, daß von 100 Grönländerinnen 88 die erste Menstruation zwischen 15—17 Jahren bekamen, bei 5 trat sie früher, bei 7 erst nach diesem Alter ein und MacDiarmid hat von den Eskimoweibern behauptet, daß sie nur im Sommer ihre Regel hätten, er schreibt somit der Winterkälte eine hemmende Einwirkung zu. Von andern wird dieser Einfluß bestritten und bei den Eskimos, wo schon nach kaum eingetretener Pubertät ein reger Geschlechtsgenuß beginnt, wird dieser wahrscheinlich für den früheren Eintritt der Menses und deren Wiederkehr auch in der arktischen Nacht maßgebend sein. Der Vollbluteskimo verheiratet sich sobald er eine Frau versorgen kann, weil er weiblicher Hilfe bedarf, um seine Felle zu gerben, seine Kleider zu nähen. Er verheiratet sich oft schon bevor er zeugungsfähig ist, und auf der Ostküste von Grönland ist es nach Nansen etwas ganz gewöhnliches (l. c. p. 116), daß er drei- bis viermal verheiratet gewesen ist, ehe jener Zeitpunkt eintritt. Ehezeremonien finden nicht statt. Der Eskimo schleppt ein Mädchen, was ihm gefällt, ohne weiteres in sein Haus. Nach Amundsen (l. c. p. 242) soll das Mädchen, wenn sie 14 Jahre alt ist, den Bräutigam aufsuchen oder er zu ihr kommen und mit ihr im Hause der Eltern wohnen — so wäre es bei den Neochtjilli-Eskimos am magnetischen Nordpol. Nach Nansen aber (l. c. p. 117) durfte sich bei den grönländischen Eskimos das Mädchen unter keiner Bedingung merken lassen, daß sie den Freier haben wollte, selbst wenn sie noch so verliebt in ihn war, und mußte sich bei der Entführung sehr sträuben, jammern und klagen. Vielweiberei ist bei den Eskimos gestattet, die meisten guten Seehundsfänger haben daher zwei Frauen, aber nie mehr. Dalager erwähnt, daß seinerzeit auf der Westküste Grönlands kaum der zwanzigste Teil der Grönländer zwei, sehr selten drei und nur ausnahmsweise vier Frauen gehabt, doch habe er einen Mann, der elf Weiber gehabt, gekannt (Nansen l. c. p. 120 Anm.).

Bei den Eskimostämmen an der Beringstraße gelten nach Nelson die zum ersten Male menstruierenden Mädchen als unrein für 40 Tage. Sie müssen sich in einem Winkel des Hauses aufhalten, mit dem Gesicht gegen die Wand gekehrt, stets ihre Kapuze über ihren Kopf ziehen und ihre Haare wirr über die Augen hängen lassen. Das

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Haus dürfen sie nur des Nachts verlassen, wenn alles schläft. Im Sommer beziehen sie ein rohes Obdach außerhalb des Hauses. Nach Ablauf der vorgeschriebenen Frist baden sie, ziehen neue Kleider an und können nun geheiratet werden.

Bei dem Kuskokwin-Eskimo am untern Yukon werden nach Nelson häufig schon Kinder miteinander verheiratet und zwar die Mädchen oft mit 3—5 Jahren. Sie bleiben dann bei ihren Eltern und der junge Gatte zieht zu diesen in ihr Haus. Ebenso bei den Cumberland-Eskimos. Und während sonst, wie wir erfahren werden, die Ehen unter Blutsverwandten bei den Eskimos verpönt sind, heiraten die Unalit-Eskimos an der Beringstraße gern Cousinen und andere Blutsverwandte, weil man annimmt, daß diese bei Hungersnot ihre Nahrung mit dem Manne teilen, während eine Frau aus fremder Familie dem Manne die Vorräte stehlen würde (Nelson). — Nach Bessels kohabitiert der Inuit (Eskimo) mit besonderer Vorliebe von hinten. Männer wie Frauen liegen nackt in der Nacht dicht aneinander unter einem Seehundsfell, dem Gast macht man Platz, indem man nur ein wenig rückt. Die Frauen prostituieren sich während der Abwesenheit ihrer Gatten. Außerdem aber gibt es auch noch Professionshuren, jedoch selten.

Ehen zweier Geschwisterkinder untereinander, wie überhaupt zwischen nahen Verwandten, sind im allgemeinen nicht erlaubt. Nicht einmal Pflegegeschwister, die zufällig zusammen erzogen sind, dürfen sich heiraten. Dasselbe Verbot findet sich unter wenig verschiedenen Formen bei den Hindus, den Chinesen, in der griechischen Kirche, bei den Altkatholiken, zum Teil auch bei den Slavonen, den Indianern und vielen andern. — Die Scheidung der Ehe ist ebenso leicht fertig wie die Heirat. Ist der Mann seiner Frau überdrüssig, so braucht er sich nur von ihr zu retirieren, wenn er sich schlafen legt und dabei kein Wort zu sprechen, dann sammelt am nächsten Morgen die Frau ihre Kleider und kehrt in aller Stille in ihr Vaterhaus zurück.

Der Hauptzweck der grönländischen Ehe ist unbedingt die Kindererzeugung, unfruchtbare Weiber sind geringgeschätzt, unfruchtbare Ehen werden oft aufgelöst. Die Fruchtbarkeit der Grönländer und amerikanischen Eskimos ist gering, 2—4 Kinder in jeder Ehe ist die Regel, wenn auch Beispiele von 6—8, ja noch mehr vorkommen. Zwillinge sind selten. Die Niederkunft der Grönländerinnen ist gewöhnlich leicht, doch kommen bisweilen auch schwere Entbindungen vor; dann halten sie nach Egede (Nansen l. c. p. 127) der Gebärenden ein Nachtgeschirr über den Kopf in der Einbildung, daß die Entbindung dann leichter und schneller gehe. Eine solche Verwendung

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dieses Möbels, sagt Nansen, dürfte einzig dastehen. Die heidnischen Grönländer töten alle Mißgeburten, die für nicht lebensfähig gehaltenen kranken Kinder und alle kleinen, deren Mutter bei der Geburt stirbt, falls keine andere Frau sie säugen kann. Gewöhnlich werden die ersteren ausgesetzt oder ins Meer geworfen.

Nach Klutschack wird das Eskimoweib durch die Entbindung auf volle vier Wochen „unrein"; sie wird schon vier Wochen vor der Niederkunft von dem Gatten getrennt und in eine separate Behausung gebracht, zu der nur die Frauen Zutritt haben. Bei den östlichen Eskimos geschieht die Entbindung von dem ersten Kinde in dem gewöhnlichen Igloo (Hütte), bei allen späteren muß sie ein besonderes zu ihrem Gebrauch gebautes Igloo beziehen (Hall), der Mann darf bei der Entbindung nicht zugegen sein. Auch die in den westlichen Gegenden wohnenden Eskimofrauen müssen in einer kleinen Hütte gebären, in welcher sie zusammen mit dem Aas irgendeines Tieres eingeschlossen werden. In dieser Hütte bleibt die Kreißende ganz allein und ohne Hilfe. Smith besuchte mehrere dieser Hütten, deren jede eine Wöchnerin, ein Neugeborenes, und eine sehr kleine, die eine Hündin und einen Wurf junger Hunde enthielt. Übrigens durchschneiden die Eskimofrauen nach Holm den Nabelstrang mit einer Muschelschale, die Graslandnegerin zerreißt ihn mit den Nägeln.

Die Eskimofrauen kommen wegen ihres breiten Beckens leicht nieder und sterben sehr selten im Wochenbett. Nach der Niederkunft dürfen sie eine Zeitlang die Hütte nicht verlassen, bisweilen erst nach zwei Monaten; nachdem sie dann ihre Kleider, die sie nie wieder tragen, mit einem andern Anzug vertauscht haben, besuchen sie alle umliegenden Häuser, dürfen aber ein volles Jahr nicht allein essen. Bei den Grönländerinnen dürfen die Wöchnerinnen nicht unter freiem Himmel essen, aus ihrem Wassergefäß darf niemand trinken, niemand bei ihrer Lampe einen Span anzünden, und sie selbst dürfen eine Zeitlang nicht darüber kochen.

Auch darf der Mann, wenn seine Frau in Wochen liegt, außer dem allernötigsten Fang, nichts arbeiten, weil sonst das Kind sterben würde; er ist auch mit der Wöchnerin unrein: Residuen eines sogenannten Männerkindbettes.

Bei Unfruchtbarkeit wenden sie Hersagen von Zauberformeln an, oder binden Stücke von Schuhsohlen der Europäer um. Denn da sie sehen, daß diese fruchtbar sind, meinen sie durch die Sohlen, die einem Europäer als Unterlage gedient, werde deren Kraft in ihre Kleider übergehen usw. Aber sie benutzen auch einen Angekok d. h. einen ihrer Propheten und Gelehrten, die die Männer bezahlen, um der Unfruchtbarkeit der Frauen abzuhelfen. — Das Anbieten der

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Frauen der Eskimos als lebende Gastgeschenke, wie es im deutschen Mittelalter zur Zeit der Minnesänger ja auch noch vorkam, ist bei den amerikanischen Eskimos noch heute sehr gebräuchlich und Amundsen lernte nur einen Ogluli-Eskimo kennen: Nulieu, von dem er sagt: Ich glaube fast, er war der einzige Eskimo, der seine Frau nicht feilbot (Amundsen l. c. p. 247). Auch kommt der Frauenaustausch öfter vor. Trotz alledem behauptet Nansen, daß die Eskimofrau eine bedeutende Rolle in der grönländischen Gesellschaft spiele, da sie in der Regel gut behandelt werde und daß, wenn man mit ihr in ihrem Hause zusammenlebe, man durchaus nicht den Eindruck empfange, daß sie irgendwie unterdrückt oder zurückgesetzt würde.[1]) Alle Männer wie Frauen sind leidenschaftliche Anhänger des Kaffees und leider auch des Branntweins. Ihre Nahrung besteht übrigens aus Fischen, Seehundfleisch und Speck und Tran. Zu Zeiten großen Hungers kommt es übrigens auch vor, daß Frauen sogar Hundekot, weil in demselben Reste von unverdauten Fischen und Hundetalg sich fanden (Amundsen l. c. p. 277/278), aufspeisten.

Daß die Eskimofrauen sich auch einer widerwillig konzipierten Frucht zu entledigen verstehen, geht aus folgender Beschreibung Bessels hervor. „Ähnlich wie sich im missionarisierten Grönland die Schwangeren des Kaminstockes (ein Stück Holz zur Ausweitung der nassen Fußbekleidung) zu diesem Zwecke bedienen, so benutzen die Itanerinnen des Smith-Sundes entweder den Peitschenstiel oder einen anderen Gegenstand und klopfen oder pressen sich damit gegen das Abdomen, welche Prozedur des Tages mehrmals wiederholt wird. Eine andere Art der Abtreibung der Leibesfrucht besteht in der Perforation der Embryonalhüllen, einer Operation, die uns in gelindes Staunen versetzt. Eine dünngeschnitzte Walroß- oder Seehundrippe ist an ihrem einen Ende messerschneidenartig zugeschärft, während das entgegengesetzte Ende stumpf und abgerundet ist. Das erstere trägt einen aus gegerbtem Seehundsfell genähten zylindrischen Überzug, der an beiden Enden offen ist und dessen Länge derjenigen des schneidenden Teiles des Knochenstückes entspricht. Sowohl an das obere als an das untere Ende dieses Futterals ist ein 15—18 Zoll langer Faden aus Renntiersehne befestigt. Wird diese Sonde in die Vagina eingeführt, so ist der schneidende Teil durch den Lederüberzug gedeckt. Wenn die Operierende weit genug in die Geschlechtsöffnung eingedrungen zu sein glaubt, so übt sie einen sanften Zug auf den am unteren Ende des Futterals befestigten Faden aus. Hier-

1) Amundsen (l. c. p. 242) sagt: in Wirklichkeit sei die Stellung der Frau bei den Eskimos auf der Halbinsel Boothia Felix nicht mehr und nicht weniger als die eines Haustieres.

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durch wird selbstverständlich die Messerschneide bloßgelegt, worauf eine halbe Umdrehung der Sonde vorgenommen wird, verbunden mit einem Stoß nach oben und innen. Nachdem die Ruptur der Embryonalhüllen erfolgt, zieht man das Instrument wieder zurück; zuvor aber wird ein Zug am oberen Faden des Messerfutterals ausgeführt, um den scharfen Teil der Sonde zu bedecken und hierdurch einer Verletzung des Geschlechtskanals vorzubeugen.

Bessels erfuhr, daß diese Operation von den Schwangeren stets selbst an sich ausgeführt wird. (Ploß-Bartels, Das Weib, XIII. Aufl., I. 926, 1905.)

Nelson berichtet außerdem von den Eskimos an der Beringstraße, daß sie früher die Töchter, wenn sie keinen Mangel an denselben hatten, gleich nach der Geburt, oft aber auch erst im Alter von 4—6 Jahren töteten. Sie führten sie zu einem Begräbnisplatz, stopften ihnen Mund und Nase mit Schnee zu und ließen sie liegen, oder sie brachten sie auf das Eis oder in die Tundra und ließen sie dort in der Kälte oder im Schneesturm umkommen.

Die Hütten der Grönländer-Eskimos stehen auf dem ausgegrabenen Boden des Landes, in dessen Vertiefung ein schmaler, enger Gang hineinführt; das Dach ist flach, etwas gewölbt, $1^1/_2$—2 m über dem Boden. Die Eskimo in King Williams-Land bauen sich gegen Ende des Herbstes Wohnungen aus Eisquadern, die nach oben durch einen dreieckigen Eisblock geschlossen sind; sie werden im Winter durch die Specklampe erleuchtet und erwärmt. Im Sommer tritt an die Stelle der Eiskammern das Zelt. Dasselbe steht auf einem mit vielen Renntierfellen bedeckten Moosboden. Die Zeltwände selbst sind aus Renntier- und Seehundfellen zusammengenäht und werden durch große Steine am Rand der Zeltdecke festgehalten; in den Zelten soll es oft sehr warm sein.

Was die Frauenarbeit betrifft, so ist das Gerben, das Kajakbeziehen und Nähen der Häute, ferner das Abziehen der Seehunde und Zerlegen der Beute nach bestimmten Regeln und Verteilung derselben, dann das Bereiten des Essens, das Nähen der Kleider und Verrichten aller häuslichen Arbeiten, außerdem aber das Bauen der Häuser, Schlagen der Zelte und Rudern der Frauenboote ihre Aufgabe, daneben ist noch der Kapelanfang als einziger Fang, den die Frauen treiben, und manchmal eine gewisse Hilfe beim Seehundsfang zu erwähnen. Das Ausnehmen der erlegten Vögel, und, wie erwähnt, das Ausziehen der Pelzstiefel des Mannes kommt ebenfalls der Frau zu. Die Frauen — nur ein einziges Mal sah Amundsen (l. c. p. 321) einen an der Nase tätowierten 25jährigen Mann, der von seiner Kindheit an gelähmt war und als Angekok (Zauberer) in

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hohem Ansehen stand — der Utchickchalli-Eskimo tätowieren sich auch an Armen und Schenkeln, wie Amundsen (l. c. p. 288 u. 289) abbildet, aber auch an den Augen, Wangen und Halse, indem ein mit Tran getränkter und mit Lampenruß geschwärzter Faden mittels einer Nadel in regelmäßigen Abständen durch die Haut gezogen wird. Der abgestreifte Ruß bleibt in der Haut zurück und heilt in dieselbe ein (cf. Otto Stoll, Geschlechtsleben, Leipzig, Veit S. 75 und Abbild. S. 79): der Aberglaube, daß die Schädel der Weiber, die nicht so tätowiert sind, im Himmel zu Trangeschirren würden (Paul Egede), soll die Ursache dieser Tätowierung sein.

Die Behandlung der Haare bei den Eskimos geschieht bloß aus Bequemlichkeit: Kurzschneiden bei den Männern, Zusammendrehen und Aufbinden bei den Frauen — wie erwähnt mit Hilfe des Urins, um sie glänzender und geschmeidiger zu machen. — Von Krankheiten, welche die Eskimos dem Eindringen der Kultur wahrscheinlich zu verdanken haben, ist in erster Linie die Tuberkulose zu nennen, deren rapides Fortschreiten in Grönland von Nansen (l. c. p. 290) teils auf die schlechtere Kleidung, teils darauf geschoben wird, daß sie jetzt das ganze Jahr hindurch in ihren feuchten, ungesunden Häusern, wo die Ansteckungskeime den vorzüglichsten Nährboden finden, leben müssen. Endlich, daß sie europäische Kost haben, während früher ihre fette Kost und besonders der Speck sie vorzüglich widerstandsfähig gegen diese Krankheit machte. Weiter haben die Pocken einen großen Teil der Bevölkerung hingerafft. Merkwürdig aber ist, daß die Grönländer zum großen Teil freigeblieben sind von Syphilis. Sie ist dort oben, sagt Nansen (l. c. p. 291), nur an einer Stelle zu finden, nämlich in Arsuk in Südgrönland, wo man die Krankheit zu isolieren sucht. Trotzdem hat sie um sich gegriffen und es ist leider Aussicht vorhanden, daß sie sich ausbreiten und die ganze Bevölkerung auch auf diese Weise verseuchen wird.“ — Die Sonnenwendfeier, das Kelandispiel, schildert Amundsen (l. c. p. 278—281) als einen langweiligen Tanz der Erwachsenen und der Kinder, mit Gesang und Trommelbegleitung, wobei es ihm unbegreiflich erschienen sei, worin das Vergnügen bei diesem Tanz bestehe. Bei jeder Arbeit wird gesungen, aber eine einförmige Melodie.

In bezug auf den Tod der Frau sagt Dalager (Nansen, l. c. p. 113): Einer Frau, die mit dem Tode ringt und sich keines besondern Ansehens erfreut, kann es wohl passieren, daß sie lebendig begraben wird, wovon wir am Orte vor kurzem ein Beispiel hatten, das recht jämmerlich war, indem mehrere erzählten, daß sie die Begrabene noch lange Zeit im Grabe nach einem Trunk hätten rufen hören. Der eigentliche Grund hierfür soll bei den Eskimos in der

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übermäßigen Furcht derselben vor der Berührung mit einem Toten liegen. Infolgedessen ziehen sie dem Sterbenden, ganz gleich, ob Mann oder Frau, oft schon lange vor dem Eintreten des Todes die Leichenkleider an und treffen alle Vorbereitungen zum Hinausbringen und Begraben der Leiche, während der Kranke ihnen von seinem Lager aus zusieht. Die Toten werden sofort nach ihrem Abscheiden hinausgebracht und zwar, wenn sie in einem Hause gestorben sind, durch das Fenster, wenn in einem Zelt, durch eine eigens für diesen Zweck in die Hinterwand gemachte Öffnung. Auf der Ostküste wird jedoch nach Holm die Leiche auch mittelst eines ihr um die Beine gebundenen Riemens von Seehundsleder aus dem Hausgang hinausgeschleift (Nansen, l. c. p. 217).

Die Toten werden entweder begraben oder ins Meer geworfen. Den Männern werden ihre Habe wie Kajak, Waffen und Anzüge, den Frauen ihre Nähutensilien, Krummesser auf oder neben das Grab, beim Versenken in das Meer an den Strand gelegt.

Wer den Toten hinausgetragen hat oder ihn oder etwas ihm Zugehöriges berührt hat, ist für einige Zeit unrein und muß sich nach Vorschrift der Angekoker gewisser Speisen oder Arbeiten enthalten.

Der Tod flößte den Eskimos nach Amundsen (l. c. p. 208) nicht die geringste Furcht ein. Waren sie krank oder ging es ihnen schlecht, dann nahmen sie mit aller Seelenruhe Abschied vom Leben und erstickten sich. Während unsers Verkehrs mit ihnen kamen zwei solcher Fälle vor. — Die guten Menschen kommen nach ihrer Ansicht auf den Mond, die bösen in die Erde hinab.

Daß die Eskimos noch heute in der Steinzeit leben, wurde sowohl von Nansen (l. c. p. 288) wie Amundsen (l. c. p. 227) bewiesen, da sie keine andere Art des Feueranzündens kennen, wie zwei Stücke Holz aneinanderzureiben und noch Lanzen, Pfeile und Bogen aus Renntierhorn gebrauchen, außerdem zum Fangen der Fische sich nur der Spieße aus Renntierhorn bedienen. Wenn man daraus aber auf einen minderwertigen Verstand derselben schließen wolle, so täte man ihnen unrecht, da die anscheinend so primitiven Gegenstände sich den vorhandenen Bedürfnissen und Verhältnissen so gut angepaßt erwiesen, wie sie nur die Erfahrung und ein kluges Ausprobieren durch die Jahrhunderte hindurch hergestellt haben konnten.

Wenn wir zum Schluß nun uns die Frage vorlegen, welche Übereinstimmungen in dem Leben des Weibes am Äquator und am Pole zu finden sind, so müssen wir zunächst noch die genauere geographische Lage ihrer Länder angeben: so liegt die Balistation von Zintgraff etwa 7° nördlich vom Äquator und auf dem 10.° östlicher

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Länge; Kaiser Wilhelmsland, der Bismarckarchipel, Neumecklenburg und Neupommern liegen 8—10° südlich vom Äquator zwischen dem 140. und 155.° östlicher Länge, und die Gefilde, welche von den verschiedensten Stämmen der Eskimos eingenommen werden, erstrecken sich über ganz Grönland vom 60. über den 80.° nördlicher Breite und vom 30.—50.° westlicher Länge; dann in Nordamerika: Baffinland, Boothia Felixland, King William-Land, Victorialand bis nach Alaska und zur Behringstraße in einer Breite von 65—80° nördlich und zwischen dem 60.—160.° westlicher Länge. Sie liegen also 75—80° in vertikaler Richtung und 160—280° in transversaler Richtung voneinander entfernt, und während die Nordkamerunneger und die Kanaken ziemlich eng umgrenzte Völkerschaften darstellen, sind die Stämme der Eskimos außerordentlich zahlreich und haben schon in vielen der erwähnten Gegenden unter dem Eindringen der Kultur sehr gelitten, so daß an manchen Orten fast nur noch Mischlinge von Europäern und Eskimos existieren. Am unberührtesten waren wohl noch die von Amundsen besuchten und beschriebenen (l. c. p. 225—339) Netschjilli-Eskimos; während die Bewohner an der westlichen amerikanischen Küste, die Kagmalli-Eskimos, unter der Zivilisation bereits so gelitten hatten, daß sie, die mehrere hundert Familien stark gewesen, im Jahre 1905 schon auf ganz wenige zusammen geschmolzen waren.

Werfen wir nun einen kurzen Rückblick auf das bisher Mitgeteilte zum Vergleich der Lebensgewohnheiten der verschiedenen Stämme, so können wir beim Vergleich der Eskimostämme mit den Kamerunnegerinnen und Kanakinnen vier Gruppen unterscheiden:

In der ersten Gruppe finden wir fast völlige Übereinstimmung in bezug auf folgende Punkte: Die Ehe zwischen Blutsverwandten ist verboten; die Geburten sind in der Regel leicht und finden in hockender, kniender oder liegender Haltung statt. Die Kinder werden lang und sehr lang gestillt. Mißgestaltete werden getötet. Die Säuglinge werden auf dem Rücken oder der Hüfte der Mutter getragen. Die Fruchtbarkeit ist nicht beträchtlich, sie schwankt zwischen zwei und vier Kindern. Tätowierungen kommen fast ausnahmslos bei Frauen vor. Das Anbieten der Frauen ist allen gemeinsam.

Sehr ähnlich ist das Verhalten in bezug auf die Ehe. Zwar zeigt dieselbe bei den Kamerunnegern den monogamischen Charakter, doch ist dem Manne der Negerin der Verkehr mit Sklavinnen gestattet zu der Zeit, in der sich seine Frau der Kohabitation enthält, z. B. während der Regel, der Schwangerschaft und Säugungsperiode. Bei den Kanaken und Eskimos gilt Vielweiberei, jedoch meist nur mit zwei

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Frauen. — Durch den Eintritt der Menses — der bei den Kamerunnegern festlich begangen wird — wird das Mädchen „unrein" und von der Familie getrennt. In bezug auf die Beschäftigung der Frauen ist dieselbe, was häusliche Arbeit, Pflege und Erziehung der Kinder, Herstellung von Gerätschaften und Unterstützung des Mannes in mancherlei Arbeiten betrifft, ebenfalls sehr ähnlich.

Zur dritten Gruppe, die uns zeigt, daß das Verhalten der Stämme wenigstens ähnlich ist, rechnen wir ihr Verhalten zum Weibe und die Stellung, die sie dem Weibe einräumen.

Die niedrigste Stellung nimmt offenbar die Kanakin ein; sie kann vom Mann verschenkt, gekauft, getötet werden, indessen ist sie materiell von ihm unabhängig, da das, was sie mit in die Ehe gebracht und in ihr miterworben hat, ihr auch bleibt. Auch die Negerin steht physisch und sozial tiefer als ihr Mann, doch ist sie ein wertvolles Eigentum desselben und die Matronen haben oft eine hohe Vertrauensstellung. Von der Eskimofrau aber sagt Nansen, daß sie eine bedeutende Rolle in der Gesellschaft spiele, da sie in der Regel gut behandelt werde, und in der Tat: bei den zahlreichen Abbildungen von Ehepaaren, die uns Amundsen geliefert hat, kann man überall fröhliche Frauengesichter und mit den Kindern zusammen auch ein wirkliches Familienglück bewundern — so gering sind ihre Ansprüche an das Leben — so groß ist ihre Genügsamkeit.

Die wichtigsten Unterschiede zwischen den genannten Völkerstämmen existieren endlich in Beziehung auf Krankheiten, von denen dieselben befallen werden, und hier sind die Eskimos entschieden in der ungünstigsten Lage, insofern sie durch die immermehr fortschreitende Zivilisation, durch die Abnahme ihrer Haupternährungsquelle (der Seehunde), durch den zunehmenden Branntweingenuß, durch die Verschlechterung ihrer Wohnungs- und Kleidungsverhältnisse und ihre um sich greifende Verarmung immer mehr die Beute von verheerenden Krankheiten, wie Tuberkulose, Skrofulose, Rhachitis, Pocken und Syphilis werden. Die weit günstigere Lage, in der sich in dieser Beziehung die Neger und Kanaken befinden, haben wir früher schon besprochen. Obwohl auch für sie der Alkohol ein furchtbares Gift ist, welches immer mehr verbreitet wird, so ist doch die Menge von Momenten, welche die Eskimostämme fortwährend dezimieren, eine so große, daß sowohl Nansen als Amundsen auf die näherrückende Gefahr einer vollständigen Vernichtung derselben in ergreifender Weise aufmerksam gemacht haben. Hoffen wir, daß deren Warnungsrufe nicht ungehört verhallen!

München, 31. Januar 1908.

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Aus der experimentell-biologischen Abteilung des Pathologischen Institutes zu Berlin. Vorstand: Professor Dr. Ad. Bickel.

482/84.

(Innere Medizin Nr. 144/46.)

Experimentelles und Klinisches zur Kenntnis der Beeinflussung der Magensaftsekretion durch Medikamente.

Von

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Die genauere Kenntnis der Bedeutung der Magensaftsekretion für die Diagnose, die Pathologie und die Therapie der Magenkrankheiten ist eine verhältnismäßig noch junge Errungenschaft der Medizin. Bedeutend älter sind diesbezügliche Studien der Physiologie, während die Pathologie bis vor wenigen Jahrzehnten die Erkrankungen des Magens nur mit geringer Liebe erforscht hat. Dieses Gebiet der inneren Medizin wurde stiefmütterlich behandelt, bis es Forschern wie Kußmaul, Ewald, Riegel u. a. gelang, die bisherigen diesbezüglichen Kenntnisse gründlich umzuarbeiten und fruchtbar zu erweitern. An diese Namen knüpft sich eine neue, moderne Periode in der Pathologie und Therapie der Verdauungsorgane, vor allem charakterisiert durch eine präzise Diagnose gegenüber den unklaren Begriffen der älteren Epoche vor diesen Forschern. Mag es beispielsweise angeführt werden, daß man bei Durchsicht der älteren Literatur in den 40er Jahren bei der Klassifizierung der Magenaffektionen kaum zwischen anderen Krankheitsbildern unterscheidet, als zwischen dem höchst unklaren und fast von jedem Autor anders definierten Begriffe der „Dyspepsie", wobei nicht einmal der Unterschied zwischen anatomisch gegebener Grundlage und funktioneller Art gemacht wird, ferner zwischen Ulcus und Carcinoma ventriculi, der Magenerweiterung und der seit Jahrzehnten klinisch nicht mehr bekannten Gastromalacie, besonders der Kinder, welche heutzutage nicht mehr als ein Krankheitsbild sui

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generis, sondern als eine postmortale Veränderung aufgefaßt wird. — In den 50er Jahren unterscheidet man noch in der Literatur zwischen „Magenentzündung" und „krankhafter Verdauung", wie wenn diese Bezeichnungen wirklich Gegensätze wären. So unklar eben waren die darunter verstandenen Begriffe. Kaum klarer sind diese im folgenden Jahrzehnt, in den 60er Jahren, wo man mit dem Worte „Gastritis" einzig das bezeichnet, was die neuere Medizin phlegmonöse Gastritis nennt, während man für „Gastritis" im modernen Sinne noch immer den unklaren, verwirrenden Begriff „Dyspepsie" braucht.

Einen Fortschritt in der weiteren Aufhellung dieser dunklen Begriffe bilden die Arbeiten zweier Engländer, welche in pathologisch-anatomischer Hinsicht einige Krankheitsbilder des Magens, besonders die entzündlichen Veränderungen der Schleimhaut, die Gastritis gut beschreiben: so die mikroskopischen Studien von Handfield Jones[1]) über die Gastritis atrophicans, den wir damit als Entdecker dieser Affektion, wenigstens im anatomischen Sinne, bezeichnen können, und die umfassendere unter Virchows Leitung durchgeführte sehr gute Abhandlung von Fox[2]) über die Mikroskopie verschiedener Magenaffektionen.

Die neue Ära auf diesem Gebiet beginnt mit der Einführung der Magenpumpe durch Kußmaul[3]) im Jahre 1869, welcher diese amerikanische Erfindung in erster Linie nicht zu diagnostischen, sondern therapeutischen Zwecken praktisch anwandte, und zunächst diese Methode auf die Behandlung der chronischen Magenerweiterung in Form von Spülungen mit alkalischen Mineralwässern und Medikamenten (Sodalösungen und Karbolsäurelösungen) beschränkte.

Einen erheblichen Schritt weiter, sowohl in der Diagnose zunächst, wie nach und nach auch in der Therapie hat uns Ewald[4]) gebracht durch die Einführung seiner ebenso einfachen wie genialen Untersuchungsmethode, des Probefrühstückes. Diese Methode hat vor allem über die Sekretionsverhältnisse des Magens Klarheit verschafft, und es ist dabei ein nicht geringes Verdienst Ewalds, die Gastritis als ein polymorphes Krankheitsbild erkannt und beschrieben zu haben, bei dessen einzelnen Differenzierungen gerade die Art der Sekretion ausschlaggebend ist. Damit wurden die alten verworrenen Begriffe

1) Assoc. med. Journ. Oct. 1853.
2) Lancet Juli 1853.
3) Deutsches Arch. f. klin. Med. 1869, Bd. 6.
4) Berl. klin. Wochenschr. 1888, Nr. 36.

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der Gastritis und Dyspepsie des weitern voneinander getrennt, nachdem Leube[1]) 1878 die Dyspepsie als eine Erkrankung, resp. Teilerkrankung ohne anatomische Grundlage charakterisiert und damit von der Gastritis als einer Krankheitsform mit anatomischem Substrate von primär oder sekundär lokalem Charakter scharf geschieden hatte. — Hand in Hand mit der Abgrenzung der Gastritis von der Dyspepsie geht auch die weitere Trennung anderer anatomisch gegebener Erkrankungen von den rein funktionellen „nervösen" Störungen, und damit erfolgt auch die wissenschaftlich präzisere Feststellung des Begriffes einer Magenneurose. Die Magenneurosen werden nun, und diese Einteilung besteht noch immer zu Recht, in sensible, motorische und sekretorische unterschieden. Diese Einteilung ist nicht pathologischer, sondern in erster Linie physiologischer Natur, und so bilden diese drei Verhältnisse, in denen sich die Hauptfunktionen der Verdauungsorgane abspielen, die Basis für die Diagnose und die Therapie auch bei den Krankheiten der Verdauungsorgane, speziell des Magens mit anatomischer Grundlage. Was diese letztere anbelangt, lassen sich die anatomischen Veränderungen am kranken Magen nach folgenden großen schematischen Grundzügen unterscheiden, welche in gewissem Maße auch bestimmten klinischen Bildern entsprechen: In der Muskularis unterscheidet man hypertrophische und atrophische Prozesse, Entzündungen (Gastritis phlegmonosa), ferner Substanzverluste (Ulkus) und Neoplasmen (Sarkome); in der Mukosa spielen entzündliche Vorgänge die Hauptrolle, die teilweise zu analogen Prozessen führen (Hypertrophie und Atrophie der Schleimhaut, Gastritis hypertrophica und atrophica), ferner sind vorwiegend hier Substanzverluste (Erosionen und Ulkus), sowie Neoplasmen (Karzinom) lokalisiert. Hand in Hand mit diesen anatomischen Prozessen, häufig und teilweise als direkte Folgen dieser lokalen Vorgänge, gehen Funktionsstörungen der Drüsensekretion vor sich, sei es, daß der Drüsenapparat anatomisch von der Schleimhauterkrankung mit ergriffen wird, sei es, daß er in bloß funktioneller Art nervöse Impulse erhält. Dies betrifft sowohl die Schleim- als auch die Magensaftdrüsen.

Das Studium dieser Drüsenfunktion unter physiologischen und besonders pathologischen Verhältnissen soll nun die Aufgabe dieser Arbeit sein, in dem Sinne, daß im folgenden dargelegt werden soll, wie die Magensaftsekretion durch Medikamente in engerem und weiterem Sinne des Wortes (z. B. Mineralwässer) beeinflußt werden kann, und unter welchen Umständen eine solche Beeinflussung vom klinisch-therapeutischen Standpunkte aus indiziert ist.

1) Deutsches Arch. f. klin. Med. 1873, Bd. 23.

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Im Interesse einer einheitlichen Darstellung soll hierbei in erster Linie die Literatur seit Anfang der 40er Jahre bis auf die neueste Zeit eingehend berücksichtigt werden. Zur Aufklärung einiger strittiger Punkte, sowie zur Kenntnis einiger bisher nicht untersuchten Fragen sollen des weiteren einige tierexperimentelle Untersuchungen einen Beitrag liefern. Außerdem sollen Beobachtungen aus der eigenen Praxis versuchen, einige theoretisch-experimentelle Fragen mit der praktisch-klinischen Therapie in Einklang zu bringen.

Neben der Motilität spielt die Sekretion, d. h. der Chemismus des Magens eine große Rolle, welche in der Physiologie besonders eingehend gewürdigt worden ist. Freilich verkennt auch die Pathologie die Wichtigkeit dieser Rolle nicht, soweit es die Diätetik betrifft. Haben doch besonders die Forschungen des letzten Jahrzehntes, welche durch Pawlows bahnbrechende Untersuchungen einen mächtigen Impuls erhielten, nachgewiesen, daß der Verdauungsapparat unter physiologischen und pathologischen Bedingungen auf die einzelnen Bestandteile der Nahrung (Eiweiß, Fett, Kohlenhydrate, Wasser, Salze und Genußmittel) in geradezu spezifischer Weise reagiert, und aus diesen Beobachtungen sind wichtige Schlußfolgerungen für die diätetische Therapie gezogen worden. Nun aber ist das Stiefkind der modernen Therapie, die Pharmakologie, auch hier etwas stiefmütterlich behandelt worden. Allerdings treffen wir in der Literatur schon seit manchen Jahrzehnten klinisch-experimentelle Beobachtungen über die Pharmakodynamik einzelner chemischer Verbindungen und Medikamente auf den Verdauungsapparat an, und die letzten Jahre haben uns verschiedene sehr interessante Arbeiten tierexperimenteller Art geliefert, aber trotzdem waren diese Beobachtungen bisher zu keinen strikten systematisch aufgebauten Direktiven für die medikamentöse Therapie der Magendarmerkrankungen verwertet worden. Dieses habe ich in meinem Buche: „Grundriß der medikamentösen Therapie der Magen- und Darmkrankheiten“ (Wiesbaden, Verlag J. F. Bergmann, II. A. 1906) zu tun versucht und möchte hierfür auch in dieser vorliegenden kleineren Arbeit einen weiteren Beitrag liefern.

Die Erkrankungen, bei denen die Art der Sekretion eine große Rolle spielt, betreffen in erster Linie die Schleimhaut des Magens in Form einer diffusen Entzündung und einer Sekretionsanomalie. Diese bestehen bei den einzelnen Krankheitsbildern entweder für sich allein (Beispiele: Gastritis chronica simplex, ohne Sekretionsstörungen, Hyperchlorhydria nervosa ohne Entzündung der Mukosa), oder sie sind in den häufigeren Fällen miteinander verbunden (Beispiel: Gastritis chronica subacida, Gastritis hyperacida, Entzündung und Sekretionsstörung, ferner Ulkus mit Hyperazidität und sekundärem

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Katarrh). Die Kombination von Entzündung mit Sekretionsstörung ist das Häufigere. Bei einzelnen Formen ist die Entzündung das Primäre und die Sekretionsanomalie ein Folgezustand hiervon (Beispiel: Gastritis chronica simplex, welche nach und nach in eine subazide Form übergeht), in anderen Fällen liegen die Verhältnisse umgekehrt vor (Beispiel: Nervöse Hyperchlorhydrie, die in eine Gastritis acida übergeht). Für die Therapie ist nun die Tatsache von großer Bedeutung, daß der enge Zusammenhang zwischen Entzündung und Sekretionsanomalie sich auch in der Einwirkung der Pharmakodynamik der einzelnen Körper, Nahrung und besonders Medikamente auf den Magen geltend macht, d. h. diejenigen Mittel, welche die Entzündnug bekämpfen, bekämpfen zugleich auch die Sekretionsstörung und umgekehrt, diejenigen, welche die Sekretionsanomalie in ungünstigem Sinne beeinflussen, verschlimmern zu gleicher Zeit auch den Entzündungsvorgang.

Diese Reaktion des kranken Magens auf die betreffenden chemischen Körper vereinfacht die Therapie. Freilich gibt es auch hier, wie auf allen Gebieten der Medizin Ausnahmen, aber im großen und ganzen besteht diese Regel zu Recht, wie in folgenden Ausführungen klargelegt werden soll.

Die Beeinflussung der Sekretion durch die einzelnen Arten chemischer Körper resp. Medikamente erfolgt auf keine einheitliche Art und Weise. Die betreffenden Substanzen setzen den Hebel ihrer Wirkung an verschiedenen Orten an, das Produkt oder die manifeste Äußerung dieser Wirkung offenbart sich dann vornehmlich in der Art der Magendrüsenfunktion, d. h. der Magensaftabscheidung. Nach pharmakodynamischen Prinzipien kann man folgende Arten von die Magensekretion beeinflussenden Körpern unterscheiden:

I. Körper, welche vom Zentralnervensystem aus die Sekretion beeinflussen. Die Reaktion der Magendrüsen ist hier keine spezifische, sondern eine bloße Teilerscheinung, eine Mitbeteiligung an der Reaktion verschiedener Organe auf die betreffende Substanz.

II. Körper, welche durch primäre Erregung anderartiger nervöser Impulse (z. B. Geschmacksnerven) sekundär die Magendrüsenfunktion beeinflussen.

III. Körper, die ihre Dynamik lokal im Magen ansetzen, aber direkt auf die Magendrüsen einwirken.

IV. Körper, die auf dem Blutwege auf die Magensaftabscheidung einwirken (rektale Anwendung succogener und pepsinogener Substanzen).

Diese Einteilung ist vom physiologisch-pharmakodynamischem Standpunkte aus wohl interessant, vom Gesichtspunkte der Therapie

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aus aber empfiehlt sich eine mehr praktische Einteilung nach der chemischen Beschaffenheit der Substanzen. Bei der Besprechung der betreffenden Körper soll natürlich obige Einteilung auch berücksichtigt werden.

So wollen wir folgende Hauptgruppen von die Magensaftabscheidung beeinflussenden Körpern unterscheiden:

I. Das Wasser, die Kohlensäure, die Alkalien und Typen von Mineralwässern.

II. Gewisse Adstringentien.

III. Gewisse Metalle.

IV. Gewisse Nervengifte der Alkaloidreihe (nach Schmiedeberg).

V. Die Stomachika (im engeren Sinne des Wortes).

Wenn nun nach diesem Plane die Beeinflussung der Magensaftsekretion in pharmakodynamischem und therapeutischen Sinne untersucht werden soll, so muß man sich zunächst vergegenwärtigen, daß eine genaue experimentelle Erforschung dieser Verhältnisse erst durch die von Pawlow eingeführte und von seinen Schülern weiter ausgebeutete Methode möglich geworden ist. Auch meine Untersuchungen, soweit sie tierexperimenteller Natur sind, wurden nach dieser Methode an der experimentell-biologischen Abteilung des Pathologischen Institutes zu Berlin angestellt, indem ich hierfür Hunde benutzte, welche zum Teil von Herrn Prof. Dr. Bickel, zum Teil von mir selbst nach Pawlow operiert worden waren. Es sei mir gestattet, an dieser Stelle auf die Art und das Prinzip der Technik dieser Operation kurz einzugehen.

Die Pawlowsche Operation hat zunächst eine Analogie in der viel älteren Methode von Thiry, der schon im Jahre 1864 am Dünndarm von Hunden einen nach außen mündenden Blindsack herstellte. Aus einer Darmschlinge schnitt Thiry ein zylindrisches Stück Darm heraus, bildete aus diesem einen Blindsack, der nach innen (nach dem Darme zu) abgeschlossen war und nach außen offen in die Bauchwunde eingenäht wurde. So konnte reiner, nicht mit Darminhalt vermischter Darmsaft gewonnen werden. Diesen Gedanken verwertete später Heidenhain, indem er einen solchen Blindsack aus dem Fundus des Magens bildete. Dieser Blindsack ergoß so sein Sekret nach außen und die Drüsenarbeit in diesem war in gewissem Grade der Abklatsch in verkleinerten Maßstabe von der Drüsenarbeit des großen Magens. Dieses Spiegelbild war aber ein ungenaues, ein verzerrtes, weil Heidenhain durch die Art der Schnittführung bei seiner Operation die Vaguszweige zum großen Teil durchschnitten hatte. Pawlow ging nun bei der Verbesserung dieser Methode[1]) von der Erkenntnis aus, daß die Drüsenfunktion des kleinen Magens nur dann mit derjenigen des großen Magens parallel läuft und qualitativ wie quantitativ koinzidiert, wenn die am Magen längs verlaufenden

1) Vgl. Pawlow, Die Arbeit der Verdauungsdrüsen. Verlag von J. F. Bergmann, Wiesbaden 1898.

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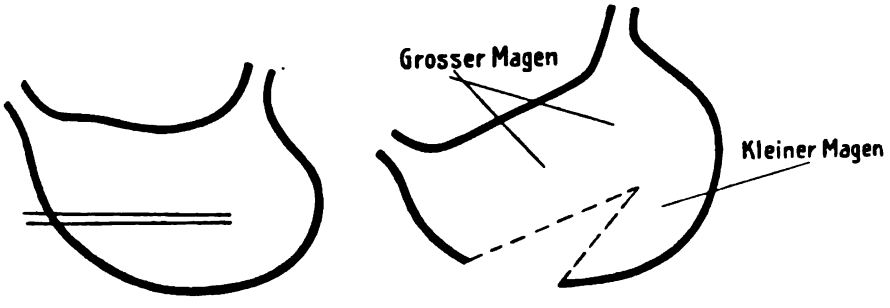
Vagusverzweigungen bei der Operation intakt gelassen werden. Nach diesem Prinzip wird auch an der experimentell-biologischen Abteilung des Pathologischen Institutes in Berlin operiert. Hierzu werden nur ausgewachsene, große, kräftige und gesunde Hunde verwendet. Vor der Operation bekommt das nüchterne Tier eine Morphiumdosis von 0,05 subkutan, einerseits damit es eventuelle Nahrungsreste noch tüchtig ausbricht, andererseits zur Erleichterung der Narkose. Auf dem Operationstische aufgespannt, wird das Tier am Abdomen rasiert, mit Schmierseife gewaschen und mit Sublimatlösung und Alkohol desinfiziert. Äthernarkose. Die Vorbereitungen des Operateurs und des Materials zur Operation entsprechen genau den Grundsätzen der Aseptik wie bei Operationen am Menschen. Mittels eines ca. 15 cm langen medianen Längschnittes wird die Bauchhöhle eröffnet und der Magen wird in toto hervorgezogen. In der Längsachse des Organes, im Fundusteile an der großen Kurvatur, wird dieses nun da, wo man den Blindsack abtrennen will, mit zwei einander parallel verlaufenden, ca. 2 cm voneinander getrennten Klemmzangen (Darmklemmen) abgeklemmt.

Nach Unterbindung der auf der Serosa verlaufenden Zweige der Arteria coron. ventr. wird nun zwischen den Klemmen die vordere und hintere Magenwand getrennt. Der Schnitt an der großen Kurvatur, etwa 2 cm von Pylorus entfernt, wird 10—12 cm fortgesetzt, immerhin so, daß am obersten Teile des Fundus noch eine intakte Brücke von 5—6 cm Breite besteht. Dadurch wird der Magen in einen großen und einen kleinen Teil geschieden, aus denen der große und der kleine Magen gebildet werden.

Abnahme der Klemmen, Unterbindung der blutenden kleineren Gefäße. Sodann wird von den Schnittflächen der beiden getrennten Magenteile aus die Mukosa abpräpariert, wobei Muskularis und Serosa nicht verletzt werden sollen. Am großen Magen erfolgt die Abtrennung der Schleimhaut in einer Breite von $1^1/_2$—2 cm, am



kleinen Magen hingegen von $2^1/_2$—3 cm. Die beiden Magenteile sollen nun an ihren Grundflächen verschlossen werden, und zwar zuerst der große Magen. Hierzu werden zunächst die abpräparierten Schleimhautflächen der vorderen und hinteren Magenwand durch Tabakbeutelnaht miteinander nach innen vernäht; dann erfolgt die Naht der Muskularis und der Serosa. Ähnlich ist der Vorgang am kleinen Magen; nur handelt es sich hier in erster Linie darum, diesen zu einem wirklichen Blindsacke zu gestalten, in der Art und Weise, daß keine offene Kommunikation zwischen der intakt gebliebenen Brücke und dem kleinen Magen bestehen darf. Deshalb wird aus seiner abpräparierten Schleimhaut eine Kuppel gebildet, deren Konvexität nach der Brücke gerichtet ist. Darauf wird auch der kleine Magen geschlossen, zunächst durch Tabakbeutelnaht die Schleimhaut, resp. die Ränder der

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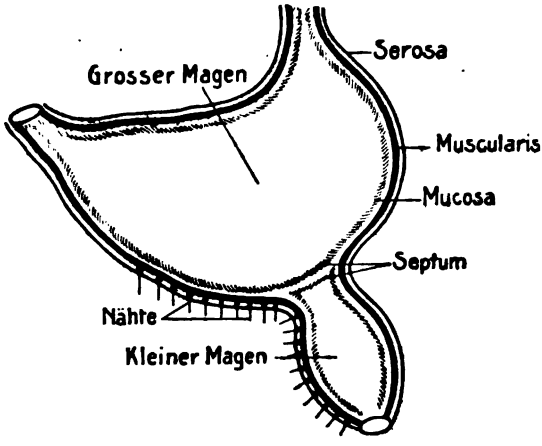
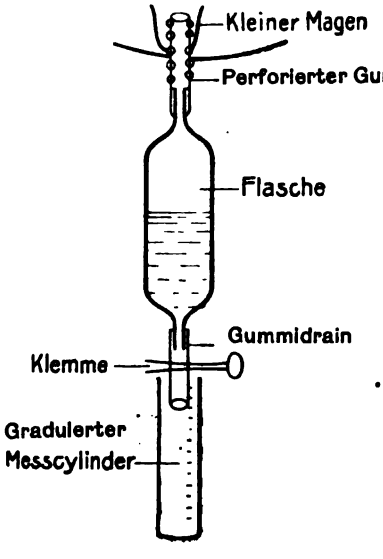
Kuppel, dann erfolgt die Naht der Muskulatur und der Serosa. Großer und kleiner Magen sind so durch ein zweifaches Septum getrennt: einerseits durch die an dieser Stelle intakte Schleimhautkuppel des kleinen Magens, andererseits durch die in der Mitte vernähten eigenen Mukosa (des großen Magens an der Brücke).



Der kleine Magen stellt so ein zylinderförmiges distal zugespitztes Gebilde vor, dessen Öffnung nun in die Bauchwunde eingenäht wird. Letztere wird bis auf die Ausmündung des Blindsackes durch drei Etagennähte geschlossen und mit Jodoformgaze und Jodoformkollodium bedeckt. — Ein solch operiertes Tier ist bei günstigem Verlaufe nach etwa einer Woche, höchsten 10 Tagen experimentierfähig, wiewohl in den meisten Fällen trotz aller Vorsichtsmaßregeln die Heilung der Bauchwunde, d. h. ihrer Muskulatur und Haut per primam nur ausnahmsweise erreichbar ist.

Zur Vornahme der unten beschriebenen Experimente wurden nur Tiere verwendet, welche sich von der Operation gut erholt hatten, munter waren, mit gutem Appetit fraßen und keine Temperaturerhöhungen aufwiesen. Auch wurden die Tiere erst dann benutzt, wenn das aus dem Blindsack aufgefangene Sekret von Wundprodukten (Blut, Eiter) möglichst rein war. Die Hauptbedingung zur Versuchsanordnung ist nun ein vollständiges Nüchternsein der Tiere, d. h. die auf die Nahrung erfolgende Sekretion mußte gänzlich abgelaufen sein. Die Tiere wurden also nüchtern gestellt. Bei Fütterung ausschließlich mit Pferdefleisch war dies schon nach 10 bis 12 Stunden der Fall, auf eine gemischte auch viel Kohlehydrate enthaltende Kost hin mußte man bis zur völligen Versiegung der Sekretion durchschnittlich 14—16 Stunden warten.



— Das nüchterne Tier wird nun in einem Gestelle aufgestellt und am Vorder- und Hinterkörper zur möglichsten Ausschaltung der Ermüdung mit Tüchern leicht suspendiert. Es soll dabei möglichst im psychischen Gleichgewichte bleiben, da seine Intelligenz nach Pawlows klassischen Versuchen auf Störungen auch mit besonderen Sekretionsvorgängen im Magen reagieren kann. Vor allem darf das Tier keine Nahrung sehen oder riechen, denn sofort würde die damit assoziierte Vor-

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stellung des Fressens, die Freßlust „psychischen Appetitsaft" auslösen und damit den Gang der Versuche erheblich verändern. Im umgekehrten Sinne würde Ärger wirken (z. B. Vorhalten einer Katze nach Bickels Versuch).

In die Öffnung des Blindsackes wird nun ein Gummidrain eingeführt, welches mit der sog. „Pincussohnschen Magenflasche" verbunden wird. Der Apparat wird mittels einer um den Leib des Hundes geschlungenen Binde festgehalten.

Aus dieser Flasche wird von Zeit zu Zeit der Magensaft entnommen und untersucht, wie weiter unten näher beschrieben werden soll.

Betrachten wir nun die Beeinflußbarkeit der Magensekretion in physiologischer und therapeutischer Hinsicht durch die Körper der oben aufgestellten Gruppe und zwar zunächst durch:

I. Das Wasser, die Kohlensäure, Alkalien und Typen von Mineralwässern.

Das Quellwasser, wie das chemisch reine destillierte Wasser ist für die Magensaftsekretion keine ganz indifferente chemische Verbindung. Daß es eine geringe Sekretion im Magen hervorruft, werden wir in den folgenden Versuchen öfters sehen.

Eine erheblich größere Anregung erfährt die Drüsensekretion des Magens durch die im Wasser suspendierte Kohlensäure. Vom klinischen Standpunkte aus wird diese schon im Jahre 1884 von Jaworski[1]) empfohlen, indem dieser Autor ihr einerseits eine Steigerung der darniederliegenden Sekretion bei subaziden Formen von Magenaffektionen nachrühmt, andererseits ihr auch eine tonisierende Wirkung auf die Atonie der Muskeln zuschreibt. Durch Anregung der Magensaftabscheidung soll sie auch den Appetit befördern. Diese Auffassungen bestätigt Pentzoldt.[2]) Die Salzsäuresekretion des Magens beginnt nach seinen Beobachtungen früher unter der Einwirkung der Kohlensäure und erreicht höhere Grade, auch die Motilität des Magens wird gesteigert, indem die Aufenthaltsdauer der Speisen im Magen je nach deren Beschaffenheit um $^1/_2$—$^3/_4$ Stunden abgekürzt wird. Weidert[3]) bestätigte neulich diese klinischen Beobachtungen. In neuester Zeit hat Pincussohn[4]) den sekretionssteigernden Einfluß der CO_2 auf die Magensaftabscheidung an Pawlowschen Hunden experimentell nachgewiesen, indem er in der Mehrzahl der Fälle eine quantitative Steigerung und eine qualitative Erhöhung (erhöhte Konzentration) des Sekretes fand. Schmiede-

1) Berl. klin. Wochenschr. 1884, Nr. 33.
2) Deutsches Arch. f. klin. Med. 1902, Bd. 73.
3) Inauguraldissertation Erlangen 1903.
4) Arbeiten aus dem Path. Inst. zu Berlin 1906.

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berg[1]) verlegt den Angriffspunkt dieses chemischen Körpers auf die Schleimhaut bzw. die Drüsen des Magens selbst, indem er sich darüber folgendermaßen äußert:

„Die Erklärung für die heilsamen Wirkungen dieser gasförmigen Säure ist darin zu suchen, daß sie auch bei Gegenwart von Alkalien wirksam bleibt. Sie durchdringt die Magenwandung von allen Seiten und wird dann nicht wie andere Säuren in den Geweben vollständig neutralisiert, sondern ist hier bei genügender Menge gleichzeitig als Bikarbonat und im absorbierten Zustande erhalten. In dieser Weise vermag die CO_2 die Funktion der Gewebe anzuregen, ohne die wesentlichen Eigenschaften der Alkalien aufzuheben. Dazu kommt als weiteres günstiges Moment, daß die Erregung stets eine mäßige bleibt und daher niemals durch ein Übermaß schaden kann."

Diese pharmakologischen Wirkungen der CO_2 indizieren ihre Anwendung sowohl bei akuten, wie auch bei gewissen subaziden Formen von chronischen Gastritiden und anderen mit Sekretionsverminderung einhergehenden Magenerkrankungen. Auch die Wirkung mancher Mineralwässer beruht teilweise auf der Anwesenheit von CO_2 (siehe unten).

Von nicht geringerer, eher noch von größerer therapeutischer Bedeutung für die Sekretionsbeeinflussung der Tätigkeit der Magendrüsen sind die Alkalien. Zur Gruppe der Alkalien in engerm Sinne rechnet Schmiedeberg pharmakologisch „alle Verbindungen der Alkali- und Erdmetalle, welche basische Eigenschaften (alkalische Reaktion) besitzen und keine giftig wirkenden Komponenten enthalten". Therapeutisch kämen die Salze der Alkali-, der Erdalkalimetalle und der Magnesiumgruppe in Betracht, in praktisch-klinischer Hinsicht und pharmakodynamisch erforscht sind nur einzelne Verbindungen bestimmter Vertreter dieser Gruppen.

Unter den Salzen der Alkalimetalle, überhaupt unter allen hier in Anwendung kommenden Alkalien spielt therapeutisch die wichtigste Rolle das Kochsalz, das Natriumchlorid; in erster Linie deshalb, weil es neben der CO_2 in den Kochsalzquellen den einzigen pharmakodynamisch wirkenden Bestandteil bildet, da andere chemische Körper nur in minimaler Quantität vorhanden sind. Bei den Physiologen und Klinikern war die Frage der Wirkung von NaCl auf die Magensaftsekretion eine langumstrittene. In klinisch-experimenteller Hinsicht standen sich die Stimmen maßgebender Forscher einander entgegengesetzt gegenüber: Beispielsweise schrieb Frerichs[2]) dem NaCl eine günstige Einwirkung auf die Magensaftabscheidung und auf den Peptonisierungsvorgang im Magen zu, während Reichmann[3]) eine gegenteilige Wirkung des Kochsalzes annahm. Diese

1) Grundriß der Pharmakologie. 1906.

2) Vgl. Boas, Diagnostik und Therapie der Magenkrankheiten. 1907.

3) Arch. f. exp. Pathologie und Pharmakologie 1887, Bd. 24.

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soll nicht in einer Steigerung, sondern in einer Hemmung der Drüsentätigkeit beruhen und zwar nicht allein bei starken Konzentrationen (5—10%), sondern auch bei schwachen Lösungen (1—2%). Die Abnahme der Azidität erklärt Reichmann einerseits durch Annahme einer den Mageninhalt neutralisierenden Transsudation aus den Blutgefäßen, andererseits durch vermehrte Schleimabsonderung. — Löwenthal[1]) weist hingegen auf die klinischen Erfolge hin, die er durch Berieselung des Magens mit warmer physiologischer NaCl-Lösung erzielt. Die verminderte Sekretion konnte er hierdurch bedeutend erhöhen.

Das entscheidende Wort in dieser Kontroverse hat zunächst seit Jahrzehnten die Empirie gesprochen, welche durch die gewiegten Stimmen reichlicher Erfahrung von Ewald, Riegel, Boas u. a. geäußert wurde, daß der mehrwöchige Gebrauch gewisser Kochsalzquellen subazide Zustände der Magensekretion erheblich bessern, ja sogar zur Heilung, d. h. zur normalen Azidität führen könne. Solche Beobachtungen kann auch der Praktiker vielfach machen, und auch ich[2]) habe darauf hingewiesen, daß der zu lange fortgesetzte Gebrauch einer Kochsalzquelle eine Gastritis subacida in eine hyperacida überführen kann.

Warum steht in diesem Falle die Empirie in einem Gegensatze zu den Resultaten der Untersuchung mancher, gewiß auch recht zuverlässiger Beobachter, wie z. B. Reichmanns? Aus verschiedenen Gründen: Erstens ist die Wirkung der NaCl-Lösungen und -Quellen auf den kranken menschlichen Magen keine rasche, sondern stellt sich erst nach längerer Anwendung (Kuren von mehreren, 3—4 Wochen) ein; zweitens ist diese Wirkung an eine bestimmte, besonders nach oben abgegrenzte Konzentration an Kochsalz gebunden; drittens ist nicht nur nach klinischen Erfahrungen, sondern auch nach Analogie meiner eigenen, allerdings mit andern Medikamenten durchgeführten, unten erwähnten Untersuchungen anzunehmen, daß die entzündlich veränderte Magenschleimhaut wohl auch auf das NaCl wenigstens graduell anders reagiert als die anatomisch intakte Mukosa.

Die empirischen Tatsachen haben im Laufe der letzten Jahre eine einwandsfreie Bestätigung ihrer Richtigkeit in biologischen Experimenten gefunden, welche am Pathologischen Institut zu Berlin an nach Pawlow operierten Hunden angestellt wurden. So prüfte zunächst Bickel[3]) die Kochsalzquellen, speziell den Wiesbadener Kochbrunnen, und kam zu folgenden Resultaten:

1) Berl. klin. Wochenschr. 1893, Nr. 47 ff.
2) Berl. klin. Wochenschr. 1906, Nr. 23.
3) Berl. klin. Wochenschr. 1906, Nr. 2.

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„Die Kohlensäure und das Kochsalz der Kochsalzwässer begünstigen die sekretorische Arbeit der Magenschleimhaut. Bei einem Vergleich des Einflusses der Kochsalzwässer mit demjenigen gewöhnlichen Leitungswassers findet man, daß große Unterschiede hinsichtlich der abgeschiedenen Sekretmenge jedenfalls nicht bestehen, daß man aus den Sekretionskurven höchstens die Tendenz herauslesen kann, es werde durch die Kochsalzwässer die Saftproduktion befördert. Bei einem Hunde mit chronischem subazidem Katarrh des großen und des kleinen Magens erfuhr unter dem Eindruck der Gabe eines Kochsalzwassers der Säuregehalt des vom kleinen Magen abgeschiedenen Sekretes eine deutliche Steigerung."

Einen Schritt weiter ging Baumstark[1]), der ebenfalls zuerst am Tierexperimente nachwies, daß die Intensität der Saftsteigerung bei den verschiedenen Kochsalzquellen variiert, indem bei den Homburger Quellen die Fähigkeit der Saftvermehrung als bedeutend größer festgestellt wurde als die des Wiesbadener Kochbrunnens. An der Hand vieler einwandfreier Versuche nimmt Baumstark für die Homburger Kochsalzwässer in Anspruch:

„daß sie nicht wie die von Bickel und seinen Mitarbeitern untersuchten Kochsalzwässer höchstens die Tendenz einer Saftvermehrung erkennen lassen, sondern daß sie die Saftsekretion der Magenschleimhaut beim Tier und dem erwachsenen Menschen in außerordentlich starkem Maße, um durchschnittlich 74,1 % (gegenüber der Wirkung gewöhnlichen Wassers) erhöhen."

Von größter Wichtigkeit für die Therapie ist nun der Umstand, daß dieser Autor durch einen glücklichen Zufall die Frage der Übertragbarkeit dieser Resultate vom Tierexperiment auf den Menschen lösen konnte. Baumstark konnte nämlich an einem von Prof. Gluck wegen vollständiger Ösophagusstenose operierten 23jährigen Mädchen mit gesunder Magenschleimhaut experimentieren. Der Patientin war vor 8 Jahren eine Gastrostomie und vor einem Jahre zu therapeutischen Zwecken eine Ösophagotomie angelegt. Der zentrale Ösophagusteil wurde vernäht und versenkt, der periphere zu einer Ösophagusfistel ausgebildet. Zur Erhaltung des Kauaktes, der Einspeichelung des Bissens und zur Ermöglichung der Speichelverdauung konnten Ösophagus- und Magenfistel durch einen Schlauch miteinander verbunden werden. An dieser Patientin experimentierte nun Baumstark mit den verschiedenen Kochsalzquellen von Homburg und kam zu dem obenerwähnten, das Tierexperiment völlig bestätigenden Resultate. Besonders bemerkenswert dabei ist der Umstand, daß beim Menschenexperiment „die Verdauung der quantitativen Magensaftabscheidung dem Kochsalzgehalt der verschiedenen Brunnen proportional ist. Das Optimum liegt beim Landgrafbrunn mit 1,4% NaCl, dann folgt die Elisabethquelle mit etwa 1% Kochsalzgehalt und erst zuletzt der Ludwigsbrunnen mit nur

1) Arch. f. Verdauungskrankheiten 1906, Bd. 12, Heft 3.

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0,7%." Diese Beobachtungen sprechen gegen die bisher übliche empirische Annahme eines Optimums bei ungefähr 1% und einer Abnahme der Wirkung, wenn ein Wasser mehr als etwa 1% Kochsalz enthält.

Des weitern ist für die Praxis an dieser Arbeit der Umstand wichtig, daß Baumstark am Tierexperiment nachweisen konnte, daß die sekretionssteigernde Wirkung der NaCl-Wässer nur dann eintritt, wenn das Wasser erstens nüchtern gegeben, und zweitens die Nahrung erst dann aufgenommen wird, wenn das Mineralwasser den Magen vollständig verlassen hat, d. h. die Verdauungsperiode muß von der Mineralwasserverabreichung durch eine genügend lange Pause getrennt sein. Kochsalzbeimengung zur Nahrung hemmt nämlich die Saftabscheidung in hohem Grade; an Tierexperimenten war die Gesamtsaftmenge der NaCl-Wasserperiode um 43 bzw. 58,8% herabgesetzt gegenüber der Gesamtsaftmenge der Wasserperiode (150 ccm mit 150 ccm Milch). Diese Hemmung der Sekretion durch NaCl-Lösungen, bei oder nach der Mahlzeit aufgenommen, bestätigt auch Frl. Rozenblat[1]) an ähnlichen Versuchen, nach welchen die Intensität der Saftverminderung der Konzentration an Kochsalz proportional ist, je konzentrierter die Lösung, desto intensiver wird die Abscheidung des Magensafts gehemmt. Auch die Sekretionssteigerung vor der Mahlzeit eingenommen wird hier bestätigt, am besten ist die Wirkung, wenn das Wasser eine Stunde vor der Verdauungsperiode genommen wird.

Mit den gleichen Resultaten studierten Rheinboldt die Wirkung des Kissinger Rakoczy-Wassers, Heinsheimer[2]) die der Baden-Badener Hauptstollenquelle und Sasaki[3]) die des Ostseewassers.

Damit ist durch diese übereinstimmenden Ergebnisse die sekretionssteigernde Wirkung des NaCl und der Kochsalzwässer endgültig erwiesen, aber damit noch nicht die Art und Weise, wie diese Wirkung zustande kommt: ihr Mechanismus. Schmiedeberg sucht diesen, soweit es sich um eine Erklärung der günstigen Wirkung bei gewissen Magenerkrankungen (subaziden Gastritiden) handelt, in einer Art nutritiver Reizung des Gewebes der Schleimhaut und damit auch der Drüsen. So äußert er sich folgendermaßen:

„Bei chronischen Erkrankungen des Magens ist der kurgemäße Gebrauch der Kochsalzquellen in vielen Fällen vorteilhaft. Die Besonderheit der Salzwirkung gegenüber anderen Reizmitteln ist darin zu suchen, daß die Salzlösung nicht bloß die Oberfläche bespült, sondern gleichsam in breitem Strome tief in die Schichten

1) Inauguraldissertation Berlin 1907.
2) Arch. f. Verdauungskrankheiten 1906, Bd. 12, Heft 2.
3) Ebenda, Heft 3.

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der Magenschleimhaut eindringt und die Ernährungszustände derselben infolge der konstanten und ein gewisses Maß nicht überschreitenden nutritiven Reizung in günstiger Weise verändert."

Diese Auffassung gewährt uns aber noch keine genauere Einsicht in den Mechanismus der Salzwirkung auf die Drüsenfunktion. Dies sucht Wohlgemuth[1]) an Tierexperimenten zu erreichen, aber trotz seines interessanten Endresultates muß die Frage, ob hierbei eine direkte Einwirkung auf die Magen- resp. Drüsennerven stattfindet, oder ob die Blutintervention zu Hilfe genommen werden muß, offen gelassen werden. Wohlgemuth hat nachgewiesen, daß das in den Magen eines chlorarmen Hundes (mehrtägige Fütterung mit ausschließlich ausgekochtem Pferdefleisch) eingeführte NaCl einen solchen Reiz auf die Magenschleimhaut bzw. Magennerven auszuüben vermag, daß der Magen, der bis dahin (im Chlorhunger) nur äußerst spärliche Mengen von Saft produzierte, fast mit einem Schlage einen reichen Saftstrom von annähernd normaler Konzentration entwickelt. Ob es sich hier um eine direkte Einwirkung auf die Magenschleimhaut und Magennerven handelt, oder ob diese vom Blute aus erfolgt, das dabei eine Anreicherung durch Kochsalz erfahren hat, läßt der Autor dahingestellt. Daß in der Tat in das Blut eingeführtes Kochsalz die Sekretion der Magendrüsen erhöht, haben Versuche von Braun, Grützner und Boas übereinstimmend ergeben.

Bei der Frage nach der Pharmakodynamik der Mineralwässer spielt in neuester Zeit die Radioaktivität eine gewisse Rolle. So stellten Bergell und Bickel[2]) am natriumhaltigen Wiesbadener Kochbrunnen eine Erhöhung der fermentativen (Pepsin-) Wirkung des Magensaftes durch Messung der peptischen Eiweißverdauung nach Mette fest, während emanationsfreies Wiesbadener Wasser auch die Pepsinwirkung, ähnlich wie die Salzsäureabscheidung während der Verdauungsperiode eingenommen, hemmt, und Rheinboldt[3]) wies an der Kissinger Rakoczyquelle, sofern diese frische Radiumemanation enthält, oder nach deren Verlust (nach 48 Stunden) dieselbe wieder auf künstlichem Wege nach dem System Bergell[4]) zugeführt bekam, eine bakterizide Wirkung (auf den Bacillus prodigiosus) nach. Die ersten beiden Autoren streifen nur beiläufig die Frage, ob die Radioaktivität auch die Menge der Salzsäureabscheidung beeinflusse, und kommen dabei zur Auffassung einer indifferenten Einwirkung des Radiums. Dies veranlaßte mich die Einwirkung radioaktiven Salzes

1) Arb. aus dem Patholog. Institut zu Berlin 1905.
2) Kongr. Wiesbaden 1905 und Zeitschr. f. klin. Med. 1905, Bd. 58, Heft 3.
3) Berl. klin. Wochenschr. 1906, Nr. 20.
4) Arb. aus dem Pharmakol. Institut Berlin 1906.

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auf die Saftabscheidung am Tierexperimente nachzuprüfen. Dazu benutzte ich sog. Emanosalkapseln nach den Angaben von Reitz durch die Höchster Farbwerke dargestellt. Die Radiumemanation ist dann an 0,5 g NaCl gebunden. Die Radioaktivität beträgt 20000 Volt pro Stunde, d. h. löst man eine solche Kapsel in einer beliebigen Quantität Wasser auf, etwa in 200 g, und prüft man den Einfluß dieser Lösung auf die Leitungsfähigkeit eines abgeschlossenen Luftraumes, so erleidet ein in diesen Raum hineingebrachtes Elektroskop pro Stunde einen Spannungsabfall von 20000 Volt, gemessen am Sengler-Sievekingschen Apparat (Mitteilung vom Erfinder Dr. Hans Reitz, Berlin-Schmargendorf). Die Versuchsanordnung war folgende: Zwei nach Pawlow operierte Hunde wurden nüchtern gestellt. Zunächst wurde ihnen je 200 ccm Leitungswasser mit Zusatz von 0,5 bzw. 1,0 g NaCl entsprechend einer resp. zwei Emanosalkapseln durch die Schlundsonde eingegossen. Darauf wurde die aus dem kleinen Magen in der Zeiteinheit von einer halben Stunde sezernierte Saftmenge, sowie das während der ganzen Sekretionsdauer abgeschiedene Saftquantum gemessen und untersucht. Diese Werte dienen als Vergleich zu der analogen Versuchsanordnung, bei welcher an Stelle des Emanosal das entsprechende Quantum gewöhnlichen Kochsalzes gegeben wurde.

Versuch Nr. 1 am Hund „Karo".

200 ccm destilliertes Wasser + 0,5 g NaCl = 1/4 % 9 h 30		200 ccm destilliertes Wasser + 0,5 g NaCl als Emanosal = 1/4 % 11 h 45	
Zeit	Menge	Zeit	Menge
10 h —	5,5	12 h 15	3,8
10 h 30	2,8	12 h 45	3,0
11 h —	0,9	1 h 15	1,6
11 h 30	0,6	1 h 45	2,0
11 h 45	0,0	2 h —	0,4
		2 h 15	0,0
1 3/4 Stunden Sekretionsd.	9,8 ccm Gesamtquant.	2 Stunden Sekretionsd.	10,8 ccm Gesamtquant.

Aus diesem und den folgenden drei Versuchen ergibt sich eindeutig, daß die Radiumemanation auf die Sekretionssteigerung, d. h. auf die Absonderung der Salzsäure keinen besonderen Einfluß hat. Die Radioaktivität verhält sich also gegenüber der Magensaftabscheidung indifferent und hat für die Verdauung nach Bickel und Bergell nur in dem Sinne eine positive Bedeutung, daß sie die Aktivität des eiweißverdauenden Fermentes, des Pepsins, fördert. Da aber nach den gleichen

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Versuch Nr. 2 am Hund „Max".

200 ccm destilliertes Wasser + 0,5 g NaCl = 1/4 % 9 h 30		200 ccm destilliertes Wasser + 0,5 g NaCl als Emanosal = 1/4 % 11 h 45	
Zeit	Menge	Zeit	Menge
10 h —	0,1	11 h 45	0,1
10 h 30	0,2	12 h 15	0,2
11 h —	0,1	12 h 30	0,0
11 h 15	0,0		
1 1/4 Stunden Sekretionsd.	0,4 ccm Gesamtquant.	3/4 Stunden Sekretionsd.	0,3 ccm Gesamtquant.

Versuch Nr. 3 am Hund „Karo".

200 ccm destilliertes Wasser + 1 g NaCl = 0,5 % 2 h 15		200 ccm destilliertes Wasser + 1 g NaCl als Emanosal = 0,5 % 4 h 15	
Zeit	Menge	Zeit	Menge
2 h 45	2,5	4 h 45	4,0
3 h 15	1,8	5 h 15	2,2
3 h 45	1,6	5 h 30	0,6
4 h —	0,6	5 h 45	0,2
4 h 15	0,1	6 h —	0,0
1 1/2 Stunden Sekretionsd.	6,6 ccm Gesamtquant.	1 1/4 Stunden Sekretionsd.	7,0 ccm Gesamtquant.

Versuch Nr. 4 am Hund „Max".

200 ccm destilliertes Wasser + 1 g NaCl = 0,5 % 12 h 30		200 ccm destilliertes Wasser + 1 g NaCl als Emanosal = 0,5 % 2 h 15	
Zeit	Menge	Zeit	Menge
1 h —	0,2	2 h 45	0,3
1 h 30	0,3	3 h 15	0,8
2h —	0,0	3 h 45	0,2
		4 h —	0,0
1 Stunde Sekretionsd.	0,5 ccm Gesamtquant.	1 1/4 Stunden Sekretionsd.	1,3 ccm Gesamtquant.

Untersuchungen jedes Mineralwasser seine Radiumemanation nach höchstens 48 Stunden verliert, ist exportiertes Mineralwasser nicht mehr radioaktiv. Die Radioaktivität ist eine Eigenschaft nur des an der Quelle getrunkenen Wassers und darin dürfte auch ein Faktor

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liegen, warum Trinkkuren am betreffenden Orte besser wirken als zu Hause durchgeführt.

Wenn wir unter den Alkalien weiter Umschau nach Verbindungen halten, welche einen unzweifelhaft steigernden Einfluß auf die Salzsäureabscheidung im Magen haben, so treffen wir nach neuesten Untersuchungen zunächst auf gewisse Kalkverbindungen, Kalziumsalze. Birk[1]) hat das dem NaCl analoge Kalziumchlorid $CaCl_2$ am Menschen geprüft und ist zu dem Resultate gelangt, daß es die Verdauung des Ewaldschen Probefrühstückes um ca. 15 Minuten abkürzt dadurch, daß es in Dosen von schon 1,0 g eine vermehrte Magensaftabscheidung hervorruft. Damit ließe sich die klinische Indikation aufstellen, bei Subaziditätszuständen das $CaCl_2$ ähnlich wie das NaCl zu geben, wenn diese Verbindung nicht eine korrosive Wirkung auf die Magenschleimhaut hätte und eine ungünstige Wirkung auf das Herz ausüben würde, wodurch ihre klinische Anwendung hinfällig wird.

Eine analoge sekretionssteigernde Wirkung wird nach Mayedas[2]) Tierexperimenten auch dem Kalziumhydroxyd $Ca(OH)_2$, d. h. dem mit diesem Salze gesättigten Wasser, Kalkwasser (Lösungsverhältnis 1 : 600) zugeschrieben und zwar soll die Sekretionssteigerung ungefähr in dem Maße erfolgen, wie diejenige der weiter unter erwähnten Lithiumsalze. Aus der folgenden chemischen Gleichung:

$$Ca(OH)_2 + H_2O + 2HCl = CaCl_2 + 3H_2O$$

ist meiner Ansicht nach die sekretionssteigernde Wirkung analog den Untersuchungen von Birk auf das durch Einwirkung der HCl entstehende Kalziumchlorid $CaCl_2$ zurückzuführen, so daß dem unzersetzten $Ca(OH)_2$ ein spezifischer Sekretionseinfluß nicht zugeschrieben werden kann. Überhaupt ist bei der Frage der Pharmakodynamik der Medikamente nicht das Medikament als solches als von Einfluß auf die Magensekretion anzusehen, sondern die durch Einwirkung der Magensalzsäure auftretende Spaltung der Substanz und eventuell die Neubildung eines weiteren chemischen Körpers, wie uns auch die folgenden eigenen Versuche näher Aufschluß geben werden.

Von Heinsheimer[3]) wurde unter den Kalksalzen das Kalziumkarbonat $CaCO_3$ einer eingehenden tierexperimentellen Prüfung unterzogen und zwar in 5prozentigen Lösungen. Das Resultat war ein analoges zur Wirkung des $CaCl_2$ und des $Ca(OH)_2$. Statt der bisher angenommenen sekretionshemmenden und säurebindenden

1) Inauguraldissertation Erlangen 1904.
2) Biochem. Zeitschr. 1907, Bd. 2, Heft 4—6.
3) Med. Klinik 1906, Nr. 24.

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Wirkung veranlaßt das $CaCO_3$ eine stürmische Steigerung der Saftabsonderung, eine lange Zeit hindurch anhaltende Sekretion enormer Saftmengen. Gleichzeitig steigt auch die Gesamtazidität erheblich. Wie kann nun dieses im Wasser unlösliche Salz einen solchen Effekt ausüben? Heinsheimer erklärt es folgendermaßen: Größere Mengen des $CaCO_3$ verharren im Magen längere Zeit und unterliegen hier der Einwirkung der freien Salzsäure, welche Kohlensäure frei macht. Diese nunmehr schußweise immer wieder frei werdende CO_2 ist es, die als mächtiges Reizmittel auf die Drüsensekretion einwirkt. Meiner Auffassung nach ist diese Erklärung nicht einwandfrei, aus dem Grunde, daß, wie wir unten sehen werden, die beim Na_2CO_3 und $NaOHCO_3$ ebenfalls stürmisch freiwerdende CO_2 hier keine Sekretionssteigerung auszulösen vermag. Aus diesem negativen Analogon möchte ich die sekretionssteigernde Wirkung in einem anderen Körper als in der Kohlensäure suchen, nämlich wiederum in dem sich im Magen unter Einfluß der HCl bildenden $CaCl_2$:

$$CaCO_3 + H_2O + 2\,HCl = CO_2 + CaCl_2 + 2\,H_2O.$$

Diese Untersuchungen erweisen die bisher üblich gewesene Anschauung, daß die Kalksalze, speziell das Kalkwasser und der kohlensaure Kalk bei der Hyperazidität resp. Hyperchlorhydrie therapeutisch indiziert seien, als nicht mehr haltbar.

Die Reihe der sekretionssteigernden Alkalien ist in neuester Zeit durch Lithiumsalze erweitert worden. Mayeda[1]) hat durch das biologische Experiment am Lithiumhydroxyd und am Lithiumkarbonat eine den entsprechenden Kalkverbindungen analoge Wirkung auf die Magendrüsen nachgewiesen.

Beim Li_2CO_3 tritt schon bei einer Konzentration von 0,25 % eine deutliche HCl-Steigerung auf, die in stärkerer Lösung von 0,35 und 0,5 % entsprechend intensiver wird. Beim LiOH tritt diese Anregung der Sekretion schon bei einer Konzentration von 0,16 % auf, welche an Wirkung derjenigen vom Li_2CO_3 in 0,25 prozentiger Lösung ungefähr gleichkommen soll. Auch bei diesen beiden Verbindungen liegt die Vermutung nahe, daß die sekretorische Wirkung auf das im Magen durch die HCl-Einwirkung entstehende Chlorid, das LiCl, das Analogon zum $CaCl_2$ in letzter Instanz zurückzuführen ist.

Durch eigene biologische Experimente kann ich die Reihe der sekretionsfördernden Medikamente um ein weiteres erweitern, über dessen Wirkung bisher die Ansichten der Autoren entgegengesetzt lauten. Es ist das Natrium citricum, speziell das neutrale zitro-

1) Bioch. Zeitschr. 1907, Bd. 2, Heft 4—6.

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nensaure Natrium. Boas[1]) rechnet diesen Körper pharmakologisch unter diejenigen Verbindungen, welche gleich einzelnen Salzen der Alkali- und Erdalkalireihe (s. u. Natriumkarbonat, Natriumbikarbonat, Magnesia usta, Magnesia ammoniophosphorica, Magnesia sulfurica) die Sekretion herabsetzen, und empfiehlt so das Natrium citricum allein oder in Verbindung mit den erwähnten Alkalien und Erdalkalien zur Bekämpfung der Sekretionssteigerungszustände. — Nun aber kommt Lachény[2]) bei seinen Versuchen mit Natrium citricum zur Annahme, daß dieses unabhängig von der dabei angewendeten Diät nicht nur ein gutes Mittel gegen Schmerzen bei Gastritis, Ulkus und nervösen Hyperästhesien sei, daß es ferner die Motilität anrege, sondern, daß es auch auf die Magensaftsekretion steigernd wirke, indem bei seiner Anwendung die Menge der Chloride und der freien Salzsäure zunehme. Diese der Boasschen Ansicht widersprechende Annahme veranlaßte mich, das zitronensaure Natrium am Tierexperimente zu prüfen. An zwei Pawlowschen Hunden wurden je zwei Beobachtungen mit einer 3prozentigen und je zwei mit einer 10prozentigen Lösung von neutralem Natrium citricum angestellt. Der Verlauf der Sekretionsperioden bei Leitungswasser ohne und mit Zusatz von zitronensaurem Natrium war folgender:

Versuch Nr. 5 am Hund „Karo".

200 ccm Leitungswasser 9 h 30		200 ccm Leitungswasser + 6 g Natrium citricum = 3 % 10 h —	
Zeit	Menge	Zeit	Menge
9 h —	2,4	10 h 30	0,2
9 h 30	1,1	11 h —	3,0
9 h 45	0,4	11 h 30	1,6
10 h —	0,0	12 h —	3,0
		12 h 30	1,2
		9 h —	0,0
1 Stunde Sekretionsd.	3,9 ccm Gesamtquant. Gesamtazidität 116	$2^1/_2$ Stunden Sekretionsdauer	9,0 ccm Gesamtquant. Gesamtazidität 124

1) Diagnostik und Therapie der Magenkrankheiten. 5. Aufl., 1907.
2) Thèse de Paris 1906.

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Versuch Nr. 6 am Hund „Moor".

200 ccm Leitungswasser		200 ccm Leitungswasser + 6 g Natrium citricum = 3 %	
8 h 30		10 h —	
Zeit	Menge	Zeit	Menge
9 h —	1,0	10 h 30	4,0
9 h 30	0,8	11 h —	3,2
9 h 45	0,6	11 h 30	2,1
10 h —	0,0	12 h —	3,0
		12 h 30	1,2
		12 h 45	0,2
		1 h —	0,0
1 Stunde Sekretionsdauer	2,4 ccm Gesamtquant. Gesamtazidität 148	$2^1/_2$ Stunden Sekretionsdauer	13,7 ccm Gesamtquant. Gesamtazidität 146

Versuch Nr. 7 am Hund „Karo".

200 ccm Leitungswasser		200 ccm Leitungswasser + 20 gr Natrium citricum = 10 %	
9 h 30		1 h 10	
Zeit	Menge	Zeit	Menge
9 h —	2,4	1 h 40	0,5
9 h 30	1,1	2 h 10	4,0
9 h 45	0,4	2 h 40	3,2
10 h —	0,0	3 h 10	0,6
		3 h 40	0,5
		4 h 10	0,3
		4 h 25	0,2
		4 h 55	0,0
1 Stunde Sekretionsdauer	3,9 ccm Gesamtquant. Gesamtazidität 116	$3^1/_4$ Stunden Sekretionsdauer	9,3 ccm Gesamtquant. Gesamtazidität 126

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Versuch Nr. 8 am Hund „Moor".

200 ccm Leitungswasser		200 ccm Leitungswasser + 20 g Natrium citricum = 10 %	
8 h 30		1 h 10	
Zeit	Menge	Zeit	Menge
9 h —	1,0	1 h 40	3,7
9 h 30	0,8	2 h 10	3,0
9 h 45	0,6	2 h 40	2,2
10 h —	0,0	3 h 10	2,0
		3 h 40	1,5
		4 h 10	0,5
		4 h 25	0,3
		4 h 40	0,0
1 Stunde Sekretionsdauer	2,4 ccm Gesamtquant. Gesamtazidität 138	3 Stunden Sekretionsdauer	13,2 ccm Gesamtquant. Gesamtazidität 144

Diese Versuche ergeben übereinstimmend folgende Tatsachen:

1. Das Natrium citricum steigert in allen Versuchen sowohl die Sekretionsdauer, als auch das Sekretionsquantum, erstere um etwa das $2^1/_2$—3fache, letztere um etwa das 3 bis 5fache gegenüber der Leitungswasserperiode.

2. Dabei wird auch die Tendenz beobachtet, die Qualität des Sekretes zu erhöhen, resp. das Sekret prozentual konzentrierter zu machen. Die diesbezüglichen Werte der Durchschnittsgesamtazidität sind aber so gering ausgesprochen, daß sie sich innerhalb der physiologischen Schwankungen bewegen und deshalb eine positive Deutung nicht zulassen.

3. Der Wechsel in der Konzentration der Lösungen (3 oder 10 %) ist auf den Sekretionsverlauf in den verschiedenen Phasen, in der Gesamtmenge der Durchschnittsazidität und der Zeitdauer der Sekretion ohne Einfluß, d. h. es finden sich ungefähr die gleichen Werte bei der schwächeren und bei der stärkeren Konzentration.

Damit ist erwiesen, daß die von Boas vertretene Qualifikation des Natrium citricum als eines Antazidums nicht haltbar ist, daß wir im Gegenteil in diesem Präparate ein sekretionssteigerndes Mittel von ausgesprochener Wirkung besitzen.

Der chemische Vorgang im Magen ist einfacher:

$$\text{Na citr.} + \text{HCl} = \text{NaCl} + \text{Ac. citr.}$$

Die Sekretionssteigerung ist hier auf beide Produkte in der Gleichung zurückzuführen, sowohl auf das NaCl wie auch auf das Ac. citr.

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Gehen wir unter den Alkalien und Erdalkalien zu denjenigen Körpern über, welche auf die Drüsensekretion des Magens hemmend wirken, bzw. die Magensaftabscheidung verringern. Die erste Stelle nimmt nach den neuesten Untersuchungen hier das Natrium bicarbonicum ein. Seine Wirkung war bis vor kurzem eine vielumstrittene. Ältere Autoren, wie du Mesnil, Linossier, Lemoine, Schüle u. a., nahmen an, „daß kleine Gaben die Salzsäuresekretion anregen, größere hemmen, schließlich aber wieder den Reiz zu erneuter Sekretion abgeben“[1]). So betont besonder Linossier[2]), daß man kleinen Gaben von Natr. bic. eine exzitierende, d. h. sekretionsbefördernde Wirkung zuschreiben müsse, während größere Dosen umgekehrt wirken sollen. Schwartzkopff[3]) hat an 24 Selbstversuchen konstatiert, daß das doppelkohlensaure Natron in Gaben von 1—3 g nach dem Probefrühstück eingenommen zwar die prozentuale Azidität herabsetze, während dabei die absolute Säuremenge eine bedeutende Steigerung erfahre. Zum ersten Male einigermaßen Klarheit in diese einander widersprechenden Auffassungen brachten die ausgedehnten Versuchsreihen Reichmanns[4]), welcher dem Natr. bic. wenigstens jede Säuresteigerung abspricht. Er kommt zu dem Resultate, daß sowohl schwache wie starke Lösungen, mögen sie dem nüchternen Magen zugeführt, oder kurze Zeit nach der Mahlzeit gegeben werden, nie eine Steigerung der Sekretion zur Folge hätten. Nach dem Essen genommen verringert Natr. bic. selbstverständlich durch Neutralisation resp. Bindung der freien HCl die qualitative Azidität, steigert sie aber niemals quantitativ. In diesem Zustande erblicken Reichmann und mit ihm auch Boas u. a. ein ziemlich indifferentes symptomatisches Mittel zur momentanen Linderung der Übersäuerung des Magens. Debove[5]) schreibt dem Natr. bic. eine ausgesprochene sekretionshemmende und säuretilgende Wirkung zu, indem er es bei Ulcus ventriculi in Dosen von 2,0 vor und 4,0 nach dem Essen gibt, in der Absicht, dadurch die schädliche Einwirkung des Magensaftes auf die Geschwüre zu vermeiden.

Der Frage einer ausgesprochenen pharmakodynamischen Wirkung des Natr. bic. auf die Funktion der Magendrüsen ist man, nachdem in der Literatur diese Kontroversen jahrelang in Ruhe gelassen wurden, erst in den letzten Jahren näher getreten und zwar ist es das Verdienst Bickels und seiner Schüler, am biologischen Experimente, an

1) Nach Boas zitiert.
2) Bull. thérap. 1896, p. 155 ss.
3) Inauguraldissertation Würzburg 1892.
4) Ther. Mitt. März 1895.
5) Gaz. hébd. 1884, no. 18.

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Pawlowschen Hunden zunächst, sodann auch am Menschen (obenerwähnte Patientin von Gluck) endlich über die vielumstrittene Frage einer relativen Indifferenz, d. h. bloß momentan einer säurebindenden oder einer -steigernden oder einer hemmenden Wirkung, Klarheit verschafft zu haben. So wies unter Bickels Leitung Heinsheimer[1]) nach, daß das Natr. bic. nur eine Sekretionshemmung, niemals eine Steigerung zur Folge hat, und zwar eine leichte Verminderung der Saftmenge und der Azidität schon bei schwachen Lösungen von 1 %, eine sehr starke und längere Zeit andauernde Herabsetzung bei höherer Konzentration, 5 %, und zwar betrifft diese Herabsetzung auch hier nicht nur die Quantität, sondern auch die Qualität (Konzentration) der HCl. Zu dem gleichen Resultate kommt auch die Arbeit von Frl. Rozenblat[2]), nach welcher die hemmende Kraft des Natr. bic. derjenigen des Natrium carbonicum, der Soda, ungefähr gleichkommen soll: Eine $^1/_2$prozentige Lösung beider Salze soll imstande sein, selbstverständlich nüchtern gegeben, die Sekretion gegenüber derjenigen von Leitungswasser bis um 30 % herabsetzen, während 4prozentige Lösungen eine Reduktion bis zu 70 % bedingen können.

Wie schon erwähnt ist die Wirkung des Natriumkarbonates derjenigen des -bikarbonates im Prinzipe gleich. Über dieses Salz liegen interessante klinische Beobachtungen von Mathieu und Laboulais[3]) vor, welche mit den neuesten tierexperimentellen Forschungen ganz im Einklang stehen. Nach den klinischen Versuchen dieser Autoren hatten zwar Gaben von 0,5—1 % Na_2CO_3, eine halbe Stunde vor dem Ewaldschen Probefrühstück genossen, keinen deutlichen Einfluß auf die Sekretion, wohl aber Dosen von 3 g. Diese Konzentration setzte den Prozentgehalt, sowie die Menge der gebundenen und der freien HCl erheblich herab, besonders 1 Stunde vor dem Probefrühstück gegeben. Dieser sekretionshemmende Einfluß war nicht nur von momentaner, sondern auch längerer Dauer und hatte kurativen Charakter bei einem an Hyperchlorhydrie leidenden jungen Manne, bei dem die Sekretion nach 14tägigem Einnehmen von täglich 4 g, d. h. von je 1 g Natr. carbon. und 2 g bei der Mahlzeit qualitativ und quantitativ erheblich herabgesetzt werden konnte. — Experimentell wurde die Sekretionshemmung durch Soda schon von Pawlow[4]) beobachtet. Er äußert sich darüber:

„Keine einzige der angewandten Sodalösungen von 0,05—1 % vermochte, wie sie in der Menge von 150 ccm in den großen Magen eingebracht wurde, auch nur

1) Med. Klinik 1906, Nr. 24.
2) Inauguraldissertation Berlin 1907.
3) Gaz. des hôp. 1894.
4) Arb. der Verdauungsdrüsen, S. 124.

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einen Tropfen Saft aus dem kleinen Magen zu treiben; es floß höchstens Schleim heraus. Somit hatte die Gegenwart der Soda im Wasser die safttreibende Wirkung des letzteren herabgesetzt. Diese Tatsachen verdienen eine große Beachtung sowohl wegen ihres klinischen Interesses, als auch besonders aus physiologischen Gründen."

Bickel[1]) hat diese Angaben Pawlows vollauf bestätigt und erweitert. Auf Sodalösungen von 0,5—1 % bleibt die Bildung eines sauren Saftes aus, und aus dem kleinen Magen fließt höchstens etwas Schleim heraus. Auch unter pathologischen Verhältnissen hat Bickel diese hemmende Wirkung festgestellt. So versiegt die durch Pilokarpininjektion hervorgerufene mächtige Sekretionssteigerung schon wenige Minuten auf die Darreichung der Sodalösung hin, und das Sekret nimmt alkalische Reaktion an. Die vermehrte Speichelabsonderung dauert indessen unverändert fort. Die Art der Wirkung dürfte wohl in einer Lähmung der Drüsenfunktionen zu suchen sein. — Die Sekretionshemmung resp. -herabsetzung durch Natr. carbon. ist auch von Heinsheimer und Rozenblat beobachtet worden (s. o.). Als Beispiel für die sekretionsherabsetzende Wirkung eines alkalischen Mineralwassers, möchte ich die experimentellen Untersuchungen von Sasaki[2]) über das Vichy-Wasser anführen. Dieses bildet einen deutlichen Gegensatz zu den alkalisch muriatischen Quellen, wie das Emser- und das Selterswasser und besonders zu den Kochsalzquellen, welche beide Arten die Sekretion deutlich steigern. — Den sekretionshemmenden Einfluß weisen deutlich auch die Bittersalze auf. So hat Birk[3]) das Analogon zum NaCl und $CaCl_2$, das Magnesiumchlorid, $MgCl_2$, einen Bestandteil der Bitterwässer, klinisch geprüft und seine sekretions- und verdauungsverzögernde Wirkung am Probefrühstück festgestellt: Dosen von 2,0—4,0 g sollen die Verdauung der letzteren um ca. 15 Minuten, Dosen von 10 g um etwa 60 Minuten verlangsamen. — Am Tierexperimente hat Heinsheimer[4]) beim Natrium sulfuricum und bei der Magnesia sulfurica schon in Konzentrationen von 3 % eine energische Herabsetzung der quantitativen HCl-Abscheidung konstatiert, während sich der Aziditätsgrad, der prozentuale HCl-Gehalt, schwankend verhielt.

Physiologisch interessant und von klinischer Bedeutung an allen den erwähnten Resultaten mit den Alkalien und den Bittersalzen ist die durchweg beobachtete, durch das Mettsche Verfahren nachgewiesene, gleichzeitige Herabsetzung auch des proteolytischen Fermentes, der Pepsinkraft. Tichomicow[5]) betont vor allem

1) Berl. Klin. Wochenschr. 1905, Nr. 128.
2) Arch. f. Verdauungskrankheiten 1906, Bd. 12, Heft 3.
3) l. c.
4) l. c.
5) Wratsch 1905, no. 2.

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den eingreifenden Einfluß der Alkalien auf das Pepsin, in dem Sinne, daß diese die aktive Form des Fermentes in die inaktive überführen sollen, wobei sich möglicherweise Salze des Fermentes bilden können.

Als Prototyp für die Verkörperung der sekretionshemmenden Wirkung der Bittersalze figuriert nach den obenerwähnten Untersuchungen Sasakis das Hunyadi-Janos-Bitterwasser, dessen graphisch dargestellte Sekretionskurve zeitlich und quantitativ erheblich unter derjenigen vom alkalischen Vichy- und besonders vom Leitungswasser verläuft.

II. Gewisse Adstringentien.

Unter dieser Gruppe sollen einige pharmakologisch wichtige Körper besprochen werden, welche teilweise schon seit vielen Jahrzehnten empirisch im Rufe einer besonderen Heilwirkung auf den kranken Magen stehen.

So eingehend die Alkalien und Bittersalze in ihrem Einflusse auf den Sekretionsverlauf im Magen untersucht worden sind, so wenig Aufmerksamkeit hat man, wenigstens in experimenteller Hinsicht, den Adstringentien zugewendet. Aber auch in der Pathologie ist die pharmakodynamisch-klinische Bedeutung dieser Körper nicht eingehend berücksichtigt worden. In bezug auf ihre Wirkung auf die anatomisch-chemische Grundlage jeder Magenerkrankung, die Entzündung und den Chemismus finden sich sowohl in den Lehrbüchern der Pathologie wie der Pharmakologie nur oberflächliche Andeutungen ohne präzise Betonung ihres differenten oder indifferenten Einflusses der betreffenden chemischen Körper.

Bei der Frage der Indikation zur Anwendung gewisser Adstringentien bei Magenerkrankungen bin ich zunächst theoretisch von der Idee ausgegangen, daß ein Adstringens, dessen Wirkung (nach Schmiedeberg) in praktischer Beziehung darin besteht, „die Intensität der Vorgänge zu vermindern, welche bei der Entzündung Platz greifen, also Schwellung und Wucherung der zelligen Gewebselemente zu mäßigen oder zu beseitigen, sowie eine übermäßige Schleimsekretion zu unterdrücken und die Exsudat- und Eiterbildung zu hemmen", diesen Einfluß mutatis mutandis auch auf die Schleimhaut des Magens ausüben und die Sekretionsvorgänge dabei nicht unbeeinflußt lassen sollte. Durch praktische Erfahrungen von der prinzipiellen Richtigkeit dieser zwar naheliegenden, aber in der Literatur bisher kaum angedeuteten Ansicht belehrt, habe ich in meinem „Grundriß der med. Therapie der Magen- und Darmkrankheiten" die Indikation aufgestellt, Erkrankungen des Magens mit Sekretionssteigerung, besonders entzündlicher

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Art, medikamentös doch lieber mit gewissen Adstringentien, als mit indifferenten Mitteln (z. B. Amaris) zu behandeln und in einer kleinen Publikation[1]) habe ich einige typische klinische Beispiele dafür angeführt. Einfach scheinen die Verhältnisse a priori da zu liegen, wo man zunächst nur mit einer entzündeten Schleimhaut des Magens zu tun hat. Warum sollten die Adstringentien hier nicht ebensogut entzündungsbekämpfend wirken, wie bei einer anderen entzündeten Mukosa? Auch noch einfach dürfte die Frage ihrer Wirkung bei einer Entzündung mit Sekretionssteigerung sein. Wenn hier die Sekretionssteigerung ein Folgezustand der Entzündung ist, so dürfte man zum voraus erwarten, daß Hand in Hand mit den Heilungsvorgängen der Entzündung auch die Sekretion geregelt würde. Anders verhält es sich mit der Frage nach der sekretortschen Wirkung einiger Adstringentien auf die nicht entzündlich veränderte Magenschleimhaut, weil bisher diesbezügliche experimentelle Untersuchungen nur spärlich angestellt wurden, und deren Verlauf noch zu keinem einheitlichen Resultate geführt hat.

An der Hand von Experimenten an Pawlowschen Hunden soll nun im folgenden versucht werden, diesen Fragen eine experimentell-biologisch begründete genauere Antwort zu geben.

Beginnen wir zunächst mit den Wismutverbindungen. — Unter diesen spielt therapeutisch von jeher eine große Rolle das Bismutum subnitricum, das Magisterium bismuti von der chemischen Formel $Bi\langle^{NO_3}_{O}$. Die alten Ärzte wandten es als ein Universalmittel gegen alle Arten von Magenkrankheiten an. Die klinische Beobachtung, daß es bei Übersäuerungszuständen des Magens besonders indiziert sei, treffen wir bei Oppolzer[2]) an, welcher dem Präparate eine bessere Wirkung als den Antacidis (kohlensaure Magnesia und kohlensaurer Kalk) zuschreibt. Hannon[3]) empfiehlt in ähnlicher Indikation das Bismutum subcarbonicum, dem er vor dem Bismutum subnitricum den Vorzug gibt. Eine Erklärung für die Wirkung des Bismutum subnitricum bei Magen- und Darmkatarrhen gibt Bricka[4]), indem er hierfür ein Freiwerden von Säure im Magen-Darmkanal annimmt, welche als leichtes Kaustikum auf die Mukosa wirkt, und die Unwirksamkeit des Bismutum carbonicum wird dadurch erklärt, daß keine kaustisch wirkende Säure frei werde. Zur Entfaltung dieses Effektes müsse aber das Präparat in reichlich großen Dosen und mit

1) Berl. klin. Wochenschr. 1906, Nr. 28.
2) Zeitschr. der k. k. Gesellsch. der Ärzte, Wien, Januar 1857.
3) Presse méd. 1856, no. 46/50.
4) Thèse Strasbourg 1864.

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einem gewissen Quantum Wasser vor der Mahlzeit gegeben werden. Ferner seien Differenzen im Säuregehalte des Mittels für dessen Wirkung nicht ohne Bedeutung, indem stark basische Präparate unwirksam seien. — Eine weitere interessante Beobachtung treffen wir bei Dunbar[1]) an, der dem Bismut in solchen Fällen eine besondere Heilwirkung zuschreibt, wo die Zunge rot und die Papillae fungiformes vergrößert seien. Dies trifft nun gewöhnlich bei Hyperazidität zu, und daraus können wir den Schluß ziehen, daß Dunbar bei Sekretionssteigerungen vom Bismut gute Erfolge sah, bzw. dieses sekretionshemmend wirkte. — Schwartzkopff[2]) schreibt bei seinen obenerwähnten klinischen Versuchen dem Bismutum subnitricum keine ausgesprochene sekretionshemmende Wirkung zu. Die Magensaftabscheidung nach Darreichung einer Wismutaufschwemmung kam meistens qualitativ und quantitativ derjenigen nach Wasseraufnahme gleich.

Die neuere Literatur beschäftigt sich fast nur mit den physikalischen Eigenschaften der Wismutsalze, d. h. mit ihrer Fähigkeit auf Substanzdefekte (Erosionen und Ulzera) der Magenschleimhaut eine schützende Decke zu bilden und so die lädierten Stellen vor der Einwirkung des sauren Magensaftes zu schützen. Daneben wird eine Sekretionshemmung nur beiläufig erwähnt, ohne darauf des näheren einzugehen. So äußert sich vom klinischen Standpunkte aus Boas[3]), nachdem er die Fleinersche Ulkusbehandlung mit großen Wismutdosen (10 bis 20 g pro die) besprochen, folgendermaßen:

„Matthes[4]) hat die günstigen Erfolge Fleiners bestätigt und führt sie auf Grund experimenteller Erfahrungen darauf zurück, daß das eingeführte Wismut eine Schleimseketion hervorruft und das Wismutschleimgemisch eine schützende Decke für etwaige Substanzverluste liefert. Fuchs[5]) konnte diese Beobachtung dahin erweitern, daß das Wismut mit Schleim vermischt zu Wismutoxyd reduziert wird. Diesen schließen sich vom klinischen Standpunkte Rosenheim, Savelieff, Crämer, Witthauer, Stintzing, Riegel im wesentlichen an."

Pentzoldt und Boas selbst verhalten sich abwartend. — Weiter sagt Boas:

„Kontraindiziert ist die Wismutbehandlung bei Magenaffektionen mit stark verminderter Salzsäureabscheidung mit Ausnahme von hämorrhagischen Erosionen und ulzerierenden Geschwülsten."

Die Auffassung des Pharmakologen über die Einwirkung des Wismutes auf den Magen vertritt Heinz[6]) wie folgt:

1) Practitioner, Sept. 1882.
2) l. c.
3) Diagnostik und Therapie der Magenkrankheiten. 1907, 5. Aufl.
4) Zentralbl. f. innere Med. 1894, Nr. 1.
5) Deutsche med. Wochenschr. 1903, Nr. 14.
6) Lehrb. der Arzneimittellehre 1907.

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„Man wendet das Bismutum subnitricum innerlich an bei rundem Magengeschwür, bei Hyperchlorhydrie, sowie bei allen Formen von Kardialgie. Die günstige Wirkung auf das Magengeschwür rührt offenbar davon her, daß das Wismutsubnitrat auf dem Geschwür einen dasselbe völlig auskleidenden Überzug bildet, der es vor mechanischen, wie chemischen Insulten durch die Ingesta schützt. Bei Kardialgie, die durch übermäßige Salzsäureproduktion seitens der sonst gesunden Schleimhaut bedingt ist (Auftreten der Schmerzen ca. $^3/_4$ Stunden nach der Mahlzeit, zur Zeit des Auftretens der freien HCl im Magen), gibt man das Wismutsubnitrat eine halbe Stunde nach dem Essen."

Schmiedeberg[1]) vertritt mehr die Ansicht einer chemischen (nicht vorwiegend physikalischen) Wirkung des Wismutes auf die Magenmukosa:

„Das basisch salpetersaure Wismut (Magisterium bismuti) ist in Wasser unlöslich und deshalb unter gewöhnlichen Verhältnissen unwirksam. Selbst in den Magen kann das völlig arsenfreie Präparat in größerem Mengen gebracht werden, ohne Schaden zu verursachen. Doch wird dabei ein kleiner Teil in der sauren Magenflüssigkeit gelöst und wirkt dann adstringierend und antiseptisch. Da die Lösung, d. h. die Umwandlung in die wirksame Verbindung durch die Verdünnung der Magensäure beschränkt ist, so kann man dieses Präparat in solchen Fällen mit Vorteil anwenden, in denen es darauf ankommt, einen gleichmäßigen gelinden Grad jener Wirkung längere Zeit, wochen- und selbst monatelang, zu unterhalten."

Experimentell wurde das Bismutum subnitricum bisher nicht eingehend und nur von einem einzigen Autor, Heinsheimer[2]), auf seine Sekretionswirkung im Magen geprüft. In seiner mehrfach zitierten Arbeit hat Heinsheimer nebenbei das Bismutum subnitricum auf dessen sekretorische Wirkung geprüft und ist zu dem Resultate gelangt, daß 5prozentige Aufschwemmungen zwar keine deutliche Verminderung der Azidität, aber eine entschiedene Herabsetzung der Saftmenge zur Folge hätten, während $2^1/_2$prozentige Aufschwemmungen einen nur geringern Einfluß auf die Sekretionsgröße aufweisen.

Durch die folgenden Versuche an Pawlowschen Hunden bezwecke ich nun die Einwirkung einiger Wismutpräparate auf die Magensekretion genauer zu prüfen. — Zunächst das Bismutum subnitricum. Unter den oben beschriebenen Vorbereitungen und Voraussetzungen nahmen die einzelnen Versuche folgenden Verlauf:

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1) l. c.
2) l. c.

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Versuch Nr. 9 am Hund „Karo".

200 ccm Leitungswasser 8 h 55		200 ccm Leitungswasser + 10 g Bismutum subnitricum = 5% 11 h —	
Zeit	Menge	Zeit	Menge
9 h 25	0,3	11 h 30	3,0
9 h 55	3,3	12 h —	1,6
10 h 22	4,0	15 h 30	0,7
10 h 55	0,0	1 h —	0,2
		1 h 30	1,2
		2 h —	0,0
$1^1/_2$ Stunden Sekretionsdauer	7,6 ccm Gesamtquant. Gesamtazidität 132	$2^1/_2$ Stunden Sekretionsdauer	6,7 ccm Gesamtquant. Gesamtazidität 128

Versuch Nr. 10 am Hund „Moor".

200 ccm Leitungswasser 8 h 55		200 ccm Leitungswasser + 10g Bismutum subnitricum = 5% 11 h —	
Zeit	Menge	Zeit	Menge
9 h 25	2,0	11 h 30	2,0
9 h 55	1,5	12 h —	1,6
10 h 25	1,0	12 h 20	0,5
10 h 40	0,0	1 h —	0,0
$1^1/_4$ Stunden Sekretionsdauer	4,5 ccm Gesamtquant. Gesamtazidität 134	$1^1/_2$ Stunden Sekretionsdauer	4,1 ccm Gesamtquant. Gesamtazidität 126

Versuch Nr. 11 am Hund „Karo".

200 ccm Leitungswasser 8 h 55		200 ccm Leitungswasser + 20 g Bismutum subnitricum = 10% 2 h —	
Zeit	Menge	Zeit	Menge
9 h 25	0,3	2 h 30	1,8
9 h 55	3,3	3 h —	1,5
10 h 25	4,0	3 h 30	0,4
10 h 55	0,0	4 h —	0,0
$1^1/_2$ Stunden Sekretionsdauer	7,6 ccm Gesamtquant. Gesamtazidität 132	$1^1/_2$ Stunden Sekretionsdauer	3,7 ccm Gesamtquant. Gesamtazidität 126

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Versuch Nr. 12 am Hund „Karo".

200 ccm Leitungswasser		200 ccm Leitungswasser + 20 g Bismutum subnitricum = 10 %	
8 h 55		2 h 30	
Zeit	Menge	Zeit	Menge
9 h 25	3,0	2 h 30	0,0
9 h 55	1,5	3 h —	0,2
10 h 25	1,0	3 h 30	0,0
10 h 40	0,0	4 h —	0,0
1 1/4 Stunden Sekretionsdauer	4,5 ccm Gesamtquant. Gesamtazidität 134	1 Stunde Sekretionsdauer	0,2 ccm Gesamtquant. Gesamtazidität ?

Versuch Nr. 13 am Hund „Karo".

200 ccm Leitungswasser		200 ccm Leitungswasser + 10 g Bismutum subnitricum = 5 %	
7 h 30		1 h —	
Zeit	Menge	Zeit	Menge
8 h —	1,3	1 h 30	2,0
8 h 30	2,2	2 h —	4,0
9 h —	1,8	2 h 30	1,4
9 h 15	1,0	3 h —	0,6
9 h 30	0,6	3 h 30	0,2
9 h 45	0,0	3 h 45	0,0
1 3/4 Stunden Sekretionsdauer	6,9 ccm Gesamtquant. Gesamtazidität 120	2 1/4 Stunden Sekretionsdauer	8,2 ccm Gesamtquant. Gesamtazidität 150

Versuch Nr. 14 am Hund „Nero".

200 ccm Leitungswasser		200 ccm Leitungswasser +20g Bismutum subnitricum = 10 %	
8 h —		3 h —	
Zeit	Menge	Zeit	Menge
8 h 30	2,0	3 h 30	1,8
9 h —	1,2	4 h —	2,2
9 h 30	1,8	4 h 30	2,0
10 h —	0,2	4 h 45	0,8
10 h 30	0,0	5 h —	0,6
		5 h 15	0,3
		5 h 30	0,0
2 Stunden Sekretionsdauer	5,2 ccm Gesamtquant. Gesamtazidität 128	2 Stunden Sekretionsdauer	7,7 ccm Gesamtquant. Gesamtazidität 130

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Versuch Nr. 15 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser + 10 g Bismutum subnitricum = 5 %	
7 h 30		1 h 30	
Zeit	Menge	Zeit	Menge
8 h —	3,2	2 h —	2,5
8 h 30	2,4	2 h 30	0,2
9 h —	2,0	3 h —	0,2
9 h 15	1,6	3 h 30	0,0
9 h 30	0,8		
9 h 45	0,0		
$1^3/_4$ Stunden Sekretionsdauer	10,0 ccm Gesamtquant. Gesamtazidität 150	$1^1/_2$ Stunden Sekretionsdauer	2,9 ccm Gesamtquant. Gesamtazidität 120

Wenn wir aus den vorstehenden Bismutversuchen uns die Frage der sekretorischen Wirkung des Bismut subnitricum auf die Drüsenfunktion des Magens vorlegen, so müssen wir uns zunächst den chemischen Vorgang vergegenwärtigen, der durch Einwirkung der HCl des Magens auf das Bismutsalz vor sich geht. Dieser entspricht folgender Gleichung:

$$Bi\langle^{NO_3}_{O} + HCl = HNO_3 + Bi\langle^{Cl}_{O}$$

In diesem Resultate der Salzsäureeinwirkung ist der sekretorische Einfluß, wie wir des nähern weiter unten sehen werden, nicht auf das Bismutoxychlorid, sondern auf die Salpetersäure zurückzuführen.

Bei der Durchsicht obiger Versuche treffen wir einen dreifachen Typus der Reaktion der Magenschleimhaut auf das Bismutum subnitricum an: 1. Eine typische Sekretionshemmung, indem die Quantität und wahrscheinlich auch die Qualität des Sekretes herabgesetzt wird; bei letzterer sind allerdings physiologische Schwankungen, wie wir im Verlaufe dieser Untersuchungen mehrfach antreffen werden, in Berücksichtigung zu ziehen, und dadurch liegen keine eindeutigen Verhältnisse vor. Auf alle Fälle kann man hier eine Neigung zur Herabsetzung der Magensaftsekretion durch das Bismutum subnitricum annehmen. Diese Art der Reaktion der Magenschleimhaut dürfte den gewöhnlichen Reaktionstypus der Magensekretion gegenüber dem Bismutnitrat bilden, ist aber nur bei einer normalen, nicht entzündlichen Schleimhaut, nach unseren Versuchstieren zu schließen, anzutreffen. 2. Einen relativen Indifferentismus der Magenschleimhaut gegenüber dem Bismutnitrat. Dieses dürfte der seltenere Reaktionstypus und ebenfalls bloß

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bei normaler Magenschleimhaut anzutreffen sein. 3. Einen Reaktionstypus im Sinne einer ausgesprochenen Hemmung der Sekretion und zwar bloß bei einer entzündlich veränderten Magenschleimhaut. Die Erklärung hierfür will ich in den folgenden Experimenten zu geben versuchen.

Diese in drei verschiedenen Arten verlaufenden Versuche lassen uns über die Wismutwirkung auf die Sekretionsverhältnisse der Magenschleimhaut folgende Schlüsse ziehen:

1. Aus allen sieben Versuchen ergibt sich vor allem übereinstimmend der Umstand, daß dem Bismutum subnitricum auf jeden Fall keine sekretionssteigernde Wirkung zugeschrieben werden kann.

2. In der Mehrzahl der Versuche zeigt in Übereinstimmung mit biologisch-experimentellen Untersuchungen (Heinsheimer und nach meinen eigenen klinischen Beobachtungen) das Bismutum subnitricum eine deutliche Hemmung auf die Magensaftsekretion und zwar ausgesprochen auf die Quantität der Salzsäure und andeutungsweise auch auf die Qualität (prozentualer HCl-Gehalt).

3. Mehr ausnahmsweise reagiert die Magensaftsekretion in ziemlich indifferenter Weise auf die Einwirkung des Bismutum subnitricum. Die Erklärung hierfür, warum in den einen Fällen die Drüsensekretion im Sinne einer Irritation, in den anderen in indifferenter Weise reagiert, ist wenigstens aus manifesten, anatomischen Gründen nicht ersichtlich, weil es sich in beiden Fällen um eine anatomisch intakte Schleimhaut handelt. In Ermangelung einer besseren Erklärung müßte man sich mit derjenigen einer „individuellen Prädisposition" für größere und geringere Empfindlichkeit gegenüber dem einwirkenden Agens behelfen.

4. Die im Sinne einer Entzündung veränderte Schleimhaut des Magens reagiert auffallend auf die Einwirkung des Bismutum subnitricum mit einer erheblichen Sekretionsherabsetzung, die bedeutend unter der sub 2 angeführten steht (Versuch am Hunde „Max" mit chronischer Gastritis).

Diese Art der Reaktion der entzündeten Magenschleimhaut, im Gegensatz zur anatomisch intakten Mukosa, ist nicht nur eine dem Bismutum subnitricum gegenüber eigentümliche, sondern, wie wir weiter unten sehen werden, teilweise in allerdings gegensätzlichem Endeffekte auch gegenüber anderen Wismutverbindungen, sowie auch weiteren nicht dem Wismut angehörenden Adstringentien.

Die folgenden Experimente befassen sich mit einem neuen Präparate, dem Bismutum bitannicum[1]), dem doppeltgerbsauren

1) Chem. Fabrik von Heyden.

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Bismutum. Das Bismutum tannicum hat in der Therapie keine Rolle gespielt, wir finden es in der Literatur kaum erwähnt. Beispielsweise spricht ihm Gutmann[1]) jeden therapeutischen Wert bei Magen- und Darmaffektionen ab und empfiehlt an seiner Stelle das Bismutum salicylicum. — Dem neuen Präparate sollen theoretisch gewisse Vorzüge vor dem alten eigen sein. So äußert sich die Fabrik im Prospekte: „Das bisherige Bismutum tannicum enthält ein schwer abspaltbares Tanninmolekül, das neue Präparat enthält außerdem noch ein zweites Tanninmolekül, welches leicht abspaltbar ist. Das Bismutum bitannicum besitzt deshalb eine stärkere Tanninheilwirkung bei gleichzeitiger Abwesenseit von unangenehmen Eigenschaften des freien Tannins."

Zunächst habe ich das Präparat an einem Hunde mit gesunder Magenschleimhaut geprüft:

Versuch Nr. 16 am Hund „Nero".

200 ccm Leitungswasser		200 ccm Leitungswasser + 20 g Bismutum bitannicum = 10 %	
8 h —		5 h —	
Zeit	Menge	Zeit	Menge
8 h 30	3,0	2 h 30	2,8
9 h —	2,2	3 h —	5,4
9 h 30	1,8	3 h 30	7,0
10 h —	0,1	4 h —	6,8
10 h 15	0,0	4 h 30	4,2
		5 h —	3,4
		Fütterung um 5 Uhr	
1½ Stunden Sekretionsdauer	7,1 ccm Gesamtquant. Gesamtazidität 132	Über 2½ St. Sekretionsdauer	29,6 ccm Gesamtquant. Gesamtazidität 180

Wir sehen aus diesen Versuchen eine erhebliche Steigerung der Sekretionsmenge und der Sekretionsdauer; die Azidität ist nicht nennenswert beeinflußt.

Einen anderen Verlauf nimmt das Experiment mit dem Hunde mit chronischer Gastritis (mit normaler Azidität):

1) Deutsche med. Wochenschr. 1886, Nr. 22.

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Versuch Nr. 17 am Hund „Max".

200 ccm Leitungswasser 8 h 30		200 ccm Leitungswasser + 10 g Bismutum bitannicum = 5 % 11 h 30	
Zeit	Menge	Zeit	Menge
9 h —	3,1	12 h —	1,6
9 h 30	3,2	12 h 30	1,6
10 h —	2,2	1 h —	1,8
10 h 30	2,4	1 h 30	0,3
11 h —	1,4	2 h —	2,0
11 h 15	1,5	2 h 15	0,6
11 h 40	0,1	2 h 30	0,1
$2^1/_2$ Stunden Sekretionsdauer	13,9 ccm Gesamtquant. Gesamtazidität 142	$2^1/_2$ Stunden Sekretionsdauer	8,0 ccm Gesamtquant. Gesamtazidität 138

Diesem Versuche liegt eine Fehlerquelle zugrunde: Das Tier war am Abend vorher später als gewohnt gefüttert worden, so daß es am Morgen des Versuchstages nicht ganz nüchtern bzw. die physiologische Sekretion auf die Fütterung noch nicht völlig abgeklungen war; deshalb ist beim Leitungswasserversuch sowohl die Sekretionsdauer als das Sekretionsquantum im Vergleiche mit ähnlichen Experimenten am gleichen Hunde erheblich gesteigert. Diese Steigerung beeinträchtigt dadurch den Resultatwert des darauf folgenden Experimentes. Vgl. folgenden Versuch:

Versuch Nr. 18 am Hund „Max".

200 ccm Leitungswasser 8 h —		200 ccm Leitungswasser + 10 g Bismutum bitannicum = 5 % 10 h 30	
Zeit	Menge	Zeit	Menge
8 h 30	0,4	11 h —	0,6
9 h —	0,6	11 h 30	0,3
9 h 30	0,6	12 h —	0,1
10 h —	0,4		
10 h 15	0,3		
10 h 30	9,0		
2 Stunden Sekretionsdauer	2,3 ccm Gesamtquant. Gesamtazidität 136	1 Stunde Sekretionsdauer	1,0 ccm Gesamtquant. Gesamtazidität appr. 120

Sehr interessant ist hier wiederum die Tatsache, daß der sekre-

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torische Apparat, wenn der Schleimhaut entzündliche Veränderungen zugrunde liegen, anders als bei gesunden Verhältnissen reagiert: statt der Steigerung der Sekretion haben wir hier eine ausgesprochene Hemmung. Beide Versuche sind mit dem gleichen Tiere angestellt. Bei dem einen ist die Sekretionsdauer durch das Präparat nicht beeinflußt, wohl aber die Sekretionsmenge um ca. 60% geringer; bei dem anderen ist sowohl die Dauer wie das Quantum erheblich herabgesetzt und beträgt kaum die Hälfte der Werte der Wasserperiode. Im Experiment Nr. 17 liegt aber, wie erwähnt, ein Fehler vor. In allen drei Fällen ist der prozentuale HCl-Gehalt des Magensaftes nicht nennenswert, d. h. nicht außerhalb der physiologischen Schwankungen beeinflußt.

Der chemische Vorgang im Magen ist demjenigen beim Bismutum subnitricum analog:

$$\text{Bi . bitannic.} + \text{HCl} = \text{Bi}\langle\begin{matrix}\text{O}\\\text{Cl}\end{matrix} + \text{Acid. tannic.}$$

Daß auch hier nicht das Bismutoxychlorid das wirksame Agens sein kann, ergibt sich schon durch den Vergleich mit der Wirkung des Bism. subnitr. Wäre in beiden Fällen das $\text{Bi}\langle\begin{matrix}\text{O}\\\text{Cl}\end{matrix}$ der sekretionssteigernde Körper, so müßten das Bismut. subnitr. und das Bismut. bitann. genau die gleiche sekretorische Wirkung sowohl auf den gesunden wie auch auf den entzündeten Magen ausüben. Da dies aber nicht der Fall ist, muß diese spezifische Wirkung in der freigewordenen Säure zu suchen sein, also im Tannin, ähnlich wie in den anderen Versuchen in der Salpetersäure. — Übrigens hat Herr Prof. Bickel nachträglich auf meine Bitte hin den Einfluß des Acid. tannic. selbst an Tierexperimenten geprüft und folgendes gefunden:

1. Versuch (Bickel).

150 ccm Leitungswasser		150 ccm 1prozentige wässerige Lösung von Acidum tannicum	
Zeit	Menge	Zeit	Menge
11 h— bis 11 h 30	0,6	1 h — bis 1 h 30	0,2
11 h 30 bis 12 h—	0,4	1 h 30 bis 2 h —	0,0
12 h— bis 12 h 30	0,6	2 h — bis 2 h 30	0,0
12 h 30 bis 1 h —	0,2	2 h 30 bis 3 h —	0,0
2 Stunden Sekretionsd.	1,8 ccm Gesamtquant.	$^1/_2$ Stunde Sekretionsd.	0,2 ccm Gesamtquant.

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2. Versuch (Bickel).

200 ccm 1 prozentige Lösung von Acidum tannicum rufen überhaupt keine Sekretion hervor.

Das Tannin hat also nach meinen Versuchen eine sekretionssteigernde Wirkung und zwar da, wo die Schleimhaut intakt ist. Die adstringierende Wirkung muß hier eine nur wenig intensive und sehr oberflächliche sein und äußert sich in ihrem Endeffekte als eine Irritation, eine Anregung der Drüsennerven bzw. der Drüsentätigkeit. Im anderen Falle ist der Endeffekt ein umgekehrter, eine Depression, eine Hemmung. Die entzündete, hyperämische und aufgelockerte Mukosa läßt eine intensivere Adstringierung zustande kommen, d. h. die eiweißhaltigen und leimgebenden Substanzen der Schleimhaut zeigen unter den Vorgängen der Entzündung die schon physiologisch vorhandene Affinität zum Tannin in erhöhtem Maße. Hand in Hand mit der Adstringierung geht auch die Hemmung der Sekretion. Es ist dies ein typisches Beispiel für den engen Zusammenhang zwischen Entzündung und Sekretionsstörung und für einen gewissen Parallelismus in der medikamentösen, therapeutischen Beeinflußbarkeit der Entzündung und der Sekretionsanomalie, wie dies schon in der Einleitung dieser Arbeit angedeutet wurde.

Des weitern wurde eine Bismutsalizylverbindung, ebenfalls ein neues Präparat, untersucht, das Bismutum bisalicylicum. Die Fabrik äußert sich folgendermaßen über das Präparat: Bismutum bisalicylicum ist eine unlösliche Verbindung von 1 Atom Wismut mit 2 Molekülen Salizylsäure, von denen das eine Molekül unter Bildung des gewöhnlichen Bismutum salicylicum sehr leicht abgespaltet wird. Die abgespaltete Salizylsäure wird in dem Maße, wie sie entsteht, sofort resorbiert. Die Abspaltung des anderen Moleküls Salizylsäure aus dem intermediär entstehenden gewöhnlichen Bismutum salicylicum erfolgt wesentlich schwerer. Das neue Bismutum bisalicylicum wirkt mit der einen Hälfte seiner Salizylsäure sehr prompt, mit der anderen Hälfte dagegen genau wie das bekannte Bismutsalizylat allmählich. Deshalb zeichnet sich das Bismutum bisalicylicum nicht nur als Magen-, Darmantiseptikum vor dem Bismutum salicylicum aus, sondern auch als schnell und stark wirkendes Salizylmittel.“ Zunächst wurde auch hier das Präparat an einem Hunde mit intakter Magenschleimhaut geprüft:

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Versuch Nr. 19 am Hund „Karo".

200 ccm Leitungswasser		200 ccm Leitungswasser + 10 g Bismutum bisalicylicum = 5 %	
7 h 30		9 h 45	
Zeit	Menge	Zeit	Menge
8 h —	1,3	10 h 15	12,5
8 h 30	2,2	10 h 45	10.0
9 h —	1,8	11 h 15	5,8
9 h 15	1,0	11 h 45	1,2
9 h 30	0,6	12 h 45	0,0
9 h 45	0,0		
$1^3/_4$ Stunden Sekretionsdauer	6,9 ccm Gesamtquant. Gesamtazidität 120	$2^1/_2$ Stunden Sekretionsdauer	29,5 ccm Gesamtquant. Gesamtazidität 132

Die sekretionssteigernde Wirkung ist hier eine auffallende. Die Sekretionsperiode ist etwa doppelt so groß, die Sekretionsmenge etwa dreimal stärker als bei der Wasserperiode. Die Azidität scheint gesteigert zu sein, immerhin nicht in typischer Weise.

Nun folgen drei Versuche an Hunden mit der chronischen Gastritis. A priori dürfte man hier vielleicht auf eine Analogie zu den vorhergehenden Versuchen gefaßt sein, allein dies ist hier nicht zutreffend, wie uns folgende Versuche beweisen:

Versuch Nr. 20 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser + 6 g Bismutum bisalicylicum = 3 %	
9 h 35		11 h 33	
Zeit	Menge	Zeit	Menge
10 h 05	0,2	10 h 05	0,2
10 h 35	0,5	12 h 35	0,6
11 h 05	0,6	1 h 05	1,2
11 h 35	0,0	1 h 35	0,2
		2 h 05	0,0
$1^1/_2$ Stunden Sekretionsdauer	1,3 ccm Gesamtquant. Gesamtazidität ca. 140	2 Stunden Sekretionsdauer	2,2 ccm Gesamtquant. Gesamtazidität 146

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Versuch Nr. 21 am Hund „Max".

200 ccm Leitungswasser 7 h 30		200 ccm Leitungswasser + 10 g Bismut. bisalicylicum = 5 % 9 h 45	
Zeit	Menge	Zeit	Menge
8 h —	3,2	10 h 15	8,5
8 h 30	2,4	10 h 45	5.5
9 h —	2,2	11 h 15	5,4
9 h 15	1,6	11 h 45	2,6
9 h 30	0,8	11 h 45	4,2
9 h 45	0,0	1 h 15	1,2
		1 h 30	0,1
$1^3/_4$ Stunden Sekretionsdauer	10,2 ccm Gesamtquant. Gesamtazidität 150	$3^1/_4$ Stunden Sekretionsdauer	27,5 ccm Gesamtquant. Gesamtazidität 125

Versuch Nr. 22 am Hund „Max".

200 ccm Leitungswasser 8 h 45		200 ccm Leitungswasser + 10 g Bismut. bisalicylicum = 5 % 1 h 45	
Zeit	Menge	Zeit	Menge
9 h 15	4,0	2 h 15	4,4
9 h 45	1,0	2 h 45	1,2
10 h 15	0,2	3 h 15	1,2
10 h 45	1,0	3 h 45	0,4
11 h 45	0,0	4 h —	0,2
		4 h 15	0,0
$2^1/_2$ Stunden Sekretionsdauer	6,2 ccm Gesamtquant. Gesamtazidität 138	2 Stunden Sekretionsdauer	7,4 ccm Gesamtquant. Gesamtazidität 142

In allen der drei letzten Versuche wird durch Einwirkung des Bismutum bisalicylicum die Sekretionsdauer und die Sekretionsmenge erheblich gesteigert: im ersten Versuch etwa um das Doppelte, im zweiten etwa das Dreifache und im dritten Versuche weniger auffallend, immerhin noch um etwa 25%. Hier dürfte der Umstand die geringere Sekretionssteigerungsfähigkeit erklären, daß diesem Wismutversuche ein Experiment mit Albargin, einer sekretionshemmenden Substanz, ziemlich unmittelbar vorangegangen und daß als Nachwirkung davon die Drüsensekretion weniger erregbar war, als wenn eine Wasserperiode unmittelbar vorausgegangen wäre. Der Einfluß

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auf die prozentuale Azidität ist auch hier nicht typisch, er schwankt innerhalb physiologischer Grenzen bald im Sinne einer leichten Steigerung, bald einer geringen Hemmung. Der die Sekretion direkt anregende Körper ist hier die im Magen freiwerdende Salizylsäure. Diese hat also einen sekretionssteigernden Einfluß, sowohl auf die gesunde wie auf die entzündliche Magenschleimhaut. Das Bismutum bisalicylicum wie folgerichtig auch das Bismutum salicylicum können also niemals zur Erzielung einer Herabsetzung der Sekretion verwendet werden. Dieses gegensätzliche Verhalten der Bismutsalizylate zu dem Bismutum subnitricum und dem Bismutum bitannicum läßt sich pharmakologisch vielleicht dadurch einigermaßen erklären, daß der schwer löslichen Salizylsäure, im Gegensatze zur Salpeter- und Digallussäure (Tannin) eine lokal ätzende Wirkung abgeht. Dadurch kommt es auch nicht zu einer Adstringierung der Magenschleimhaut. Die Pharmakodynamik dürfte also hier eine ganz andere sein und ihr freilich nicht ein klares chemisch-anatomisches Substrat zugrunde liegen, wie dem Tanninsalze. Dementsprechend ist auch der Endeffekt der Wirkung ein anderer.

Zum Abschluß dieser Wismutversuche habe ich, lediglich von praktischen Gründen geleitet, zwei Körper untersucht, welche in der neueren medikamentösen Therapie häufig in Verbindung mit Bismutum subnitricum verordnet werden, das Anästhesin und Orthoformum novum. Da die Hauptindikation ihrer internen Anwendung das Ulcus ventriculi ist, halte ich es für geboten, sich über den Einfluß dieser Lokalanästhetika auf die Magensaftabscheidung zu orientieren und lasse deshalb zunächst Versuche über Aufschwemmungen mit Anästhesin resp. Orthoform ohne Zusatz von Bismutum subnitricum folgen.

Die beiden folgenden Versuche sind am Hunde mit dem Magenkatarrh vorgenommen.

Versuch Nr. 23 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser + 2 g Anästhesin = 1 %	
7 h 45		10 h 45	
Zeit	Menge	Zeit	Menge
8 h 05	0,4	11 h 15	0,7
8 h 45	0,0	11 h 55	0,5
9 h —	0,0	12 h —	0,2
9 h 15	0,0	12 h 15	0,0
½ Stunde Sekretionsdauer	0,4 ccm Gesamtquant. Gesamtazidität ?	1 Stunde Sekretionsdauer	1,4 ccm Gesamtquant. Gesamtazidität 140?

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Versuch Nr. 24 am Hund „Max".

200 ccm Leitungswasser 8 h 30		200 ccm Leitungswasser + 2 g Anästhesin = 1 % 10 h 30	
Zeit	Menge	Zeit	Menge
9 h —	2,5	11 h —	2,5
9 h 30	1,0	11 h 15	1,6
10 h —	2,1	11 h 45	1,5
10 h 15	0,3	12 h 15	1,2
10 h 30	0,0	12 h 45	0,8
		1 h 45	0,8
		2 h 15	0,6
		2 h 45	0,2
		3 h —	0,0
$1^1/_2$ Stunden Sekretionsdauer	5,9 ccm Gesamtquant. Gesamtazidität 128	4 Stunden Sekretionsdauer	9,2 ccm Gesamtquant. Gesamtazidität 134

Der erste Versuch zeigt die Tendenz zu einer Steigerung, der zweite eine Steigerung mäßigen Grades in bezug auf die Saftmenge und eine relativ längere Sekretionsperiode. Auch hier läßt sich keine besonders typische Beeinflussung der Azidität konstatieren.

Ein ähnliches Bild bieten die drei folgenden Versuche mit Orthoform (Orthoformum novum):

Versuch Nr. 25 am Hund „Karo".

200 ccm Leitungswasser 7 h 45		200 ccm Leitungswasser + 2 g Orthoformum novum = 1 % 9 h 15	
Zeit	Menge	Zeit	Menge
8 h 15	0,3	9 h 45	0,0
8 h 45	0,2	10 h 15	0,5
9 h —	0,1	10 h 30	0,3
9 h 15	0,0	10 h 45	0,0
1 Stunde Sekretionsdauer	0,6 ccm Gesamtquant. Gesamtazidität ?	1 Stunde Sekretionsdauer	0,8 ccm Gesamtquant. Gesamtazidität ?

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Versuch Nr. 26 am Hund „Max".

200 ccm Leitungswasser 7 h 45		200 ccm Leitungswasser + 2 g Orthoformum novum = 1 % 9 h 15	
Zeit	Menge	Zeit	Menge
8 h 15	0,4	9 h 45	0,0
8 h 45	0,0	10 h 15	0,9
9 h —	0,0	10 h 30	0,4
9 h 15	0,0	10 h 45	0,0
$^1/_2$ Stunde Sekretionsdauer	0,4 ccm Gesamtquant. Gesamtazidität ?	1 Stunde Sekretionsdauer	1,3 ccm Gesamtquant. Gesamtazidität 140 ?

Versuch Nr. 27 am Hund „Max".

200 ccm Leitungswasser 8 h —		200 ccm Leitungswasser + 10 g Orthoformum novum = 0,5 % 2 h —	
Zeit	Menge	Zeit	Menge
8 h 30	4,0	2 h 30	0,7
9 h —	1,4	3 h —	1,0
9 h 30	1,6	3 h 30	0,3
10 h —	0,4	4 h —	0,2
10 h 15	0,1	4 h 30	0,0
$1^3/_4$ Stunden Sekretionsdauer	7,5 ccm Gesamtquant. Gesamtazidität 136	2 Stunden Sekretionsdauer	2,2 ccm Gesamtquant. Gesamtazidität 128

Auch hier tritt in zwei Versuchen die Neigung des Orthoforms zutage die Sekretion zu erhöhen, aber in bedeutend weniger ausgesprochener Weise als beim Anästhesin. Im dritten Versuch hingegen findet sich eine Sekretionshemmung, welche ihre Erklärung in einem unmittelbar vorausgegangenen Albarginversuche finden dürfte. Die ganz leichte Sekretionssteigerung äußert sich sowohl beim Hund mit gesunder, als auch beim Hunde mit entzündeter Magenschleimhaut. Da beim Anästhesin und beim Orthoform der gleiche Hund (mit der Gastritis) unter den gleichen Bedingungen zum Experimentieren diente, dürfen wir die Anästhesin- und die Orthoformversuche in ihren Resultaten miteinander vergleichen und wohl den Schluß ziehen, daß das Orthoform die Magenschleimhaut bzw. die Magendrüsen weniger „reizt" als das Anästhesin. Ob die beiden chemisch

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komplizierten Körper, Derivate der Amidobenzoesäure (Para-Amidobenzoesäureäthylester = Anästhesin), resp. der Oxybenzoesäure (Meta-amido-para-Oxybenzoesäuremethylester = Orthoformum novum) durch die einwirkende HCl Veränderungen erleiden, ist fraglich, jedenfalls sind aber erhebliche Umbildungen nicht möglich, und so müssen wir wohl annehmen, daß diese Körper als solche die Wirkung auf die Sekretion ausüben, und daß diese keine erheblich irritative ist.

Nun folgt je ein Versuch eines Gemisches von Orthoform bzw. Anästhesin und Bismutum subnitricum im Verhältnis 1:9g auf 200g Wasser, also ungefähr in einer therapeutisch üblichen Dosis. Die Experimente sind am gleichen Tiere (mit Gastritis) durchgeführt.

Versuch Nr. 28 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser + 9 g Bismutum subnitricum = 1 g Orthoformum novum	
8 h 30		11 h —	
Zeit	Menge	Zeit	Menge
9 h —	1,0	11 h 30	0,2
9 h 30	2,0	12 h —	1,4
10 h —	0,6	12 h 30	1,3
10 h 30	1,9	1 h —	1,0
10 h 45	0,8	1 h 30	0,1
11 h —	0,1		
2 Stunden Sekretionsdauer	6,4 ccm Gesamtquant. Gesamtazidität 130	2 Stunden Sekretionsdauer	4,0 ccm Gesamtquant. Gesamtazidität 128

Versuch Nr. 29 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser + 9 g Bismutum subnitricum = 1 g Anästhesin	
8 h 30		1 h 30	
Zeit	Menge	Zeit	Menge
9 h —	1,0	2 h —	0,6
9 h 30	2,1	2 h 30	0,8
10 h —	0,6	3 h —	1,0
10 h 30	1,9	3 h 15	0,6
10 h 45	0,8	3 h 30	0,1
11 h	0,1		
2 Stunden Sekretionsdauer	6,5 ccm Gesamtquant. Gesamtazidität 130	$1^1/_2$ Stunden Sekretionsdauer	3,1 ccm Gesamtquant. Gesamtazidität 132

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Diese Versuche ergeben eine deutliche Herabsetzung der Sekretionsmenge. Sie sind sehr interessant, weil sie einerseits die obenerwähnte sekretorische Hemmung des Bismutum subnitricum bestätigen, andererseits, weil in dem Gemische die hemmende Komponente (das Bismutum subnitricum) die steigernde (das Anästhesin resp. Orthoform) an Wirkung übertrifft, und weil dadurch die Resultante eine ausgesprochene Hemmung ist. — Über die prozentuale Azidität läßt sich auch hier nichts Charakteristisches sagen.

Die Versuche könnten auch den Schluß zulassen, daß das Anästhesin die hemmende Wismutwirkung weniger beeinträchtigen würde als das Orthoform. Demgegenüber ist zu sagen, daß der Anästhesinversuch im Anschluß an das Orthoformexperiment gemacht worden ist, wo also eine hemmende Nachwirkung auf den Drüsenapparat (wegen der Wismutkomponente) nicht ausgeschlossen war. Der Unterschied in den gefundenen Mengewerten ist überdies hier zu klein, um daraus bindende Schlüsse auf eine spezifische Anästhesin- oder Orthoform-Komponentenwirkung zu ziehen. Das Hauptinteresse ist das Resultat dieser Versuche, daß ein Gemisch dieser therapeutisch wichtigen Medikamente sekretionshemmend wirkt.

Seit vielen Jahrzehnten hat das Argentum nitricum sich des Rufes eines guten „Magenmittels" erfreut. Schon im Jahre 1829 wurde es von Johnson[1]) gegen die Magenschmerzen von Epileptikern als lokales Analgetikum angewendet. In einer Art Monographie über das Präparat empfiehlt es Krüger[2]) bei Gastromalacie, ferner bei Pyrosis gastrica und cardiaca, sowie auch bei Gastralgia nervosa. Lane[3]) schreibt die gleichen therapeutischen Eigenschaften dem weniger kaustisch wirkenden Silberoxyd zu. Auf die Pharmakodynamik des Arg. nitr. geht Küchenmeister[4]) näher ein, indem er bei Empfehlung des Medikamentes gegen Ulcus ventriculi darauf hinweist, daß es die Eigenschaft habe, mit den Albuminaten gallertartige Verbindungen einzugehen und nach erfolgter Ätzung des Geschwüres dasselbe mit einer Decke von koaguliertem Eiweiß zu versehen, ähnlich wie die Gerbsäure, die arsenige Säure, der Bleiessig, der Alaun usw. Mit den Jahren hat das Arg. nitr. bei Magenkrankheiten, besonders beim Ulkus, immer mehr Anhänger gefunden, wie Ewald, Liebermeister, Rosenheim, Boas, Lion u. a., aber auch Gegner, wie Rosenthal, Roßbach und Nothnagel.

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1) Nach Baibakoff zitiert (siehe unten).
2) Prakt. und krit. Mitteilungen von Pfaff, 7. Jahrg. 1841.
3) Lancet, July 1841.
4) Wiener med. Wochenschr. 1854, Nr. 14 und 15.

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Die Mehrzahl der Autoren, welche das salpetersaure Silber bei Magenerkrankungen anwenden, halten besonders diejenigen Zustände als für die Silbertherapie geeignet, welche mit einer Steigerung der HCl-Sekretion verbunden sind, von der Annahme ausgehend, daß das Präparat sekretionshemmend auf die Drüsenfunktion des Magens wirke. Diese Frage nach der Beeinflußbarkeit der Magensaftabscheidung durch das Arg. nitr. hat neulich Baibakoff[1]) klinisch eingehend geprüft und ist zu dem Resultate gelangt, daß in der Mehrzahl der Fälle das Medikament eine säuresteigernde Wirkung besonders in quantitativer, prozentualer Hinsicht aufweise. Dieser Autor hat durch das Probefrühstück von Ewald sowohl Sekretionsneurosen als auch Gastritiden mit normaler und anormaler Azidität, sowie Fälle von Ulcus ventriculi geprüft und hat als Resultat seiner Untersuchungen die Indikation aufgestellt, das Arg. nitr. bei Zuständen von Subazidität, besonders von schleimigem Katarrh zu verordnen, während seine Anwendung bei denjenigen Magenerkrankungen, die mit einer gesteigerten Azidität einhergehen, kontraindiziert sei. Dabei gibt der Autor aber auch an, daß manche seiner Fälle mit einer Sekretionsherabsetzung auf die Arg. nitr.-Anwendung reagiert hätten; mithin ist die Wirkung des Präparates auf die Magendrüsenfunktion keine einheitliche, d. h. sie kann unter Umständen eine hemmende sein. Worin dieser doppelte Modus der Sekretionswirkung besteht, resp. warum der eine Magen im Sinne einer Irritation, der andere einer Depression reagiert, hat der Autor nicht untersucht und in dieser Unterlassung liegen, meiner Ansicht nach, die Mängel obiger Indikationsaufstellung. Aus Analogie zu den anderen hier besprochenen Tierexperimenten habe ich nun versucht, in der Frage der Einwirkung der Silbersalze auf experimentell-biologischem Wege einige Klarheit zu verschaffen und habe als Prototypen der Silberwirkung nicht das Arg. nitr. als solches, sondern zwei organische Silberverbindungen geprüft, denen ich aus pharmakologischen und klinischen Gründen vor dem Salpetersilber den Vorzug gebe, das Protargol und das Albargin.

Das Protargol, ein Silbereiweißderivat mit 8,3% Silber in organischer Bindung, wurde zuerst in einer 0,5prozentigen Lösung an einem Hunde mit normaler Beschaffenheit der Magenschleimhaut geprüft und der Versuch verlief folgendermaßen:

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1) Arch. f. Verdauungskrankheiten 1906, Bd. 12, Heft 1.

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Versuch Nr. 30 am Hund „Nero".

200 ccm Leitungswasser 8 h —		200 ccm Leitungswasser + 1 g Protargol = 0,5 % 10 h 15	
Zeit	Menge	Zeit	Menge
8 h 30	2,0	10 h 45	5,8
9 h —	1,2	11 h 15	6,0
9 h 30	1,8	11 h 45	6,2
10 h —	0,2	12 h 15	2,3
10 h 15	0,0	12 h 45	2,0
		1 h 15	1,8
		1 h 45	0,8
		2 h 15	0,5
		2 h 45	0,4
		3 h —	0,2
1 3/4 Stunden Sekretionsdauer	5,2 ccm Gesamtquant. Gesamtazidität 123	4 1/4 Stunden Sekretionsdauer	26,0 ccm Gesamtquant. Gesamtazidität 134

Aus diesem Versuche ergibt sich eine hochgradige Steigerung der Sekretionsmenge in der Zeiteinheit (von 1/2 Stunde), des Gesamtsekretionsquantums um etwa das Fünffache gegenüber der Wasserperiode und der Sekretionsdauer um mehr als das Doppelte. In bezug auf die prozentuale Azidität scheint die Neigung zu einer leichten Erhöhung derselben zu bestehen, jedoch auch hier in keiner typischen Weise.

Ein ganz anderes Bild hingegen bieten die folgenden vier Versuche am Hunde mit der chronischen Gastritis (ohne Aziditätsstörung).

Versuch Nr. 31 am Hunde „Max".

200 ccm Leitungswasser 8 h 30		200 ccm Leitungswasser + 1 g Protargol = 0,5 % 3 h —	
Zeit	Menge	Zeit	Menge
9 h —	2,5	3 h 30	2,8
9 h 30	1,0	4 h —	0,6
10 h —	2,1	4 h 30	0,2
10 h 15	0,3	5 h	0,1
10 h 30	0,0		
1 1/2 Stunden Sekretionsdauer	5,9 ccm Gesamtquant. Gesamtazidität 128	1 1/2 Stunden Sekretionsdauer	3,7 ccm Gesamtquant. Gesamtazidität 122

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Versuch Nr. 32 am Hund „Max".

200 ccm Leitungswasser 8 h 30		200 ccm Leitungswasser + 1 g Protargol = 0,5 % 2 h 30	
Zeit	Menge	Zeit	Menge
9 h —	3,1	3 h —	0,5
9 h 30	3,5	3 h 30	1,0
10 h —	2,2	4 h —	0,4
10 h 30	2,4	4 h 30	0,2
11 h —	1,0	5 h —	0,0
11 h 15	1,6		
11 h 30	0,1		
$2^1/_2$ Stunden Sekretionsdauer	13,9 ccm Gesamtquant. Gesamtazidität 142	2 Stunden Sekretionsdauer	2,1 ccm Gesamtquant. Gesamtazidität 134
Nicht ganz nüchtern!			

Versuch Nr. 33 am Hund „Max".

200 ccm Leitungswasser 8 h —		200 ccm Leitungswasser + 1 g Protargol = 0,2 % 1 h 30	
Zeit	Menge	Zeit	Menge
8 h 30	0,4	2 h —	0,3
9 h —	0,6	2 h 30	0,1
9 h 30	0,6	3 h —	0,1
10 h —	0,4		
10 h 15	0,3		
10 h 30	0,0		
2 Stunden Sekretionsdauer	2,3 ccm Gesamtquant. Gesamtazidität 136	1 Stunde Sekretionsdauer	0,5 ccm Gesamtquant. Gesamtazidität 134

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Versuch Nr. 34 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1 g Protargol = 0,5 %	
8 h —		10 h 15	
Zeit	Menge	Zeit	Menge
8 h 30	4,0	10 h 45	2,2
9 h —	1,4	11 h 15	0,6
9 h 30	1,6	11 h 45	0,2
10 h —	0,4	12 h —	0,0
10 h 30	0,1		
2 Stunden Sekretionsdauer	7,5 ccm Gesamtquant. Gesamtazidität 136	$1^1/_4$ Stunden Sekretionsdauer	3,0 ccm Gesamtquant. Gesamtazidität 140

Aus allen diesen vier Versuchen ergeben sich hier bei einer entzündlich veränderten Magenschleimhaut gerade die umgekehrten Verhältnisse wie beim nicht entzündeten Organe: Die Sekretion ist erheblich herabgesetzt, im ersten und dritten Versuche dieser Reihe auf etwa die Hälfte, im zweiten und vierten auf etwa ein Fünftel gegenüber der Wirkung von Leitungswasser. Die Sekretionsdauer ist derjenigen des Wassers ungefähr gleich, eher noch kleiner und beträgt bloß die Hälfte bis ein Drittel der Absonderungszeit beim entsprechenden Versuche am Hunde mit intakter Schleimhaut.

In analoger Weise wurde das Albargin in den gleichen Konzentrationen wie das Protargol geprüft. Zunächst wiederum an einem Hunde mit intakter Magenschleimhaut.

Versuch Nr. 35 am Hund „Nero".

200 ccm Leitungswasser		200 ccm Leitungswasser + 0,4 g Albargin = 0,2 %	
10 h —		2 h —	
Zeit	Menge	Zeit	Menge
10 h 30	3,0	2 h 30	4,8
11 h —	3,0	3 h —	6,0
11 h 30	1,2	3 h 30	3,8
12 h —	2,2	4 h —	1,2
12 h 30	2,0	4 h 30	0,8
1 h —	1,6	5 h —	0,4
1 h 30	1,0	5 h 15	0,2
2 h —	0,5	5 h 30	0,0
4 Stunden Sekretionsdauer	14,5 ccm Gesamtquant. Gesamtazidität 140	3 Stunden Sekretionsdauer	17,2 ccm Gesamtquant. Gesamtazidität 144
Nicht ganz nüchtern!			

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An diesem Experimente ist vor allem ein Fehler zu korrigieren: Das Tier wurde am Vorabend, durch ein Versehen des Wärters, statt mit Pferdefleisch mit gemischter Nahrung („Charité-Futter") gefüttert, welches, wie oben erwähnt, eine bedeutend längere Sekretionsdauer zur Folge hat. Daher war der Hund am Versuchstage nicht „ganz nüchtern", sonst hätte die Magensaftabscheidung auf Leitungswasser nicht 4 Stunden beanspruchen können. Bei völliger Nüchternheit beträgt die relativ lange Sekretionsdauer bei diesem Tiere in den anderen Versuchen nur $1^1/_2$—2 Stunden. Trotzdem haben wir im vorliegenden Experiment eine Erhöhung des Sekretionsquantums, welche bei Ausschaltung des erwähnten Versuchsfehlers einen bedeutend höheren relativen Wert gewinnen würde. Wir kommen damit zu dem Resultat, daß das Albargin analog dem Protargol auf den anatomisch intakten Magen eine die Sekretion anregende Wirkung ausübt. Die prozentuale Gesamtazidität ist nicht wesentlich beeinflußt.

Wie liegen nun diese Verhältnisse bei einer entzündlich veränderten Magenschleimhaut? Die Antwort hierauf geben uns folgende Versuche:

Versuch Nr. 36 am Hund „Max".

200 ccm Leitungswasser 8 h 45		200 ccm Leitungswasser + 1 g Albargin = 0,5 % 11 h —	
Zeit	Menge	Zeit	Menge
9 h 15	4,0	11 h 30	0,8
9 h 45	1,0	12 h —	2,0
10 h 15	0,2	12 h 30	1,8
10 h 45	1,0	1 h —	0,6
11 h —	0,0	1 h 15	0,2
		1 h 30	0,0
$1^3/_4$ Stunden Sekretionsdauer	6,2 ccm Gesamtquant. Gesamtazidität 138	2 Stunden Sekretionsdauer	5,4 ccm Gesamtquant. Gasamtazidität 138
Nicht ganz nüchtern!			

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Versuch Nr. 37 am Hund „Max".

200 ccm Leitungswasser 8 h —		200 ccm Leitungswasser + 0,4 g Albargin = 0,2 % 12 h —	
Zeit	Menge	Zeit	Menge
8 h 30	0,4	12 h 30	0,2
9 h —	0,6	1 h —	0,2
9 h 30	0,6	1 h 30	0,0
10 h —	0,4		
10 h 15	0,3		
10 h 30	0,0		
2 Stunden Sekretionsdauer	2,3 ccm Gesamtquant. Gesamtazidität 136	1 Stunde Sekretionsdauer	0,4 ccm Gesamtquant. Gesamtazidität ?

Versuch Nr. 38 am Hund „Max".

200 ccm Leitungswasser 8 h —		200 ccm Leitungswasser + 1 g Albargin = 0,5 % 12 h —	
Zeit	Menge	Zeit	Menge
8 h 30	4,0	12 h 30	0,3
9 h —	1,4	1 h —	1,3
9 h 30	1,6	1 h 30	0,3
10 h —	0,4	2 h —	0,1
10 h 30	0,1		
2 Stunden Sekretionsdauer	7,5 ccm Gesamtquant. Gesamtazidität 135	$1^1/_2$ Stunden Sekretionsdauer	2,0 ccm Gesamtquant. Gesamtazidität 138

Versuch Nr. 39 am Hund „Max".

200 ccm Leitungswasser 8 h —		200 ccm Leitungswasser + 1 g Albargin = 0,5 % 10 h 15	
Zeit	Menge	Zeit	Menge
8 h 30	1,2	10 h 45	0,2
9 h —	0,8	11 h 15	0,2
9 h 30	0,7	11 h 45	0,2
10 h —	0,2	12 h 15	0,2
10 h 15	0,0	12 h 45	0,0
$1^3/_4$ Stunden Sekretionsdauer	2,9 ccm Gesamtquant. Gesamtazidität 144	2 Stunden Sekretionsdauer	0,8 ccm Gesamtquant. Gesamtazidität 140?

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Bei Versuch Nr. 36 handelt es sich um den gleichen, vorstehend erwähnten Versuchsfehler; trotzdem das Tier nicht ganz nüchtern war, ist auch hier eine Reduktion der Sekretion, allerdings unerheblich, zu konstatieren. Viel typischer ist diese sekretionshemmende Wirkung in den drei folgenden Experimenten ausgedrückt: Das abgeschiedene Magensaftquantum beträgt bloß ein Drittel, ein Viertel, sogar bloß ein Fünftel der auf Leitungswasser abgeschiedenen Menge. Die Sekretionsdauer ist in keiner ausgesprochenen Weise beeinflußt, durchschnittlich überhaupt nicht verkürzt, die prozentuale Salzsäure scheint auch hier auf Albargin eine leichte Reduktion zu erfahren, immerhin in den Grenzen der physiologischen Schwankungen und reagiert also auch auf dieses Adstringens nicht typisch.

Vergleichsweise bestehen zwischen Protargol und Albargin analoge pharmakodynamische Wirkungen auf den Magendrüsenapparat, entsprechend der analogen chemischen Beschaffenheit beider Präparate. Wenn nun aber das Argentum nitricum in beiden Medikamenten der aktive Bestandteil ist, so könnte man aus theoretischen Gründen erwarten, daß derjenige Körper, der mehr Arg. nitr. enthält, das Albargin (15% Ag), auch pharmakodynamisch wirksamer resp. mehr sekretionshemmend wäre als das silberärmere Protargol (8,3% Ag). Dies ist aber, nach den vorstehenden biologischen Versuchen zu schließen, nicht der Fall: Beide Präparate üben ungefähr die gleiche intensive Wirkung auf die Magendrüsensekretion aus. Mithin ist der Effekt nicht vom prozentualen Gehalt an Silber abhängig und somit auch nicht vom Quantum des bei beiden Körpern durch Einwirkung von HCl entstehenden Silberchlorids, AgCl. Gewisse, nicht aufgeklärte Eigentümlichkeiten in der individuellen organischen Beschaffenheit der Körper scheinen auch hier eine Rolle zu spielen.

In neuester Zeit sind auch einige Adstringentien der Ferri- und Ferroreihe auf ihre Einwirkung auf die Magensaftabscheidung untersucht worden und zwar von Feigl.[1]) Die Eisenverbindungen haben in der direkten Behandlung von Magenerkrankungen keine große Rolle gespielt; höchstens zur Behandlung der konsekutiven Anämie wandte man gewisse Eisenpräparate an. Bourget[2]) hat vor einigen Jahren eine Eisenbehandlung des Ulcus ventriculi empfohlen, die einerseits bezweckt, direkt blutstillend zu wirken, andererseits auch die Anämie zu bekämpfen. Die Methode besteht in Magenspülungen mit 2prozentigen Eisenchloridlösungen mit Zusatz von $^1/_2$ prozentigem Kali

1) Biochem. Zeitschr. 1908, Bd. 5, Heft 1.

2) Therap. Monatshefte, Juni 1900 und Krankh. des Magens und ihre Behandl. Wiesbaden 1906.

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chloricum, Zurücklassung von 50—60g dieser Lösung im Magen und nach 5 Minuten Fällung des Eisens durch Trinken einer 2 prozentigen Natrium bicarbonicum-Lösung. Darin liegt auch ein direktes, die Sekretion herabsetzendes Moment. Eine experimentelle Begründung dieser Methode lieferten die Untersuchungen von Feigl über den sekretorischen Einfluß des Eisenchlorids $FeCl_3$ (Ferrum sesquichloratum der Pharmakopöe). Lösungen von 0,5% haben zu dem Resultate geführt, daß „Ferrichloridlösungen bzw. darin als wesentliches Moment die Ferri-Ionen eine Verkürzung des Sekretionsvorganges auf mindestens die Hälfte der Zeit geben. Diese sezernierte Saftmenge hält sich durchschnittlich auf ein Drittel der Menge nach der Verabreichung von Wasser." — Im Gegensatz hierzu ist den Ferrosulfatlösungen ein wesentlicher, starker Einfluß auf die Drüsentätigkeit abzusprechen. Sie sollen nur in geringen Grenzen eine Hemmung verursachen und vor allem die Zeitdauer der Sekretion nicht verkürzen. Eine ähnliche, mehr indifferente Wirkung übt das kolloidale Ferrihydroxyd aus, während das Ferricitrat, welches Feigl, veranlaßt durch meine Versuche mit Natriumcitrat (s. ob.), prüfte, in analoger Weise, wie dieser Körper, nur in bedeutend geringerem Grade sich als sekretionstreibend erwies. Von größerer praktischer Bedeutung sind gleichartige experimentelle Untersuchungen desselben Autors mit Typen von Eisenwässern. Das Roncegnowasser, in welchem auf die Magendrüsen die Komplexwirkung des Eisens und des Arsentrioxyds (arsenige Säure) zur Geltung kommen, erwies sich als sekretionshemmend, was auf die Ferri-Ionen zurückzuführen ist, da das AsO_3 als solches erregend wirken solle. Umgekehrt steigert der ebenfalls Eisen enthaltende Schwalbacher Stahlbrunnen die Sekretion, und zwar wegen der in ihm enthaltenen freien CO_2, deren Pharmakodynamik auf die Drüsenfunktion in irritativem Sinne stärker ausgeprägt ist als diejenige depressiver Art des Eisens. Aus dieser Antagonistenwirkung eines Salzgemisches (resp. Mineralwassers) geht als Resultante, als Endeffekt die stärkere CO_2-Wirkung hervor. Auf rein experimentellem Wege gibt die obenerwähnte Arbeit von Rozenblat mit einem Gemische von Natrium bicarbonicum und Natriumchlorid lehrreichen Aufschluß.

III. Gewisse Metalle.

Der Frage nach der ev. Einwirkung metallischer Elemente auf die Magensekretion ist zum ersten Male am biologischen Tierexperimente durch meine Untersuchungen nähergetreten worden. Hierzu

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veranlaßte mich eine Publikation von G. Klemperer[1]). Dieser Autor ging von dem Grundgedanken aus, bei der Behandlung des Ulcus ventriculi und der Darmblutungen ein Ersatzpräparat für die unter Umständen, allerdings sehr selten, giftig wirkenden Wismutverbindungen (besonders Bismutum subnitricum) zu finden, welches im Magen- wie Darmsaft unlöslich, unresorbierbar und ungiftig sein solle, ferner die Schleimhäute in keiner Weise reizen dürfe und sich mit der Gewebslymphe bzw. dem Magenschleim zu einem festen Gerinnsel vereinigen müsse. Eine solche ideale Substanz glaubte Klemperer in feinst gepulvertem, metallischem Aluminium, der Aluminiumbronze, gefunden zu haben, welche, um eine Aufschwemmung in Wasser zu erleichtern, mit einer Glyzerinpaste verrieben ist. Das Präparat wird von den Chemischen Werken in Charlottenburg hergestellt in Pastillen zu 5 g, d. h. 2,5 g Aluminium pulverisatum + 1,5 g Glyzerin + 1,0 g Wasser und Escalin (Schorfbildner, von eschara = Schorf) genannt. Klemperer erblickt die Indikation zur therapeutischen Verwendbarkeit des Präparates an Stelle des Bismutum subnitricum nicht nur in den oben erwähnten Eigenschaften, sondern auch in den beiden Umständen noch, daß es einerseits die Fähigkeit habe, die Magenschleimhaut und besonders Defekte derselben (Ulzera) mit einer dichten, fest anhaftenden und damit mechanisch schützenden Decke zu überziehen, andererseits auch, und dies deutet der Autor allerdings nur indirekt an, daß es die Sekretion nicht erheblich alteriere. So sagt er: „Auf den Säuregehalt des Magensaftes hat das Escalin hier keine vermindernde Wirkung ausgeübt, wie denn auch die Beschwerden bei nervöser Hyperazidität des Magens durch das Mittel nicht beeinflußt wurden. Es schien mir beinahe, als ob man diese Tatsache differential-diagnostisch verwerten könnte."

In Anbetracht der Rolle, welche die Hyperazidität bei Ulcus ventriculi spielt, prüfte ich am Tierexperiment nach Pawlow das Escalin auf seine sekretorische Wirkung und zwar zunächst an vier verschiedenen Hunden mit gesunder, nicht entzündeter Schleimhaut in folgenden acht Versuchen:

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1) Ther. der Gegenwart, Mai 1907.

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Versuch Nr. 40 am Hund „Karo".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1 Tablette Escalin = 2,5 g metall. Aluminium	
9 h 24		11 h 03	
Zeit	Menge	Zeit	Menge
9 h 40	0,2	11 h 33	3,6
9 h 51	0,9	11 h 53	4,0
10 h 17	3,7	12 h 13	0,8
10 h 27	0,6	12 h 33	1,5
10 h 40	0,6	12 h 53	1,4
10 h 55	0,0	1 h 13	3,0
		1 h 33	1,6
		2 h 03	0,0
$1^1/_4$ Stunden Sekretionsdauer	6,0 ccm Gesamtquant. Gesamtazidität 110	3 Stunden Sekretionsdauer	15,9 ccm Gesamtquant. Gesamtazidität 108

Versuch Nr. 41 am Hund „Bernhardiner I".

200 ccm Leitungswasser		200 ccm Milch + 1 Tablette Escalin = 2,5 g metall. Aluminium	
9 h 26		11 h 30	
Zeit	Menge	Zeit	Menge
9 h 42	0,0	11 h 50	1,6
9 h 53	0,0	12 h 10	1,3
10 h 18	0,2	12 h 30	2,0
10 h 29	0,0	12 h 50	1,0
10 h 39	0,1	1 h 10	2,0
10 h 50	0,0	1 h 30	1,1
		1 h 50	0,6
		2 h 20	0,0
$^1/_2$ Stunde Sekretionsdauer	0,3 ccm Gesamtquant. Gesamtazidität ?	$2^1/_2$ Stunden Sekretionsdauer	9,6 ccm Gesamtquant. Gesamtazidität 110

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Versuch Nr. 42 am Hund „Bernhardiner II".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1 Tablette Escalin = 2,5 g metall. Aluminium	
9 h 30		11 h 15	
Zeit	Menge	Zeit	Menge
9 h 45	0,1	11 h 35	0,2
9 h 55	0,2	11 h 55	1,4
10 h 20	0,1	12 h 15	3,8
10 h 30	0,1	12 h 35	6,0
10 h 40	0,1	12 h 55	1,2
10 h 50	0,0	1 h 15	2,0
		1 h 35	1,0
		2 h 15	2,4
		2 h 45	2,3
		5 h —	0,5
		Die Sekretion dauert fort	
1 Stunde Sekretionsdauer	0,6 ccm Gesamtquant. Gesamtazidität ?	Über 5½ St. beobachtete Sekretionsdauer	20,8 ccm Gesamtquant. Gesamtzaidität 142

Versuch Nr. 43 am Hund „Moor".

200 ccm Leitungswasser		200 ccm Leitungswasser — 1 Tablette Escalin — 2,5 g metall. Aluminium	
9 h 33		11 h 22	
Zeit	Menge	Zeit	Menge
9 h 45	0,8	11 h 42	1,6
9 h 55	0,6	12 h 02	1,4
10 h 25	1,4	12 h 22	0,8
10 h 35	0,6	12 h 42	0,6
10 h 45	0,4	1 h 02	2,6
10 h 55	0,3	1 h 22	1,3
12 h 05	0,2	1 h 42	1,0
11 h 15	0,2	2 h 22	1,0
11 h 20	0,0	2 h 52	6,0
		5 h —	5,0
		Die Sekretion dauert fort	
1¾ Stunden Sekretionsdauer	4,5 ccm Gesamtquant. Gesamtazidität 124	Über 5 Stunden beobachtete Sekretionsdauer	21,3 ccm Gesamtquant. Gesamtazidität 132

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Versuch Nr. 44 am Hund „Karo".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1 Tablette Escalin = 2,5 g metall. Aluminium	
8 h 35		11 h 12	
Zeit	Menge	Zeit	Menge
8 h 50	2,4	11 h 45	9,0
9 h 05	1,6	12 h 15	2,5
9 h 20	1,0	12 h 45	3,0
9 h 35	0,4	1 h 15	1,8
10 h 05	1,0	1 h 45	1,2
10 h 35	1,4	2 h 15	6,1
10 h 05	0,0	2 h 45	2,4
		3 h 15	2,0
		3 h 45	1,5
		4 h 15	4,0
		4 h 45	4,5
		Die Sekretion dauert fort	
$1^1/_4$ Stunden Sekretionsdauer	7,8 ccm Gesamtquant. Gesamtazidität 130	Über 5 Stunden beobachtete Sekretionsdauer	38,0 ccm Gesamtquant. Gesamtazidität 135

Versuch Nr. 45 am Hund „Bernhardiner I".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1 Tablette Escalin = 2,5 g metall. Aluminium	
8 h 40		10 h 32	
Zeit	Menge	Zeit	Menge
8 h 55	0,0	11 h 02	2,4
9 h 10	0,0	11 h 32	3,4
9 h 25	0,0	12 h 02	3,6
9 h 30	0,1	12 h 32	1,6
10 h 10	0,0	1 h 02	3,0
		1 h 32	3,0
		2 h 02	2,0
		2 h 32	5,0
		3 h 02	2,5
		3 h 45	2,0
		4 h 02	1,8
		4 h 32	1,5
		Die Sekretion dauert fort	
1 Stunde Sekretionsdauer	0,1 ccm Gesamtquant. Gesamtazidität ?	Über $5^1/_2$ St. beobachtete Sekretionsdauer	31,8 ccm Gesamtquant. Gesamtazidität 104

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Versuch Nr. 46 am Hund „Bernhardiner II".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1 Tablette Escalin = 2,5 g metall. Aluminium	
8 h 45		11 h 15	
Zeit	Menge	Zeit	Menge
9 h —	0,0	11 h 45	5,0
9 h 15	0,6	12 h 15	10,5
9 h 30	0,8	12 h 45	8,5
9 h 45	0,1	1 h 15	8,3
10 h 15	0,4	1 h 41	6,1
10 h 45	1,0	2 h 15	6,5
10 h 55	0,3	2 h 45	5,3
11 h 05	0,0	3 h 15	4,0
		3 h 45	3,5
		4 h 15	6,0
		4 h 45	4,0
		Die Sekretion dauert fort	
2 Stunden Sekretionsdauer	3,2 ccm Gesamtquant. Gesamtazidität 130	Über 5 Stunden beobachtete Sekretionsdauer	67,7 ccm Gesamtquant. Gesamtazidität 116

Versuch Nr. 47 am Hund „Moor".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1 Tablette Escalin = 2,5 g metall. Aluminium	
8 h 50		10 h 30	
Zeit	Menge	Zeit	Menge
9 h 05	0,1	11 h —	1,0
9 h 20	0,0	11 h 30	2,0
9 h 55	0,1	12 h —	2,2
9 h 50	0,0	12 h 30	1,5
		1 h —	2,0
		1 h 30	2,5
		2 h —	2,5
		2 h 30	3,0
		3 h —	2,0
		3 h 30	1,0
		4 h —	2,1
		4 h 30	1,8
		Die Sekretion dauert fort	
$^3/_4$ Stunden Sekretionsdauer	0,2 ccm Gesamtquant. Gesamtazidität ?	Über $5^1/_2$ St. Sekretionsdauer	23,6 ccm Gesamtquant. Gesamtazidität 118

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Diese Versuche geben ein unerwartetes, verblüffendes Resultat: durchweg eine sehr bedeutende, teilweise sogar eine stürmische Steigerung der Sekretionsdauer und der Gesamtsekretion. Die prozentuale Azidität war bald erhöht, bald verringert, immerhin nicht typisch beeinflußt. Dafür, daß nicht die Glyzerinkomponente des Escalins die Steigerung hervorruft, spricht wohl der Versuch, dessen Resultat auch beiläufig Kast[1]) bestätigt.

Versuch Nr. 48 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1,5 g Glyzerin = Gehalt einer Escalinpastile	
7 h 45		12 h 15	
Zeit	Menge	Zeit	Menge
8 h 15	0,4	12 h 45	0,3
8 h 45	0,0	1 h 15	0,1
9 h —	0,0	1 h 45	0,0
9 h 15	0,0		
½ Stunde Sekretionsdauer	0,4 ccm Gesamtquant. Gesamtazidität ?	½ Stunde Sekretionsdauer	0,4 ccm Gesamtquant. Gesamtazidität ?

Diese erhebliche Sekretionssteigerungsfähigkeit des Escalins wurde von Bickel[2]) auch am Menschenexperiment festgestellt, und dieser Autor äußert sich darüber folgendermaßen; „Alle Metalle, welche bei Gegenwart verdünnter Salzsäure Wasserstoff entwickeln, wie z. B. Eisen, Mangan, Aluminium usw. wirken reizend auf die Magendrüsen und rufen eine lebhafte Sekretion hervor. Metalle, die in dem obengenannten Sinne keine Wasserstoffentwickler sind, wie z. B. metallisches Wismut, Silber, Gold, verhalten sich gegen die Magenschleimhaut indifferent.

Escalin, eine Aluminiumglyzerinpaste, zersetzt sich im menschlichen Magensafte unter Wasserstoffentwicklung und ruft eine sehr starke Saftsekretion hervor. Rodari hat letzteres zuerst an Magenblindsackhunden entdeckt; Bickel bestätigte es an einem von Gluck operierten ösophagotomierten Magenfistelmenschen. Das Escalin ist demnach wegen seiner stark reizenden und safttreibenden Eigenschaften beim Ulcus ventriculi nicht indiziert. Auch eine spezifische blutstillende Wirkung ließ sich im Tierversuche nicht nachweisen."

1) Arch. f. Verd.-Krankh. 1906, Bd. 12, Heft 6.
2) Berl. Klin. Wochenschr. 1907, Nr. 30 und 33.

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Über das mit Escalin angestellte Experimentum ad hominem äußerte sich neulich Bickel[1]) folgendermaßen:

1. Versuch (Bickel). 2. Versuch (Bickel).

150 ccm Wasser	150 ccm Wasser —3 Escalintabl.	150 ccm Wasser	150 ccm Wasser —3 Escalintabl.
Sekretionsmenge		Sekretionsmenge	
9,2	12,5	3,7	19,4
9,1	8,0	2,0	2,6
2,15	7,6	9,0	0,9
1,0	3,6	3,2	3,1
1,75	5,8	8,8	14,0
1,8	4,5	8,1	10,2
3,4	5,5	10,8	7,8
1,1	8,8	5,6	0,8
1,1	10,4	1,4	5,4
0,15	9,9	0,2	14,4
0,15		0,0	4,2
— —		0,0	7,6
	Die Sekretion dauert fort		Die Sekretion dauert fort
Sa. 30,9 ccm	76,6 ccm	52,8 ccm	90,4 ccm
Sekretionsmenge		Sekretionsmenge	

Klemperer hat nun gegen dieses abfällige Urteil Bickels in der Publikation von Mai[2]) energisch Stellung genommen. Mai stellt einen sekretionssteigernden Einfluß des Präparates entschieden in Abrede, indem er die Wirkung des Escalin bei 20 Patienten mit normalen, subaziden und hyperaziden Zuständen des Magens prüft, wobei er allerdings nicht unterscheidet, ob es sich um Gastritiden oder Neurosen des Magens handelt. Wie verhält es sich nun bei solchen diametralen Gegensätzen mit der endgültigen Antwort nach der klinischen Brauchbarkeit des Escalins? Diese geben zum Teil meine folgenden zwei Versuche an einem Hunde mit chronischer Gastritis (ohne Aziditätsanomalie).

1) Berl. Klin. Wochenschr. 1907, No. 48.
2) Therap. der Gegenwart, November 1907.

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Versuch Nr. 49 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser + 1 Escalintablette = 2,5 metall. Aluminium	
8 h —		1 h 30	
Zeit	Menge	Zeit	Menge
8 h 30	1,2	2 h —	0,1
9 h —	0,8	2 h 30	0,1
9 h 30	0,7	3 h —	0.2
10 h —	0,2		
10 h 15	0,0		
$1^3/_4$ Stunden Sekretionsdauer	2,9 ccm Gesamtquant. Gesamtazidität 119	$1^1/_2$ Stunden Sekretionsdauer	0,4 ccm Gesamtquant. Gesamtazidität ?

Versuch Nr. 50 am Hund „Max".

200 ccm Leitungswasser		200 ccm Leitungswasser — 1 Escalintablette — 2,5 g metall. Aluminium	
8 h 30		12 h —	
Zeit	Menge	Zeit	Menge
9 h —	1,8	12 h 30	0,7
9 h 30	1,0	1 h —	0,4
10 h —	0,6	1 h 30	0,2
10 h 30	0,4	2 h —	0,0
11 h —	0,2	2 h 30	0,0
11 h 30	0,0		
$2^1/_2$ Stunden Sekretionsdauer	4,0 ccm Gesamtquant. Gesamtazidität 124	$1^1/_2$ Stunden Sekretionsdauer	1,3 ccm Gesamtquant. Gesamtazidität appr. 120

Diese Experimente sind von einer prinzipiellen Bedeutung, erstens, weil sie im allgemeinen einen weiteren Beitrag liefern für die von mir festgestellte, andersartige, gewissermaßen spezifische Reaktion der entzündeten Magenschleimhaut gegenüber derjenigen der intakten Mukosa auf gewisse Körper; zweitens, weil sie im speziellen den Beweis liefern, daß auch das Escalin, bzw. das metallische Aluminium, in seinem Effekte auf die Magendrüsensekretion ein Analogon zu diesen Körpern bildet.

Inwieweit also das Präparat Klemperers, nach diesen Versuchen wenigstens zu schließen, zur Behandlung von Magenerkrankungen an-

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gezeigt ist, soll noch in den epikritischen Schlußbemerkungen zu dieser Arbeit näher erörtert werden. An dieser Stelle erübrigt es, noch auf die Art der Pharmakodynamik der Escalineinwirkung auf die HCl-Sekretion des Magens näher einzugehen.

Zunächst ist es feststehend, daß die Sekretionssteigerung bei nicht entzündlicher Schleimhaut eine spezifische Wirkung des metallischen Aluminiums ist und nicht etwa auf die Glyzerinkomponente zurückgeführt werden kann (vergl. Versuch Nr. 48). Feigl hat meine Experimente wiederholt, und zwar erstrecken sich seine Versuche auf zwei Hunde, deren Magenschleimhaut, den Resultaten nach zu schließen, intakt war. So kommt Feigl zu dem prinzipiell gleichen Resultate wie ich in den ersten acht Escalinversuchen.

Auf welche Faktoren soll nun die Sekretionssteigerung zurückgeführt werden? Da das Aluminium in feingepulvertem Zustande sich selbst in sehr wenig konzentrierter HCl leicht löst, und dabei diese Säure verzehrt, sollte man theoretisch erwarten, daß es schon dadurch die Sekretion in herabsetzendem Sinne hemmen würde. Außerdem könnte von der dabei entstehenden Chloridverbindung eine adstringierende und damit auch sekretionshemmende Einwirkung erwartet werden. Ist doch der chemische Vorgang folgender:

$$Al + 3HCl = AlCl_3 + 4H.$$

Die experimentellen Versuche (meine ersten acht Versuche und diejenigen Feigls) ergeben aber gerade das umgekehrte Verhalten: keine Hemmung, sondern eine ganz erhebliche Steigerung. Bickel und Feigl erklären dieses Phänomen durch die Einwirkung des freiwerdenden Wasserstoffes auf die Magendrüsen, bzw. deren Nerven und suchen eine Erklärung hierfür in dem analogen Verhalten der Kohlensäure, als eines Sekretionssteigerers par excellence (am Tierexperimente durch Weidert und Pincussohn nachgewiesen, siehe oben). — Warum ist nach diesen Autoren gerade der Wasserstoff das sekretionstreibende Agens? Erstens, weil der im Magen durch die Salzsäure nicht chemisch beeinflußte Überschuß von unlöslichem metallischen Aluminium, der neben dem versetzten Aluminium vorhanden ist, als ungelöster Körper chemisch nicht wirken („corpora non agunt nisi soluta"), ebensowenig rein mechanisch eine Wirkung nach analogen, auf Fremdkörper im Magen bezüglichen Untersuchungen von Pawlow und Bickel (Talk) ausüben könne. Zweitens, weil das sehr leicht lösliche und zerfließliche Aluminiumchlorid $AlCl_3$ rasch aus dem Magen weggeschwemmt werde, so daß es keine erhebliche Wirkung entfalten könne. Mithin wäre dies nur in einer Wasserstoffentwicklung, ähnlich der Kohlensäureentstehung und ihrer entsprechenden Wirkung zu suchen.

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Wie können wir diese Theorie mit den letzten Versuchen am Gastritis-Hunde in Einklang bringen, wo das Präparat erheblich sekretionshemmend wirkt? Soll in dem einen Falle (gesunde Schleimhaut) der Wasserstoff auf die Drüsenarbeit irritativ, in dem anderen Falle (Entzündung der Mukosa) depressiv wirken? Dies wäre zwar möglich, obwohl sonstige Beobachtungen über derartig gegensätzliche Wasserstoffwirkungen auf den Magen fehlen. Deshalb läßt es sich auch denken, und dies scheint mir persönlich die wahrscheinlichere Deutung zu sein, daß in der chemischen Umsetzung $Al + 3HCl = AlCl_3 + 3H$ die Chloridverbindung das wirksame Agens wäre. Um so leichter ist diese Theorie erklärlich, wenn wir uns erinnern, daß wir in $AlCl_3$ eine Verbindung besitzen, die trotz des leichten spezifischen Gewichtes des Aluminiums in ihrer lokalen Wirkung sich gleich wie die entsprechenden Verbindungen von schweren Metallen verhält (Schmiedeberg). Wir haben somit ein kräftiges Adstringens vor uns, das auf die kranke (entzündete) Magenschleimhaut so wirkt, wie das Silberchlorid AgCl, welches bei der Einwirkung der HCl des Magens auf anorganische und organische Silberverbinbindungen (Argent. nitricum, Protargol, Albargin, s. oben) entsteht. Nach meiner oben besprochenen Theorie einer größeren Affinität der Adstringentien zu den chemischen Bestandteilen einer entzündeten Schleimhaut behufs Bildung des diffusen, adstringierenden Vorganges und damit auch der Adstringierung, resp. Hemmung der Sekretion gegenüber der nicht entzündeten Schleimhaut, ist die Annahme naheliegend, daß hier die Sekretionshemmung auf die Einwirkung des Aluminiumchlorids und nicht des Wasserstoffes zurückzuführen ist. Ebenso dürfte die Steigerung bei gesunden, anatomisch intakten Verhältnissen der Magenschleimhaut, nach den oben angeführten Versuchen zu schließen, auf der Wirkung der gleichen Verbindung beruhen, nur mit dem Unterschiede eben, daß es hier nicht zu einer Adstringierung in chemisch-physikalischem Sinne, sondern zu einer mehr funktionellen, allerdings noch nicht näher aufgeklärten Irritation der Magendrüsen resp. ihrer Nerven kommt. Diese Theorie scheint mir die Doppelwirkung des Aluminiums bzw. des Escalins besser zu erklären, als die Wasserstofftheorie von Bickel und Feigl.

Feigl hat, veranlaßt durch meine Escalin- bzw. Aluminiumversuche, weitere reine, metallische Elemente geprüft: Beim Mangan fand er eine dem Aluminium analoge lebhafte Steigerung der Sekretion, bei Zinn eine minimale Anregung, beim Wismut eine völlige Indifferenz, ebenso beim Silber und Gold. In bezug auf das Eisen hat Feigl schon vor meinen Aluminiumuntersuchungen eine

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erhebliche Sekretionssteigerung dieses Metalles konstatiert. — Seine Untersuchungen erstrecken sich teilweise auf von mir operierte und experimentell benutzte Hunde, teilweise auf andere Tiere, bei denen es sich jedenfalls um normale, nicht entzündliche Magenschleimhaut gehandelt hat.

Zum Abschluß meiner eigenen experimentellen Untersuchungen möchte ich noch einen Umstand erwähnen, der sich wie ein roter Faden durch die sämtlichen Versuche zieht und den ich jeweilen bei jedem Experimente angedeutet habe: Es handelt sich um die relative Konstanz des prozentualen Salzsäuregehaltes des Magensaftes gegenüber der auch durch Medikamente bedingten Variabilität der Magensaft- resp. Salzsäure**menge.**

Daß es sich um eine Beeinflussung dieser letzteren als solcher, und nicht etwa beispielsweise um eine Steigerung durch Transsudation von Wasser aus der Magenwand in das Innere des Magens handelt, beweist der Umstand, daß die Gesamtazidität bei der Steigerung der Quantität nicht herabgesetzt wird. Schon Pawlow hat betont, daß es ihm so gut wie nie gelungen sei, die prozentuale Salzsäurereaktion typisch in einer Weise zu verändern, daß dabei nicht physiologische Schwankungen diese unerheblichen Konzentrationsänderungen erklären könnten, und es ist das Verdienst Bickels, durch viele Experimente diese Andeutungen auch für die klinisch wichtigen Anschauungen und Erkenntnisse erweitert zu haben. Auch betont dieser Autor die Konstanz der Magensaftkonzentration gegenüber der Schwankung in der Magensaftmenge. — Nachuntersuchungen der Ergebnisse der Tierexperimente und der oben erwähnten ösophagotomierten Magenfistel-Patientin von Bickel durch Homberg, Röder, Sommerfeld und Rubow haben ergeben, daß der reine unverdünnte Magensaft des Menschen ungefähr identisch ist mit demjenigen des Hundes, indem seine Konzentration zwischen 0,4—0,5% schwanken soll, innerhalb dieser Grenzen aber auch konstant bleibt. Diese übereinstimmenden Beobachtungen, welche einen neuen Beweis für die Übertragbarkeit der Resultate der Tierexperimente auf den Menschen liefern, veranlaßten Bickel[1]), dem klinisch bisher gut fundierten Bilde der Hyperazidität (Hyperchlorhydrie) als einer qualitativen Sekretionssteigerung die physiologische Basis zu nehmen, indem er nachwies, daß bei gleicher sekretorischer, aber ungleicher motorischer Leistung des Magens der Mageninhalt verschieden sauer reagieren kann. Je nachdem nämlich das Probe-

1) Bioch. Zeitschr., Bd. 1, Heft 1 und 2, 1906. Deutsche med. Wochenschr. No. 30, 1907. Zeitschr. f. physik. und diätet. Therapie, Bd. 11, 1907/08.

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frühstück in den Darm befördert worden ist, bekommt man beim Aushebern mehr oder weniger reinen Magensaft, der je reiner um so saurer reagiert, als wenn er mit dem Probefrühstück stark verdünnt ist. So kommt Bickel zum Resultate, daß die Motilitätszustände (normale Motilität, Hypermotilität und Submotilität resp. Atonie) die Konzentration des am Menschen durch das Probefrühstück entnommenen Magensaftes in physiologischer Weise beeinflussen. Auf weitere wichtige Einzelheiten dieser sehr interessanten Arbeit kann ich hier nicht näher eingehen. Ich möchte nur noch darauf hinweisen, daß auch meine Tierexperimente dafür sprechen, daß Hyperchlorhydrie und Hypersekretion im Grunde genommen dasselbe sind, d. h. daß die Hyperchlorhydrie eine durch besondere Motilitätsverhältnisse beeinflußte Hypersekretion ist. — Diese Autoren erwähnen auch bei einzelnen Versuchen einen gewissen Parallelismus zwischen der Magensaft- bzw. Säuresekretion und der Verdauungskraft des Pepsins. Ich selbst habe diese Fermentkraft nicht bei allen Versuchen geprüft, sondern nur hier und da bei einzelnen Körpern nach dem Mettschen Verfahren Stichproben angestellt, wobei ich den Eindruck bekommen habe, daß die Hemmung der Sekretion in allerdings unerheblicher Weise auch die peptische Kraft herabsetzt, während diese bei der Steigerung der Magensaftabscheidung gegenüber der Verdauungskraft der Wasserperiode keine wesentlichen Unterschiede aufweist.

Im Interesse der Vollständigkeit dieser Arbeit kommen noch zwei Gruppen von die Magensaftabscheidung beeinflussenden Substanzen zur kurzen Erörterung.

IV. Gewisse Gifte der Alkaloidreihe.

Ich gehe auf diese Verhältnisse hier nur summarisch ein, einerseits weil ich an der Hand der wichtigeren klinischen Literatur schon in meinem Buche ihre therapeutische Bedeutung und Indikation besprochen habe, andererseits weil ich mich in dieser Arbeit mit der tierexperimentellen Untersuchung dieser Gruppe nicht befaßt habe und somit darin auch nichts Neues zur Kenntnis ihrer Wirkung auf die Magensekretion beitragen kann.

Die hier in Betracht kommenden Vertreter der Alkaloidreihe stehen in der Art ihrer Pharmakodynamik in striktem Gegensatze zu den chemischen Körpern der bisher besprochenen Gruppen. Diese setzen ihre Wirkung auf die Magendrüsenfunktion lokal im Magen selbst an, die Derivate der Alkaloidreihe wirken in erster Linie auf das Nerven-

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system und speziell auf das zentrale Nervensystem, auf dem Blutwege können sie auch die Magensaftsekretion beeinflussen. Die Reaktion der Magendrüsen ist hier meistens nur eine Teilerscheinung, eine Mitbeteiligung an der Reaktion anderer Drüsen.

Die betreffenden Alkaloide spielen gegenüber den obenerwähnten ersten zwei Gruppen als Mittel zur Regulierung der Sekretion, resp. Bekämpfung der Sekretionsanomalien eine sehr kleine Rolle, einmal weil sie heftige Nervengifte sind, sodann auch weil sie keine spezifische, nur den Magendarmkanal betreffende Wirkung ausüben. Sie kommen nur als Adjuvantien in Verbindung mit den spezifisch wirkenden Alkalien und Adstringentien zur therapeutischen Anwendung und sind dabei nicht unentbehrlich.

Folgende sind sekretionssteigernde Alkaloidgifte: In erster Linie das Morphin. Die alte Lehre über dessen Sekretionswirkung auf die Magendrüsen lautete bis zu Riegels[1]) Untersuchungen gerade umgekehrt. Das Morphin sollte eine typische Sekretionshemmung bewirken. Riegel hat nun sowohl an Pawlowschen Hunden, wie an klinischen Versuchen den strikten Nachweis erbracht, daß das Morphin bei subkutaner, wie rektaler Anwendung zunächst allerdings die Sekretion hemmt, ungefähr während der ersten $^3/_4$—1 Stunde, daß dann aber eine Steigerung der Sekretionsmenge nach und nach sich einstellt, welche diejenige der nicht morphinisierten Versuchsobjekte weit überschreitet. v. Aldor[2]) hat diese Versuche am Menschen wiederholt und bestätigt und ebenso Hirsch[3]), der am Hundeexperimente nach der anfänglichen Hemmung, mit welcher zugleich ein Spasmus des Pylorus besteht, eine geradezu enorme Saftsekretion beobachtet hat, die seiner Ansicht nach zentralen Ursprunges ist. Kürzlich hat Pewsner[4]) auch das salzsaure Äthylmorphin, das Dionin experimentell geprüft mit dem Resultat, daß der Steigerung hier keine Hemmung vorausgehe, sondern daß erstere sofort eintrete. Nach dem gleichen Autor hat das Physostigmin eine dem Morphin analoge Wirkung: auch zunächst eine Hemmung, dann eine Steigerung, während das Pilokarpin, wie das Dionin nur in intensiverer Art, eine Sekretionssteigerung ohne vorausgegangene Herabsetzung bewirkt. Die Sekretionsanregung dieses Präparates hat ebenfalls zuerst Riegel an Tierexperimenten und an klinischen Versuchen nachgewiesen. Alle diese Mittel beeinflussen nur die Quantität, niemals aber die Qualität des Magensaftes, dessen Konzentration ziemlich konstant bleibt.

1) Zeitschr. f. klin. Med. 1899, Bd. 27 und ebenda 1900 Bd. 40.
2) Ebenda.
3) Zentralbl. f. innere Med. 1900, Nr. 2.
4) Biochem. Zeitschr. 1907, Bd. 2, Heft 4—6.

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Als sekretionsherabsetzendes Medikament hat das Atropin eine gewisse therapeutische Bedeutung, besonders in Gestalt des Extractum belladonnae. Es ist wiederum das Verdienst Riegels, experimentell und klinisch die hochgradige Fähigkeit des Atropins, die Sekretionsmenge herabzusetzen, untersucht zu haben. Am Probefrühstück konstatierte Riegel auch eine qualitative Reduktion des HCl-Gehaltes. Nach den oben angeführten Ergebnissen Bickels über Identität von Hypersekretion und Hyperchlorhydrie sind bei diesen Befunden Riegels Fehlerquellen nicht ausgeschlossen. Die neuesten Untersuchungen Pewsners am Hunde betonen die Konstanz des HCl-Gehaltes auch bei der Herabsetzung des Quantums durch Atropin. Nach diesem Autor hat das Euphthalmin eine dem Atropin analoge saftherabsetzende Wirkung.

Die gleiche Eigenschaft wird von Pickardt[1]) dem Skopolamin (Scopolaminum hydrobromicum) zugeschrieben. Dieser Autor hat das intensiv wirkende Präparat in mehreren Fällen in Dosen von 0,0003 zweimal täglich bis zu 4 Wochen lang ohne jede Nebenwirkung bei Gastrosukorrhœ mit sehr gutem Erfolge angewendet.

V. Die Stomachika.

Trotz des auf eine spezifische Einwirkung auf den Magen hindeutenden Namens ist gerade die Pharmakodynamik dieser Mittel gegenüber derjenigen der vier vorstehenden Gruppen am wenigsten ausgeprägt, bzw. untersucht. Die Stomachika haben für die sekretorische Funktion des Magens eine relativ geringe Bedeutung, und ich darf diese hier wohl nur insoweit kurz besprechen, als dies zu den Ausführungen in meinem Buche eine Ergänzung bedeutet.

Zunächst kommen hier die Amara in Frage. Die durch Buchheim[2]) und Schrenk[3]) begründete und vielfach vertretene Ansicht, daß die Amara infolge ihres Tanningehaltes die Magensaftsekretion hemmen, ist durch neuere Untersuchungen an Pawlowschen Hunden als unhaltbar erwiesen worden. So fand Borissow[4]), daß nach ihrer Aufnahme eine Sekretionssteigerung im Magen auftritt, die aber nicht durch lokale Drüsenreizung im Magen zu erklären sei, sondern nur auf reflektorischem Wege (nach Art des Appetitsaftes) durch Reizung der Geschmacksnerven zustande kommt. Diesen Standpunkt nimmt

1) Therap. der Gegenwart, Juni 1903.
2) Beiträge zur Arzneimittellehre. Leipzig 1849.
3) Inaug.-Dissertation Dorpat 1849.
4) Arch. f. exp. Path. und Pharmak., 1904, Bd. 51, Heft 4 u. 6.

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auch Straschesko[1]) ein. Einen Schritt weiter geht Hoppe[2]), der diese Steigerung nicht nur als einen Reflex von den Geschmacks- und Riechnerven ausgehend auffaßt, sondern der daneben auch einen reflektorisch-chemischen Einfluß von der Magen- und Darmschleimhaut mitwirken läßt. Nach Hoppe entfalten aber alle Bittermittel ihre sekretionssteigernde Wirkung nicht an sich, sondern nur wenn nach ihrer Einnahme dem Tiere Nahrung beigebracht wird. Große Mengen der Amara hingegen sollen aber eine Hemmung der Sekretion bedingen.

Die Salzsäure hat als Stomachikum in den letzten Jahren an ihrem früheren Ansehen viel eingebüßt, seitdem die Untersuchungen von Heichelheim und Kramer, ferner Riegels, v. Noordens und Honigmanns ihr jeden Charakter als Magensafttreiber abgesprochen haben. (Sie ist ein vorzüglicher Treiber der Pankreassekretion.) Nur Bickel[3]) und Heinsheimer[4]) schreiben ihr, ähnlich Hoppe den Amaris, eine sekretionserregende Wirkung zu, wenn nach ihrer Einnahme Nahrung verabreicht wird, und zwar besteht diese Sekretionserregung nur beim Hunde mit einer chronischen subaziden Gastritis. Die HCl befähigt nach diesem Autor also nur die kranke Magenschleimhaut zu erhöhter sekretorischer Arbeit, während die gesunde darauf nicht reagiert.

Das Orexinum tannicum soll nach Hoppe auch nur bei Gastritis eine leichte Sekretionssteigerung bedingen, während bei gesunder Magenschleimhaut die Sekretion in keiner Weise alteriert wird. Es ist dies eine Bestätigung analoger Beobachtungen am Menschen von Glücksziegel[5]), Mathes[6]) und Kornfeld[7]).

Das neueste Stomachikum, das Acidol (Betainchlorhydrat = Chlorhydrat von Trimethylaminoessigsäure) ist von Heinsheimer[8]) auf seine sekretorische Wirkung an Tierexperimenten geprüft worden. Das Präparat zerfällt in wäßriger Lösung in HCl + Betainsäure. Es enthält nach Flatow 23,78 % HCl. Die Betainkomponente ist nach diesem Autor für die Magensaftsekretion völlig indifferent. Die frei werdende HCl-Komponente soll, insoweit unmittelbar nach Einnahme des Präparates Nahrung genommen wird, die Saftsekretion zu Anfang

1) Wratsch 1905, Nr. 2.
2) Berl. Klin. Wochenschr. 1905, Nr. 33, u. Inaug.-Dissert. Berlin 1906.
3) Berl. Klin. Wochenschr. 1905, Nr. 28.
4) Arch. f. Verdauungskrankh. 1904, Bd. 12, Heft 2.
5) Prager med. Wochenschr. 1890, Nr. 13, nach Hoppe zitiert.
6) Münchner med. Wochenschr. 1891, nach Hoppe zitiert.
7) Wiener klin. Wochenschr. 1891, nach Hoppe zitiert.
8) Arch. f. Verdauungskrankh. 1906, Bd. 12, Heft 2, nach Hoppe zitiert.

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der Verdauungsperiode leicht steigern; diese Reizung soll aber bald abklingen. Der therapeutische Wert dieses Präparates dürfte also, nach diesen Tierversuchen zu schließen, nicht hoch anzuschlagen sein.

Epikrise.

Welche Schlüsse für die praktische Therapie können wir aus den vorstehenden Erörterungen und Untersuchungen ziehen?

Die Konsequenzen aus Untersuchungen am Tierexperimente gezogen, insofern ihnen ein gewisser therapeutischer Wert inneliegen soll, müssen auf den kranken Menschen übertragen werden können. Daß eine solche Übertragung zulässig ist, haben die ausgedehnten Experimenta ad hominem von Bickel, Heinsheimer u. a. nicht nur wahrscheinlich gemacht, sondern auch bewiesen. Ganz besonders spricht der Bickelsche Versuch am ösophagotomierten Magenfistelmädchen (mit gesunder Magenschleimhaut) mit dem zuerst von mir untersuchten Escalin für die Analogie zwischen der Reaktion des Hunde- und des Menschenmagens und damit auch für die Zulässigkeit von Schlüssen therapeutisch-klinischer Natur aus dem Tierexperiment auf den Menschen.

Von einer prinzipiellen Bedeutung aus meinen Untersuchungen ist in erster Linie der durch die Prüfung verschiedenartiger Medikamente eruierte Umstand, daß die gesunde und die kranke Magenschleimhaut durchaus nicht in gleicher Weise auf den gleichen Körper sekretorisch reagieren. Dies ist hier für die in der Magentherapie wichtigsten Adstringentien nachgewiesen, so für gewisse Bismutverbindungen, wie z. B. in ausgesprochener Weise für das Bismutum bitannicum und weniger intensiv für das Bismutum subnitricum. Damit ist aber nicht gesagt, daß die gesunde und kranke Mukosa auf jeden Körper der betreffenden Reihe einander gegensätzlich reagieren. Maßgebend für die Art der Sekretion ist die Einwirkung einer der durch die HCl des Magens frei werdenden Komponente. Beispielsweise wirkt die Salizylsäurekomponente am Bismutum bisalicylicum unter allen Umständen sekretionsbefördernd, sowohl auf die gesunde wie auf die kranke Magenschleimhaut. — In typischer Weise haben wir die einander entgegengesetzt verlaufenden Sekretionzustände beim gesunden und beim entzündeten Magen, bei den organischen Derivaten des Argentum nitricum, beim Protargol und Albargin. Noch auffallender sind diese Verhältnisse beim Escalin ausgeprägt.

Welche Schlüsse ergeben sich daraus für die medikamentöse Therapie einzelner Magenerkrankungen?

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Zunächst die Tatsache, daß der diagnostische Unterschied zwischen Magenneurosen als einer Erkrankung mit völlig intakter Schleimhaut und Gastritis, möge sie primärer oder sekundärer Natur sein, möge sie für sich allein bestehen oder eine Begleiterscheinung eines andern Magenleidens sein (z. B. Ulkus), auch in therapeutischer Hinsicht, soweit dabei die Bekämpfung einer Sekretionsanomalie in Frage kommt, zu Recht besteht. Es ist also nicht gleichgültig, kurzweg von „subaziden oder superaziden Zuständen" des Magens zu reden und je nachdem neben der diätetischen Behandlung ein Medikament zu verabreichen. Die Frage, welches Medikament im individuellen Falle in Betracht kommt, entscheidet einzig die Untersuchung am Patienten, nicht seine subjektiven, wenn manchmal auch charakteristisch scheinenden Angaben, sondern die physikalische und chemische Prüfung des Magenchemismus, des Mageninhaltes. Die einfachsten und zuverlässigsten Mittel unserer Diagnostik hierfür sind: die Spülung des nüchternen Magens und die Prüfung des Spülwassers auf das Vorhandensein oder Fehlen eines wesentlichen Schleimgehaltes und das altbewährte Probefrühstück nach Ewald. Dieses werde zunächst untersucht auf das Vorhandensein oder Fehlen der die Entzündung charakterisierenden Merkmale (Schleimverhältnisse usw.), sodann auf die Aziditätsverhältnisse. Sind diese abnorm, so unterscheidet man in der Grundlage dieser Abnormität, ob ein entzündlicher oder ein rein nervöser Zustand vorliegt. Die anatomische Beschaffenheit der Magenschleimhaut entscheidet dann in erster Linie, welches unter den von mir untersuchten Medikamenten zur Anwendung kommen soll. So würde sich beispielsweise die Indikation ergeben, eine Gastritis hyperacida mit Protargol oder Albargin zu behandeln, während bei einer nervösen Hypersekretion diese Präparate strikte kontraindiziert erscheinen. Umgekehrt erscheinen diese Präparate als indiziert bei der Behandlung einer nervösen Subazidität, weil sie auf diesem Boden mächtig sekretionsanregend wirken, während ihre Anwendung bei der Gastritis subacida therapeutisch unlogisch, geradezu paradox sein würde.

Das Bismutum subnitricum hat im Gegensatze zu den erwähnten Körpern einen relativ indifferenten Einfluß auf die Sekretion des anatomisch intakten Magens; hier und da sieht man in den Versuchen die Tendenz zur Saftherabsetzung, eine solche ist aber ausgesprochener bei der entzündeten Magenschleimhaut. Das Anwendungsgebiet des Präparates ist also hier ein größeres in dem Sinne, weil es sowohl bei entzündlichen, wie nicht entzündlichen Hyperaziditätszuständen gegeben werden kann. Die vorliegenden Versuche mit dem Präparate haben die von mir anderen Ortes geäußerte klinische

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Beobachtung, daß das Bismutum subnitricum eine säureherabsetzende Eigenschaft besitze, bestätigt, während die gleiche Ansicht über das Argentum nitricum (bei Hyperaziditäten überhaupt) nach Versuchen mit seinen Derivaten einer Modifizierung bedarf.

Der Umstand, daß die Adstringentien in ihrer Wirkung auf die Magensaftabscheidung bisher kaum experimentell und überhaupt nicht vergleichsweise bei gesunder und entzündeter Magenschleimhaut geprüft worden sind, erklärt auch die über ihren sekretorischen Einfluß in der Literatur einander widersprechenden Angaben. Die Lösung dieser Widersprüche liegt nun in der Erkenntnis der verschiedenartigen Reaktionsfähigkeit der Magenschleimhaut gegenüber gewissen Adstringentien, je nach der anatomischen Beschaffenheit der erstern.

Besonderes Interesse geben die praktischen Ergebnisse dieser Untersuchungen für die Frage der Therapie bei Ulcus ventriculi. Es ist ja eine wesentliche Aufgabe der Therapie, die häufig bestehende Hypersekretion zu bekämpfen, überhaupt die Sekretion, welche die Heilung des Ulkus durch chemische Irritation verzögert, herabzusetzen. Eines alten Rufes für die Behandlung des Ulcus ventriculi erfreut sich das Bismutum subnitricum. Die meisten Autoren schreiben ihm nur eine physikalisch-mechanische Wirkung als Deckmittel des Substanzverlustes zu. Meine Experimente haben erwiesen, daß dem Präparate auch eine chemische sekretionsherabsetzende Wirkung innewohnt, die bei der nicht entzündeten Schleimhaut zwar gering, bei der entzündlich veränderten aber intensiver ist.

Die beiden Lokalanästhetika, welche bei der symptomatischen Ulkustherapie sich gut bewährt haben, und gewöhnlich als Zusatz zum Bismutum subnitricum verordnet werden, das Orthoform und das Anästhesin, haben zwar keinen sekretionshemmenden Einfluß auf die Drüsenfunktion, sondern die Tendenz zu einer unerheblichen Steigerung derselben. In Verbindung resp. in einem mechanischen Gemenge mit dem Bismut wiegt die Komponente des letzteren in ihrem Einflusse auf die Magenschleimhaut vor mit dem Resultate einer Sekretionsherabsetzung. Diese Medikation hat also nicht nur eine praktische, sondern auch eine experimentell begründete Basis. — Als Ersatzmittel für das Bismutum subnitricum kann die Bismuttanninverbindung, das Bismutum bitannicum in Frage kommen, jedoch nur unter gewissen, bei den folgenden Präparaten zu erörternden Umständen. Die Bismutsalizylverbindungen hingegen erscheinen bei allen Zuständen, wo die Sekretion nicht gesteigert, sondern herabgesetzt werden soll, als kontraindiziert.

Auch die Ulkusbehandlung mit Argentum nitricum und seinen Derivaten, die viele Freunde aber auch viele Gegner hat, dürfte

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durch diese experimentell-biologischen Untersuchungen eine pharmakodynamisch festere Basis erhalten. Hier ist ausschlaggebend für die Frage, soll ein Ulkus mit Argentum nitricum bzw. Protargol oder Albargin behandelt werden, oder muß man eine andere Therapie einschlagen, die diffuse Beschaffenheit der Magenschleimhaut, d. h. die Feststellung des Vorhandenseins oder Fehlens eines Katarrhs neben dem Ulkus. — Über das summarische Verhältnis der Ulcera ventriculi mit und ohne diffuse Gastritis findet man sowohl in der pathologisch-anatomischen, wie in der klinischen Literatur wenig Angaben. Rütimeyer[1]) erwähnt bei 25 Fällen einen Prozentsatz von 68 % = $^2/_3$ mit und 32 = $^1/_3$ ohne konkomitierende Gastritis.

Das Verhältnis, ob Gastritis da sei oder fehle, ist bisher für die Therapie kaum verwertet worden, und doch spielt diese Frage für die Argentumbehandlung nach dem Ergebnis der betreffenden Tierversuche die Hauptrolle. Darnach zu schließen darf man wohl die Indikation aufstellen, nur solche Fälle von Ulcus ventriculi mit Argentum nitricum und seinen Derivaten, wie Protargol und Albargin, zu behandeln, welche mit einer diffusen Gastritis kompliziert sind, während die Silbertherapie bei Magengeschwüren ohne Gastritis kontraindiziert ist und durch die Bismutbehandlung ersetzt werden soll.

Das Prinzipielle an diesen Beobachtungen findet auch eine Stütze in der analogen Wirkung des Escalins auf die anatomisch gesunde und die entzündete Magenschleimhaut des Hundes und in den heftigen Kontroversen Bickels, der den experimentellen, und Klemperer, der den klinischen Standpunkt vertritt. Diese heftige Fehde wurde zunächst durch meine Experimente an Hunden mit intakter Magenschleimhaut veranlaßt, indem Bickel diese Untersuchungen ausdehnte, bestätigte und einen weiteren Beleg in seinem Experimente am Mädchen mit intakter Magenschleimhaut fand. Die hierbei durchweg auftretende enorme Sekretionssteigerung veranlaßt Bickel, dem Escalin jede Brauchbarkeit als Ulkusmedikament abzusprechen, während Klemperer an klinischen Beobachtungen zu dem Resultate gekommen sein will, im Escalin ein dem Bismutum subnitricum mindestens gleichkommendes, wenn nicht dasselbe sogar weit übertreffendes Ersatzmittel für dieses gefunden zu haben. — Die Entscheidung, wer hier recht hat, geben meine Versuche am Hunde mit der chronischen Gastritis. Diese Experimente zeigen beim Escalin ein gleiches, wenn auch intensiv stärker ausgeprägtes Verhalten, wie gewisse Adstringentien,

1) Geogr. Verbreitung und Diagnose des Ulcus ventriculi rotundum. Wiesbaden 1906.

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z. B. Protargol und Albargin. Die nicht entzündlich veränderte Magenschleimhaut reagiert auf das Escalin mit einer stürmischen Sekretion, die entzündete Mukosa mit einer erheblichen Sekretionsherabsetzung. Darin liegt des Rätsels Lösung und so kann man sagen, daß beide Gegner in gewissem Maße recht haben. — Gewiß hat das Escalin eine enorme safttreibende Wirkung. Diese trifft aber nach meinen Untersuchungen über Escalin und nach anderen analogen Beispielen nur für den Fall einer nicht entzündlich veränderten Beschaffenheit der Magenschleimhaut zu, während bei einer Gastritis (idiopathischer oder symptomatischer Art, also auch beim Ulkus) die Wirkung des Escalins das reine Gegenteil ist: eine typische Sekretionshemmung. — Die Frage nach der therapeutischen Verwendbarkeit des Escalins beim Ulcus ventriculi kann mithin folgendermaßen beantwortet werden: Auf alle Fälle ist das Escalin da strikte kontraindiziert, wo gastritische Zustände an der Magenschleimhaut fehlen, mithin auch bei Ulcus ventriculi ohne konkomitierende Gastritis. Wohl aber scheint, nach dem Tierversuche zu schließen, das Präparat beim Ulkus mit Gastritis indiziert zu sein. Für solche Fälle mag also Klemperer seinem Präparate eine gewisse therapeutische Bedeutung einer sekretorischen Herabsetzung neben der supponierten hämostyptischen Wirkung zuschreiben. —

Es dürfte eine dankbare Aufgabe der klinischen Untersuchung sein, an einer großen Versuchsreihe von Patienten die hier gezogenen Schlüsse weiter zu prüfen.

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(Gynäkologie Nr. 176.)

Hyperemesis gravidarum[1]).

Von

Dr. Adam Czyżewicz jun.,

Lemberg.

Es werden wohl nicht viele pathologische Zustände bekannt sein, wo die Beschreibungen und Ansichten betreffs der Entstehungsursache so verschieden wären, wie bei Hyperemesis gravidarum. Die ältesten Zeiten überließen uns in Manuskripten und zahlreichen Publikationen Andeutungen, welche als Beweis dienen, daß die eigentümliche Krankheit schon in den ersten Entwicklungsstufen der ärztlichen Wissenschaft bemerkt wurde und daß es nie an Proben fehlte um ihrer Ursache näher zu kommen und sie richtiger zu behandeln. Es kann dies auch nicht wundern, da, wie allgemein bekannt, das Erbrechen sehr oft in den ersten Schwangerschaftsmonaten vorkommt, und unstillbares Erbrechen nur ausschließlich bei Schwangeren, die immer mit spezieller Ehrfurcht und Achtung behandelt waren, zu treffen ist. Diese losen Andeutungen, mehr oder weniger gründlichen Beschreibungen und Proben, der Krankheitsursache näher zu kommen, hat P. Jaffé in seinem Aufsatze über unstillbares Erbrechen in vortrefflicher Weise zusammengestellt. Nach seinen Angaben geschieht der Krankheit schon in den Werken von Paul von Ägina Erwähnung. Schon dieser trachtet sie als spezielle Krankheitsform zu eliminieren, und gibt ihr den Namen „Vomitus assiduus", betrachtet sie aber als nicht gefährlich für Mutter oder Kind. Seine Lehre blieb lange Zeit unverändert, sie ist im 16. Jahrhundert in Mercados Werken aufzufinden, und erst im 18. Jahrhundert forschen nach der Ursache des Leidens Mauriceau und

1) Aus der Klinik der k. k. Hebammenschule des Prof. Dr. A. Czyżewicz in Lemberg. Vortrag, gehalten am 25. Juli 1907 in der gynäkolog. Sektion des X. Kongresses polnischer Ärzte und Naturforscher in Lemberg.

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Delamotte und glauben dieselbe in einer Sympathie zwischen Magen und Gebärmutter, was einen gegenseitigen Einfluß ermöglicht, gefunden zu haben.

Die erste Hälfte des 19. Jahrhunderts hat nicht viel geändert. Außer einer Arbeit von Burns (1809) hinterließ sie aber die Überzeugung, daß die Krankheit nicht so gefahrlos ist, wie dies Paul von Ägina geglaubt hat, und doch in einzelnen Fällen schlecht enden kann. Die Angst vor diesem argen Ausgange hat Simons (1813) bewogen, zum ersten Male die Schwangerschaft im 7. Mondesmonate wegen Hyperemesis zu unterbrechen. Endlich gab 1848 Dubois eine ausführliche Beschreibung der Krankheit und gleich darauf nahm die Pariser Akademie die weiteren Forschungen in ihre Hände, um zunächst festzustellen, ob und wann eine Unterbrechung der Schwangerschaft berechtigt ist. Von nun an macht die Lehre vom unstillbaren Erbrechen rasche Fortschritte, und liefert bald den Beweis, daß die Krankheit gar nicht so einfach ist, wie das früher angenommen wurde. Trotz zahlreicher Forschungen, die jahrjährlich sie aufzuklären suchen, und zwar von verschiedenen Standpunkten, bleibt sie bis heute ein Rätsel, eines von den wenigen, an deren Lösung Arbeiten vieler Gelehrten scheitern. Der klinische Verlauf, die der Mutter und dem Kinde drohenden Gefahren, das Verhalten verschiedener Organe und die Sektionsbefunde sind schon genügend bekannt, die Ätiologie und Heilkunde bildet dagegen so ein Gemenge von verschiedenen Ansichten, daß selbst die Orientierung darin schwer wird. Fast jeder, welcher daran gearbeitet hat, vertritt eine andere Anschauung, auf eine kleine Zahl seiner eigenen Fälle gestützt, da angesichts der Seltenheit der Krankheit niemand über eine größere einheitliche Statistik verfügt, fast jeder gibt eine Reihe glücklich mit seiner Methode, die in anderer Händen als vollkommen wertlos erscheint, geheilter Fälle an. Der Effekt langjähriger Nachforschungen ist dieser, daß ein praktischer Arzt, nach Durchmusterung der Literatur über Hyperemesis gravidarum, nicht nur kein klares Bild über die Ätiologie und Therapie der Krankheit hat, sondern das Vertrauen zu wissenschaftlichen Forschungen verliert und in so ein Gemenge von direkt sich widersprechenden Ansichten gelangt, daß er, sich selbst überlassen, zufällig eine der vielen Thesen aufnimmt und danach handelt, indem er ganz recht bemerkt, daß bis zur definitiven Entscheidung des Streites exakte Methoden der Therapie nicht gestellt werden können. Dem therapeutischen Effekt entsprechend entstehen Anhänger und Gegner der einen oder der anderen Ansicht und der wissenschaftliche Kampf entbrennt von neuem.

Ehe ich die Ursache erörtere, warum so verschiedene Anschau-

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ungen bestehen können und sie, soweit es geht, in Ordnung bringe, will ich zunächst kurz die bisher angegebenen referieren. Eine chronologische Reihenfolge führt hier nicht zum Ziele, da viele ältere schon längst vergessene Ansichten oft von neuem auftauchen und es scheint mir besser, alle die Hypothesen nach pathologischen Prozessen, worin sie die Ursache suchen, einzuteilen, wie dies auch J. W. Williams getan hat.

Den publizierten Hypothesen entsprechend, kann die Hyperemesis gravidarum durch folgende Annahmen aufgeklärt werden:

1. Sie kann auftreten bei Krankheiten des Magens.
2. Sie kann auf reflektorischem Wege zustande kommen.
3. Sie kann auf nervöser Basis beruhen.
4. Sie kann als eine Toxämie betrachtet werden.

Ad. 1. Fälle von unstillbarem Erbrechen Schwangerer bei Magenkrankheiten sind nicht selten beschrieben worden und zwar bei Entzündungen des Magens, Karzinom (Záborszky), Gastromalakie, Ulcus rotundum, bei Erosionen der Magenschleimhaut, pathologischen Zuständen des Darmes usw., überall ist aber von Anfang an die Meinung vertreten, daß man in diesen Fällen eigentlich nicht von Hyperemesis gravidarum als klinisch isolierter Krankheitsform sprechen darf, da Erbrechen auch bei nicht schwangeren Personen aufgetreten wäre, und die Schwangerschaft nur als sekundäre Ursache angesehen werden kann. Vorwiegend ist aber bei vielen Forschern dieser Gruppe die Ansicht angedeutet, daß Krankheiten des Intestinaltraktus das Zustandekommen von unstillbarem Erbrechen erleichtern, daß sie also zwar nicht als primäre, wohl aber als sekundäre Ursache gelten können.

Ad. 2. Die lange Zeit sehr verbreitete reflektorische Theorie hat auch noch jetzt zahlreiche Anhänger. Im allgemeinen beruht sie auf Anastomosen zwischen den Ästen des N. vagus und N. sympathicus, wovon der erste den Magen- und Darmtraktus versorgt, der andere vorwiegend unten in der Bauchhöhle zahlreiche Ganglien im Bereiche der Genitalorgane bildet und sich auch über alle Bauchorgane verbreitet, überall mit Ästen des N. vagus anastomisierend. Es geht schon aus diesem Bilde klar hervor, daß Reize, aus den Geschlechtsteilen stammend, leicht entweder auf direktem Wege oder auf indirektem durch die Medulla spinalis, übertragen werden und Magensymptome hervorrufen können. Auf diesem innigen Zusammenhange beider Nervengebiete und auf dieser Leichtigkeit im Übersenden der Reize haben viele ihre Ansichten basiert und da, trotz Existenz derselben Verhältnisse bei jeder Frau, unstillbares Erbrechen sehr selten vorkommt und nur ausschließlich bei Schwangeren, die Tatsache durch

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Annahme von prädisponierenden Momenten, z. B. Darmtraktuskrankheiten, nervöser Reizbarkeit, durch die Schwangerschaft gesteigert usw., zu erörtern gesucht. Meinungsdifferenzen sind hier fast nicht zu finden. Was aber den eigentlichen, die Krankheit hervorrufenden Reiz anbetrifft, sind die Ansichten sehr verschieden und es werden fast alle Frauenkrankheiten in Rede gezogen. Es wird nun nach der Ursache gesucht bei Erosionen des Scheidenteiles, Entzündungen der unteren Hälfte der Gebärmutter oder der oberen und des Peritoneum (Horwitz), in endometritischen Prozessen (Jaggard), Krankheiten der Eihäute, übermäßiger Ausdehnung der Gebärmutter bei Hydramnios oder Zwillingen, Bildung von Molen, Rigidität des Muttermundes (Kehrer) oder auch der Gebärmutter in toto, weiter in einer Reizbarkeit der Gegend des inneren Muttermundes (Bantock, Mueller), Cervixrissen (Sänger), in einer reflektorischen Wirkung der wachsenden Gebärmutter (Baisch, Oelschläger). Anämie des Peritoneums wegen starker Spannung auf dem sich vergrößernden Uterus (Tuszkai), einer Gehirnanämie beim Aufstehen vom Bette (Evans), in Schwangerschaftswehen (Gottschalk). Deviationen der Gebärmutter vorwiegend Retroflexio und folgendem Druck auf die Nervenganglien (Graily Hewitt), im Drucke auf diese Ganglien durch Exsudate, oder später zurückbleibende Adhäsionen, vorwiegend im Bereich des Promontoriums (Müller), einer gesteigerten Magensaftabsonderung neben reflektorischer Wirkung des schwangeren Uterus (Monin), in einer reflektorischen Pyloruskontraktion oder Zusammenziehung der Muskeln des ganzen Intestinaltraktus (Goffroy), einer reflektorisch auf Grund von Frauenkrankheiten mit Gravidität kompliziert auftretenden Neurosis ventriculi (Sutugin), einer Reizung des Spinalbrechzentrums direkt durch Druck der Gebärmutter oder auch indirekt (Windscheid) und in vielen anderen pathologischen Zuständen.

Wie oben angedeutet, stammt diese Theoriengruppe aus ältester Zeit und hat auch bis heute zahlreiche Anhänger, verliert aber, angesichts neuer Forschungen, die auf ganz anderem Wege zur Aufklärung der Krankheit schreiten, immer mehr ihren Boden. Sie bestand immer und besteht auch noch jetzt aus dem Grunde, daß sehr oft die Heilung bestehender Frauenkrankheiten einen segensreichen Einfluß auf das Erbrechen hat; es ist nur absolut nicht festzustellen, ob diese Besserung post hoc oder propter hoc eingetreten war.

Ad 3. Die Neurosentheorie verdankt ihre Verbreitung den Arbeiten von Ahlfeld und Kaltenbach, obwohl sie schon viel früher aufgebaut wurde in den Publikationen von Krieger, Eulenburg, Anquetin, Braxton Hicks, welche, auf negativen Befund der Autopsien gestützt, zur Annahme von nervösen Ursachen neigten und

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zwar solchen, die tödlich sein können, ohne Spuren zu hinterlassen. Ihre Lehre war aber wenig bekannt bis zur Zeit, wo eine Polemik um Priorität zwischen Chazan, Ahlfeld und Kaltenbach sie allgemein verbreitet hat. Als Ausgangspunkt in dieser Hypothesengruppe wird eine Nervenkrankheit angenommen, und zwar geben die zwei ersten als Ursache diverse Neurosen an, die alle einen identischen Einfluß auf das Entstehen der Hyperemesis haben sollen, der letztere dagegen speziell die Hysterie. Diesem wissenschaftlichen Streite haben sich gleich zahlreiche Diskussionen in gynäkologischen Gesellschaften und Zeitungen angeschlossen, zwischen den Anhängern der neuen Lehren (Muret, Frank, Gräfe, Dorff, Opitz, Klein, Schäffer) und ihren Gegnern (Windscheid, Zweifel, Feinberg), Diskussionen, wo jeder durch immer neue Beweise seine Anschauung zu begründen suchte und die, trotz enormer Arbeitsausdehnung, nicht nur zu keiner einheitlichen Ansicht über die Ätiologie der Krankheit geführt, sondern nur den ganzen Standpunkt noch bedeutend mehr verwickelt haben.

Ad 4. Die jüngste von allen, erst in letzter Zeit aufgestellte, toxämische Theorie wurde auch anfangs nicht so allgemein anerkannt, wie die anderen, und blieb, als mehrere Forscher sie angenommen haben, auch nicht einheitlich sondern gleich in mehrere Gruppen eingeteilt. Ihre Grundsteine sind in der Arbeit von Fischl (1884) zu finden. Er nimmt zwar auch eine nervöse Prädisposition an, glaubt aber, daß als eigentliche Ursache eine Koprostase mit Bildung anormaler chemischer Prozesse beschuldigt werden kann. Neun Jahre später publiziert Lindemann (1893) ein Sektionsprotokoll nach Hyperemesis gravidarum und beschreibt ausführlich Veränderungen der Leber, Milz und Nieren als parenchymatöse und fettige Degenerationen, Entzündungszustände der peripheren Nerven, bei wenig Aushungerungserscheinungen und stellt als erster die Meinung auf, daß angesichts des Sektionsbefundes, der lebhaft an eine chronische Vergiftung erinnert, die Hyperemesis als Vergiftung mit noch unbekannten Toxinen angesehen werden darf. Dies war der Ausgang der ganzen Theorie, und als ihn V. Antouchevitsch (1897) in einer Sitzung des Ärztekongresses in Moskau von neuem aufgenommen und auf die Ähnlichkeit zwischen der Hyperemesis und den Symptomen bei Tieren, denen man eine Nahrung ohne Natrium und Kalium gereicht, verwiesen hat, beginnen Untersuchungen des Stoffwechsels, Beobachtungen der Krankheit vom neuen Standpunkte und die Theorie ist im Aufschwung begriffen.

Die Anhänger dieser Hypothese sind insofern einig, daß sie als Ursache des unstillbaren Erbrechens eine Vergiftung mit im Blut

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deponierten Stoffen annehmen und insofern verschiedener Meinung, daß sie verschiedene Stoffe beschuldigen. Immer heben sie aber hervor, daß die Theorie nicht auf alle Fälle von Hyperemesis passen kann. Alle diese Nachforschungen gaben als Resultat eine Reihe von verschiedenen Ansichten, je nach dem Körper, welcher als Agens beschuldigt war. Sie sind folgende:

a) Turenne sucht die Giftquelle in einer Absonderung des Corpus luteum, welches als Drüse mit innerer Ausscheidung angesehen werden muß. Ihr Produkt ruft normal eine Hyperämie der Genitalorgane hervor, vor und während der Periode, allgemeine Symptome bei amenorrhoischen Frauen und mutmaßliche Schwangerschaftssymptome, die bei nervöser Prädisposition sich bis zur Hyperemesis steigern können. Die Ursache, daß die Erscheinungen bei Nichtschwangeren fehlen, wäre darin zu suchen, daß das Gift vom menstruellen Blute fortgeschwemmt wird; die Periode wäre nun als eigentliche, gewöhnliche Folge der Wirkung des Corpus luteum-Giftes anzusehen. In Fällen, wo die Menstruation aufhört, also auch in der Schwangerschaft, gelangt das Gift in den Blutkreislauf und ruft Vergiftungserscheinungen hervor.

b) Dirmoser hält die Hyperemesis für eine Autointoxikation mit Produkten eines anormalen Stoffwechsels im Darmtraktus und spezialisiert dies folgendermaßen: Die Nerven des Darmes und Magens stehen in enger Verbindung mit denen der Genitalorgane, und in normalem Zustande üben die letzteren ihren regulierenden Einfluß auf den Magen im Wege der Anastomosen zwischen N. vagus und N. sympathicus aus. Die Vergrößerung der Gebärmutter, Veränderung der Eierstöcke, des Peritoneums usw. in der Schwangerschaft übt einen mächtigen Reiz auf die hier liegenden Nervenenden und ruft reflektorisch eine Behinderung der Magenfunktion, ja sogar antiperistaltische Bewegungen hervor. Demzufolge kommt es zu anormalen Fäulnisprozessen, zur Bildung giftiger Körper und angesichts der verlangsamten Peristaltik zu deren leichter Einsaugung. Das Ganze kann wohl bei gesunden Frauen, um so eher bei nervös belasteten vorkommen.

Kurz gesagt hält also Dirmoser das unstillbare Erbrechen für eine Autointoxikation mit anormalen Stoffwechselprodukten und begründet seine Ansicht in erster Reihe mit atypischen Harnbestandteilen, wie Azeton, Indoxyl, Skatoxyl, Schwefelverbindungen usw., die er in seinen Fällen in größerer Menge nachgewiesen hat.

c) Pinard, Champetier de Ribes und Bouffe de Saint Blaise halten die Krankheit für eine Vergiftung auf Grund einer schlechten Leberfunktion, und als Stützpunkt nehmen sie Sektionsbefunde an, wo

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in der Leber Veränderungen wie bei Atrophia hepatis flava festgestellt wurden. Auch früher waren schon ähnliche Befunde bekannt, nur nicht zur Aufklärung der Ätiologie des unstillbaren Erbrechens herangezogen. Im Jahre 1879 hat sie schon Matthews Duncan gefunden, später Stone, Ewing und Edgar, aber erst J. Whitridge Williams hat im Jahre 1905 auf Grund von drei beobachteten Fällen, durch gründliche Untersuchung des Stoffwechsels die Hypothese wissenschaftlich begründet. Diese Untersuchung lehrte, daß, von Anfang der Krankheit an, im Urin Ammoniak zu finden ist, und zwar in großer Menge, bis 46% (gegen normal 3—5%), was entweder für schwere Schädigung der Leber spricht oder für Existenz eines saueren Giftes im Blutkreislauf, das zu neutralisieren ist — später kann man auch Eiweiß konstatieren. Dieser Befund beweist eine primäre Läsion der Leber und sekundäre der Nieren durch das im Blut kreisende Toxin.

d) Karl Behm sucht 1903 die Vergiftungsursache im Fruchtei. Nach dieser Meinung ist die Hyperemesis gravidarum eine Intoxikation, von der Eioberfläche ausgehend, wahrscheinlich synzytialen Ursprunges, und als Beweis dient die Tatsache, daß nicht nur bei kranken, sondern auch bei gesunden Schwangeren Zottenelemente im mütterlichen Blute zu treffen sind. Wenn man dabei beachtet, daß das Erbrechen gewöhnlich mit Beginn der Schwangerschaft sich einstellt und in 5—6 Wochen seinen Höhepunkt erreicht, d. h. zu einer Zeit, wo an einem großen Teile der Eioberfläche Zotten zugrunde gehen, das Chorion laeve bildend, so ist es wahrscheinlich, daß Zottenelemente, speziell das Synzytium, in das mütterliche Blut gelangen und die Vergiftung verursachen können. Auch die Tatsache, daß mit der vollkommenen Entwicklung der Plazenta, d. h. im 4 bis 5. Schwangerschaftsmonat, das Erbrechen gewöhnlich zu Ende geht, spricht für diese Ansicht.

Die Ursache der Krankheit wäre somit im Synzytium zu suchen, und alle Krankheiten des Genitalapparates, Neurosen usw. als prädisponierendes oder die Krankheit verschlimmerndes Moment zu halten.

e) Die letzte Hypothese sucht die Ursache der Vergiftung der Schwangeren in Produkten des Fruchteies (Clivio) oder in Stoffwechselprodukten der Frucht (Czempin, Starzewski). Der erste von den dreien publizierte 1901 seine Ansicht, daß das Fruchtei giftige Substanzen absondert, welche, in das mütterliche Blut gelangt, eine Intoxikation hervorrufen, unter anderem durch Erbrechen gekennzeichnet; bei regelwidriger Funktion des Intestinaltraktus, Krank-

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heiten der Geschlechtsorgane, Neurosen usw. kann dann leicht das Erbrechen sich zu einer Hyperemesis steigern.

Czempin trat mit seiner Meinung zuerst 1893 in einer Diskussion im Berliner physiologischen Institut auf und muß somit als Vater der Theorie von der Vergiftung der Mutter durch das Kind angesehen werden. Zu dieser Kenntnis war er durch Untersuchung der Plazentastruktur gelangt. Es war ihm klar, daß ein so kompliziertes Organ gewiß wohl auch seine spezielle Funktion haben muß und von dem Standpunkte ausgehend hat er seine Hypothese entwickelt: Die wachsende Frucht bildet eine ganze Menge von Giften, welche, in das mütterliche Blut gelangt, eine Intoxikation hervorrufen. Tatsächlich kommt es dazu im Anfange der Schwangerschaft, da die Plazenta noch nicht fähig ist, das Gift zu neutralisieren. Als Symptome treten hervor: Erbrechen, Kopfweh, Kopfschwindel usw. Später binden die Plazentarzellen das Gift und der mütterliche Organismus bildet Gegengifte, zum Neutralisieren der doch ins Blut hereingeschwemmten Gifte bestimmt, und die obigen Symptome verschwinden. Bei regelwidriger Funktion des Fruchtkuchens können die fötalen Gifte in das mütterliche Blut in größeren Mengen gelangen und unstillbares Erbrechen oder Eklampsie hervorrufen.

Die letzte Arbeit, von J. Starzewski in den Jahren 1895—1897 in unserer Schule durchgeführt und im Jahre 1906 veröffentlicht, hebt den Begriff einer „Schwangerschaftsinfektion“ hervor und klärt ausführlich die Symptome einer physiologischen und pathologischen Gravidität mit dem Eindringen von fötalen Stoffwechselprodukten in das mütterliche Blut auf. Dieses Exkret des Fruchteies übt seinen Einfluß auf Drüsen des mütterlichen Körpers aus und zwar höchstwahrscheinlich auf dem Wege einer atypischen Funktion des Nervensystems und auch durch direkte Wirkung auf die Drüsensubstanz.

„Wir dürfen nicht fehlgehen — sagt Starzewski im physio-
„logischen Teile — wenn wir in analoger Weise auch das Er-
„brechen Schwangerer, jenes so überaus häufige Symptom der
„Schwangerschaftsinfektion, erklären. Auch hier hätten wir zwei
„Ursachen: die eine, die indirekte, die durch das zentrale Nerven-
„system wirkende, die andere, die direkte, der Reizzustand, in
„welchen die Magenwand durch das Fruchteiexkret versetzt wird,
„dessen Ausscheidung vielleicht ebenso, wie die des Morphins,
„durch die Magenschleimhaut in die Magenhöhle erfolgt. In
„unseren gegen dieses Leiden gerichteten therapeutischen Maß-
„nahmen ist auch diese Erklärung gerechtfertigt. Denn einer-
„seits erzielen wir gute Resultate, wenn wir Mittel verabreichen,
„welche die Reizbarkeit des Nervensystems herabsetzen, andrer-

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„seit schaffen wir den Kranken bedeutende Linderung, wenn wir „deren Magen einer systematischen Spülbehandlung unterziehen."

Und im pathologischen Teile schreibt er weiter, daß, wenn die Fähigkeit des mütterlichen Organismus, ein wirksames Schwangerschaftsantitoxin zu bilden, bei Schwangerschaftsinfektion zu gering ist, die Symptome nach Ende der ersten Schwangerschaftshälfte nicht schwinden, sondern im Gegenteil noch manchmal zunehmen. Hierher gehört unter anderen auch die Hyperemesis gravidarum.

So sieht, in kurzen Zügen, das Bild unseres Wissens über die Hyperemesis gravidarum aus, dies ist das Ansichtengemenge, das wir von unseren Vorgängern geerbt haben. Wenn jemand daraus eine klare Vorstellung der Ätiologie der Krankheit gewinnen wollte, ohne eigene Erfahrung in Frage zu ziehen, so möchte er sehr bald zur Überzeugung kommen, daß dies nicht möglich ist. Er dürfte auch nicht einer von diesen Theorien den Vorzug geben, auf das allgemeine Ansehen des einen oder des anderen Verfassers gestützt, da hier im ganzen die glänzendsten Namen sich gegenseitig bekämpfen.

Eine Lösung des Rätsels ist nicht weit zu suchen; sie springt von selbst in die Augen mit Aufstellung der Ansicht, daß die Krankheit, von der alle sprechen, keine einheitliche Form ist, sondern ein Konglomerat von verschiedenen pathologischen Prozessen, die nur das Symptom des schwer zu stillenden Erbrechens gemein haben, sonst aber verschieden voneinander sind. Bis neulich war es ganz ähnlich mit der Influenza, von der, auf Grund zahlreicher Untersuchungen, immer mehr Fälle isoliert werden und die demzufolge immer seltener, aber immer mehr bekannt und spezialisiert wird.

In vielen Aufsätzen habe ich die Ansicht gefunden, daß das unstillbare Erbrechen aus verschiedenen Ursachen entstehen kann, nirgends dagegen mit der Meinung, daß man mit dem Namen Hyperemesis nur einige Krankheitsformen mit gründlich definierten Symptomen und Verlauf belegen darf, den Rest dagegen mit ihrer eigenen Bezeichnung nennen muß. Und doch sollte es so sein, da doch grundsätzlich Erbrechen auf hysterischer Basis oder bei Pylorusverengung wegen Karzinom von einem bei allgemeiner Vergiftung verschieden ist. Wir sind eben nur gewohnt, jedes Erbrechen bei einer Schwangeren der Schwangerschaft zuzuschreiben und jedes, welches mit gewöhnlichen Mitteln nicht zu stillen ist, und wobei es zu Sinken des Körpergewichtes aus irgendwelcher Ursache kommt, mit dem Namen Hyperemesis zu belegen, mit einem Namen, der eigentlich nur leerer Schall ist und gar nicht existenzberechtigt. Als Beweis zahlreiche Protokolle von Sektionen, welche bei Schwangeren mit Hyperemesis

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Carcinoma pylori, Peritonitis, Tuberkulose usw. gefunden haben. Daß in diesen Fällen nicht eine anormale Krankheitsursache, sondern ein Diagnosenfehler vorlag, liegt auf der Hand. Und so kann es nachher nicht wundern, daß einer als Grund der Hyperemesis Hysterie, der andere Retroflexion oder Druck auf Nervenganglien usw. angibt, und daß jeder in diesen Fällen seine Ansicht mit gutem therapeutischen Effekte bekräftigt, es kann aber auch nicht wundern, daß, wenn der andere die Anschauung des ersten annimmt, er leicht auf einen Fall treffen kann, wo die angebotene Behandlung wirkungslos bleibt und der klinische Verlauf eine ganz andere Ursache zutage fördert. Sie hatten beide zwei verschiedene Krankheiten vor sich, die nur ähnliche Symptome aufgewiesen haben, was ja leicht vorkommen kann. Als Beispiel will ich nur den Schmerz in der Herzgrube hervorheben. Er stellt sich bei Krankheiten des Magens, des Herzens, der Leber, bei frühzeitiger Perimetritis usw. ein. Wenn man diesen Schmerz als selbständige Krankheit ansehen wollte, so könnte man auch verschiedene Ursachen und Behandlungsmethoden angeben.

Wie also oben angedeutet, kann ich die Hyperemesis gravidarum, oder, bei anderer Benennung, den Vomitus gravidarum perniciosus, nicht für eine selbständige Krankheit halten, sondern muß sie als Symptomenkomplex bezeichnen, der aus verschiedenen Ursachen entstehen kann und demgemäß verschiedenen Krankheiten zugeschrieben werden muß.

Beweise dafür liefert eine Durchmusterung publizierter Beschreibungen des Leidens. Kaltenbach z. B. gibt an, daß er bei seinen Patientinnen Merkmale der Hysterie gefunden hat, viele andere treten dem gleich entgegen, Williams konstatierte im Urin größere Ammoniakmengen und erst sub finem Eiweiß, Dirmoser erwidert ihm gleich, daß Eiweiß oft von Anfang an vorkommt, Ammoniak ganz wenig zu finden ist, dagegen Indoxyl und Skatoxyl vermehrt sind usw. usw. Man kann nicht glauben, daß so ernste Forscher Irrtümer in kardinalen Untersuchungen begehen, oder nicht wahren Befund veröffentlichen, und es ist viel rationeller anzunehmen, daß sie vor zwei verschiedenen Krankheiten gestanden, die sie zwar diagnostiziert und genügend erklärt, aber falsch mit demselben Namen belegt haben.

Wo ist nun die Ursache dieser Verwirrung zu suchen? Es ist mir vollkommen klar, daß sie im Namen des Leidens liegt, der keinen pathologischen Prozeß, sondern nur ein Krankheitssymptom bezeichnet. „Hyperemesis gravidarum“ sagt nur, daß bei einer schwangeren Frau Erbrechen vorkommt, welches mit unseren Mitteln nicht zu

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stillen ist — „Vomitus gravidarum perniciosus“ ist insofern eine bessere Bezeichnung, daß sie gleich den gefährlichen Einfluß auf die Patientin andeutet. An diesen schon existierenden Namen wurden erst später Symptome angekettet, welche teilweise, was das Erbrechen, Verminderung des Körpergewichtes, Verhalten des Pulses usw. anbetrifft, übereinstimmen, oder wieder sich gegenseitig bekämpfen, wie z. B. im Verhalten der Temperatur, des Urins, im Krankheitsverlauf, der Prognose, dem Ausgang usw. Dieser Fehler steht in unserer Nomenklatur nicht vereinzelt da und beruht auf der Benennung der Krankheit nach einem ihrer Symptome, wenn auch nach dem am meisten charakteristischen. Fast bis vor kurzem haben alle „Icterus gravis“ bei Neugeborenen diagnostiziert, statt von „Sepsis“ zu sprechen, „Icterus catarrhalis“, statt „Duodenitis“ oder „Cholangitis catarrhalis“ mit Ikterus und auch jetzt tun dies noch viele. Ganz ähnlich kann man heute noch Diagnosen antreffen, wie „Epistaxis“, „Hämaturia“, „Hämatemesis“ usw., obschon sie alle überzeugt sind, daß dies keine Bezeichnungen einer selbständigen Krankheit sind. Mit Aufschwung der ärztlichen Wissenschaft werden diese Namen, als Diagnosen, vertrieben und bleiben nur als Bezeichnung einzelner Symptome, und nirgends in Krankenhäusern sind sie mehr auf den Tafeln zu sehen. Nur einzig für Krankheiten, von denen wir eigentlich nicht wissen, was sie sind, haben sie sich noch erhalten. In der Geburtshilfe gibt es solcher zweie: die Eklampsie und Hyperemesis. Die erste besitzt glücklicherweise einen Namen, der nichts sagt von ihren Symptomen, und daher ungestört auch weiter bestehen kann, die zweite gibt von Anfang an durch ihre Benennung eine falsche Vorstellung, weil sie nicht auf die Krankheit selbst, sondern auf ihr Zeichen hinweist.

Nach dem Gesagten sollte man eigentlich die ganze Krankheit, samt ihrem Namen, aus dem medizinischen Wörterbuche streichen. Bisher war das, angesichts unserer Angewöhnung, nicht möglich, ich bin aber fest überzeugt, daß sie später auch wirklich verschwinden wird. Vorläufig will ich sie nur auf einzelne Komponenten zerlegen, dieselben dort einreihen, wo sie hingehören und eine Gruppe isolieren, welcher ich den Namen „Hyperemesis gravidarum vera“ gebe und denselben so lange gebrauche, bis er durch einen besseren, die eigentliche Krankheitsursache bezeichnenenden, vertreten wird.

Auf diesem Wege müssen zunächst die Fälle eliminiert werden, deren Ursachen man schon in allgemein bekannten pathologischen Zuständen gefunden hat. In erster Reihe treten die Fälle zur Seite, wo Erbrechen durch Magen- und Darmkrankheiten hervorgerufen wird, da es doch bei Magenkarzinom, Bauchfellentzündung usw. zur

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Regel gehört, auch wenn die Patientin nicht schwanger ist, und da es, von Kachexie begleitet, auch bei Männern zustande kommen kann. Die Schwangerschaft ist dabei nur ein zufälliger Befund. Sie kann zwar den Zustand verschlimmern, indem eine den Schwangeren eigentümliche Reizbarkeit, wahrscheinlich auf Grund der Graviditätsinfektion im Sinne Starzewskis, hinzutritt, man kann dies aber höchstens als Kombination zweier Ursachen: der physiologischen Gravidität und der pathologischen Magenkrankheit, ansehen und von einem verschlimmernden Einfluß der Schwangerschaft z. B. auf Magenkrebs sprechen, nie aber von einer speziellen Krankheitsform, der Hyperemesis und nie deren eigentliche Ursache im graviden Uterus suchen, die eigentliche Krankheit als Nebensache betrachtend. In diesem Falle muß die erste Diagnose bestehen und nur hinzugefügt werden, daß deren Symptome bei bestehender Schwangerschaft verschlimmert werden. Nur so ein Standpunkt kann einer wahren Kritik widerstehen.

Dasselbe gilt auch für die Neurosentheorie. Auch hier kann das, nebenbei fast immer stillbare, Erbrechen auf Grund einer Neurose mit dem, welches als Symptom einer oft durch Degenerationen tödlichen Krankheit auftritt, nicht identifiziert werden. Daß eine bestehende Hysterie verschiedene Symptome zahlreicher Krankheiten nachahmen kann, ist allgemein bekannt, aber trotzdem unterscheidet doch niemand als spezielle Krankheit z. B. eine Hemiplegie und teilt sie nicht in eine auf Grund von Gehirnveränderungen entstandene und eine zweite funktionelle, sondern jeder diagnostiziert die Grundkrankheit und fügt eine Beschreibung ihrer momentanen Symptome bei. So muß man auch Hysterie erkennen, wenn man ihre charakteristischen Merkmale gefunden, und beifügen, daß sie im gegebenen Falle die Symptome der Hyperemesis nachahmt. Es kommt dies vorwiegend bei Schwangeren vor und kann gedeutet werden entweder als Kombination zweier Krankheitszustände oder als Neurose, die angesichts psychischer Reize und der Überzeugung, daß Erbrechen in der Gravidität leicht vorkommen kann, diese Form angenommen hat. Daß so ein psychischer Einfluß wohl möglich ist, dafür liefert den Beweis ein mir bekannter Fall, wo einige Male wegen Hyperemesis ein Abort eingeleitet wurde, bis endlich dieselben Symptome einmal außerhalb der Schwangerschaft zutage getreten — und sehr leicht geheilt wurden. Es ist vollkommen klar, daß eine antihysterische Behandlung in diesem Falle auch während der Schwangerschaft prompt wirken und neue Anhänger der Kaltenbachschen Theorie, die doch ganz auf Fällen von Hysterie sub forma einer Hyperemesis basiert ist, schaffen möchte.

Dasselbe gilt auch für alle anderen Neurosen, um so mehr, da sie

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in der Gravidität gesteigert sind. Bei der Besprechung der Hyperemesis gravidarum müssen nun alle diese Fälle zur Seite geschoben werden, weil sie keine Hyperemesis sind, nur andere Krankheiten, weil sie eine andere, nicht symptomatische, sondern ursächliche Behandlung verlangen und die, wenn nur die Grundkrankheit heilbar ist, auch sehr leicht geheilt werden.

Die reflektorische und toxämische Hypothese erfordern eine besondere Besprechung. Was die erste anbetrifft, ist es wahrlich schwer, die Hyperemesis als Symptom von Erosionen, Deviationen, Entzündungen der Genitalien usw. anzuschauen, da dies mit dem lokalen Charakter dieser Krankheiten in Widerspruch steht und irreleiten muß und es scheint viel begreiflicher, wie dies schon immer öfter in der Literatur hervorgehoben wird, anzunehmen, daß der Zusammenhang zwischen den beiden Leiden ein zufälliger ist und sie aufeinander keinerlei Wirkung ausüben. Wenn man in der Praxis alles genau beobachtet, so trifft man oft Fälle, wo z. B. eine Retroflexion neben einer ausgesprochenen Hysterie besteht, und trotzdem tritt in der Schwangerschaft keine Hyperemesis, ja überhaupt kein Erbrechen auf. Wenn die reflektorische Theorie wahr wäre, so müßten gerade solche Fälle voranstehen, was aber gar nicht übereinstimmt. Dasselbe gilt auch für Entzündungen der Genitalien, Erosionen usw. und diese Tatsachen haben auch viele Forscher gezwungen, den Einfluß von Frauenleiden auf den Magen zu bezweifeln. — Der Reflex, sei es direkt oder indirekt, auf dem Wege eines Spinalzentrum, durch Druck auf neben der Gebärmutter liegende Nervenganglien hervorgerufen, kann auch nicht erhalten werden, da sehr oft neben dem Uterus Geschwülste liegen, die einen bedeutend stärkeren Druck ausüben, wie die schwangere Gebärmutter, und trotzdem kein unstillbares Erbrechen außerhalb der Schwangerschaft hervorrufen. Während der Gravidität ist nur ein Fall von Lapeyre 1901 beschrieben, wo ein Ovarialkystom neben Schwangerschaft und Hyperemesis bestand und wo ein Beweis erbracht wurde, daß die letztere nur von der Schwangerschaft abhängig war und die Exstirpation des Tumor erfolglos blieb.

Tuszkais Ansicht hat schon vor mir W. J. Brock 1883 widerlegt, indem er angegeben hat, daß die Hyperemesis vorwiegend bei Mehrgeschwängerten vorkommt, wo bei Schlaffheit der Muskeln und des Muttermundes eine gesteigerte Spannung der Gebärmutterwand und des Peritoneum nicht annehmbar ist. Er fügt nebenbei noch hinzu, daß die Krankheit bei Rigidität des Muttermundes nicht vorkommt, was auch wirklich wahr ist und gegen Reizung des Muttermundes als Ursache spricht.

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Das Gesagte zusammenfassend, kann man wohl behaupten, daß bei verschiedenen pathologischen Zuständen der Genitalorgane, ein bis jetzt mit dem Namen Hyperemesis gravidarum belegter Symptomenkomplex vorkommen kann, daß aber kein Beweis vorliegt, ihn als Reflex zu bezeichnen. Im Gegenteil spricht viel dafür, daß er kein Reflex ist, und daß man nach seiner Ursache in einer ganz anderen Richtung forschen muß.

Es bleibt noch zuletzt die toxämische Theorie. An sie will ich mich wenden und nach ihr den mit dem Namen Hyperemesis gravidarum belegten Symptomenkomplex zu deuten trachten. Zunächst sind hier nach Dirmosers Ansicht entstandene Fälle auszuschließen. Wenn die Krankheit eine Autointoxikation aus dem Darmtraktus sein sollte, so müßten diese Fälle überhaupt den Darmintoxikationen, wie z.B. nach Genuß von faulendem Fleisch, nach atypischen Verdauungsprozessen usw. angereiht werden und den ganzen Symptomenkomplex darf man dann wieder nicht für eine selbständige Krankheit, sondern muß sie für eine Erscheinung der Vergiftung halten. Gegen die Annahme, daß die Schwangerschaft reflektorisch die Peristaltik der Därme hemmt, oder gar Antiperistaltik hervorruft, spricht die Tatsache, daß bei Uterustumoren, z. B. Myomen, kein Erbrechen vorkommt, nur speziell bei Gravidität. Es muß somit gerade in der letzteren die Ursache gesucht werden.

Ganz ähnlich kann man auch die Leber nicht beschuldigen. Gegegebenenfalls müßte es doch einmal vorkommen, daß die Leber auch außerhalb der Schwangerschaft insuffizient wird, und es müßte dann zu einer Hyperemesis gravidarum bei einer nicht graviden Frau oder gar bei einem Manne kommen, wo doch die Krankheit schon nicht speziell mit der Schwangerschaft zusammenhängen und ihren Namen verdienen könnte. Viel eher ist es anzunehmen, daß die Leberveränderungen sekundär sind, durch ein im Blute kreisendes, den Schwangeren eigentümliches, also mit der Schwangerschaft im engen Zusammenhange stehendes Gift hervorgerufen.

Nolens, volens sind wir genötigt, die Ursache der Hyperemesis in der schwangeren Gebärmutter zu suchen, und da diese sich von einer nicht schwangeren nur durch das darin sich entwickelnde Ei unterscheidet, in dem Eie selbst. Mit Turennes Ansicht, daß diese Ursache in der Ausscheidung des Corpus luteum liegt, kann ich nicht einig werden, da so ein gelbes Körperchen bei jeder Schwangeren existiert und trotzdem Hyperemesis zu seltenen Erscheinungen gehört und da Turenne auch die Menstruation als Folge derselben Ausscheidung ansieht, obwohl heute allgemein gelehrt wird, daß die Menstruation und Ovulationen in keinem ursächlichen Zusammenhange

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stehen und eine Periode vor der Ovulation zustande kommen kann, also zur Zeit, wo noch kein Corpus luteum existiert. Wenn man nun für den Ausgangspunkt das Fruchtei halten will, so ist nur zwischen der synzytialen Theorie und der Schwangerschaftsinfektion zu wählen. Wir haben noch zu wenig Belege, um zwischen diesen beiden Ansichten zu entscheiden, weil deren Anatomie und Chemie kaum angefangen ist, schon jetzt sind aber zwei Punkte zu konstatieren, welche zur Annahme der Schwangerschaftsinfektion drängen, und zwar:

1. Hyperemesis kommt bei Dissemination des Chorionepithelioma malignum nicht vor, obwohl hier das Synzytialgewebe überall in Metastasen zu finden ist, und es gerade da seine spezifische Wirkung, Erbrechen hervorzurufen, entfalten sollte, wenn es eine solche besitzt.

2. Es ist allgemein bekannt, daß Erbrechen nach Absterben der Frucht sistiert, wenn auch eine Fehlgeburt noch gar nicht im Gange ist und das Synzytium noch seine Lebensfähigkeit bewahren konnte.

Dieser Weg der Deduktion führt also zur Behauptung, daß der als „Hyperemesis gravidarum“ oder „Vomitus gravidarum perniciosus“ bekannte Symptomenkomplex die Folge einer Schwangerschaftsinfektion im Sinne Starzewskis ist oder, anders gesagt, die Folge der Vergiftung der Mutter durch Exkrete der Frucht. Ob sich das bei aktiver Mitbeteiligung der Plazenta vollzieht, wie dies Czempin will, oder auch ohne diese, bin ich nicht imstande zu entscheiden. Natürlich handelt es sich hier nur speziell um eine Form der Hyperemesis, wo keine ausreichende Ursache zu finden ist, denn anderenfalls ist sie nur ein Symptom des bestehenden Leidens, d. h. nur eine Form, für die ich den Namen Hyperemesis bis zu der Zeit, wo sie entsprechender benannt wird, bewahrt habe. Jedenfalls auch diese ist nur ein Symptom einer allgemeinen Toxämie, wo die Frucht als Agens gilt, eines pathologischen Zustandes, welcher bei vermehrter Toxizität der Fruchtexkrete, oder verminderter Widerstandsfähigkeit der Mutter zustande kommt. Diese Toxämie verursacht aber so zahlreiche pathologische Veränderungen, daß man darin spezielle Gruppen als einzelne Krankheitsformen zu unterscheiden berechtigt ist. Als solche gilt nach meiner Ansicht die Hyperemesisform, für die ich den Namen „Hyperemesis vera“ vorschlage, im Gegensatze zur „Hyperemesis spuria“, die durch verschiedene Krankheiten verursacht sich nicht als wissenschaftliche Einheit isolieren läßt, ähnlich wie z. B. die Angina pectoris spuria.

Wenn meine Meinung auch wirklich wahr ist, so müssen die konstatierten Symptome, nicht nur die klinischen, sondern auch die

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Sektionsbefunde ein Merkmal der Toxämie tragen, die doch ihre Grundursache ist und dementsprechend möchte ich an diesem Platze kurze Krankengeschichten von Fällen, die mir in der hiesigen k. k. Hebammenschule zur Beobachtung kamen, anschließen.

1. Nr. 767, 1903/4. M. S., eine 27jährige Hausmeistersehefrau aus Lemberg, aufgenommen am 10. VIII. 1904. Eine Nullipara, die seit 22. V. 1904 amenorrhoisch war, und daraus auf bestehende Schwangerschaft schloß. Durch die ganze Zeit litt Pat. an heftigem Erbrechen, welches zunächst ein paarmal des Tages, später gleich nach jedem Bissen sich einstellte. Infolgedessen kam es zu Kräfteverfall und Abmagerung.

Die Untersuchung ergab eine hochgradige Abmagerung und Erbrechen tatsächlich nach jedem verschluckten Bissen. Das Erbrochene bestand aus unverdauten, eben genossenen Speisen und Flüssigkeiten, mit Magenschleim vermischt. — Der vergrößerte Uterus, mit allen Zeichen einer etwa 3monatlichen Schwangerschaft, reichte bis 3 Querfinger über die Symphyse. Gut beweglich war er vollkommen frei von krankhaften Veränderungen. Ganz ähnlich auch die Adnexe. Im Bereich der sensiblen Nerven keinerlei Veränderungen. Neurosen ausgeschlossen. Im Urin eine Spur von Eiweiß.

Die Diagnose „Hyperemesis gravidarum", war nach obigem Befunde sicher und da keinerlei Erkrankungen irgendwelcher Organe einen Anhaltspunkt gaben, mußte deren Ursache in einer Schwangerschaftsinfektion gesucht werden. Da keine momentane Lebensgefahr drohte, habe ich beschlossen, das Leiden bei Erhaltung der Schwangerschaft zu behandeln. Es wurde ausschließlich flüssige Diät gereicht, vorwiegend Milch, dann nur eiskalte Getränke in ganz kleinen Dosen, Mittel, welche die Reizbarkeit des Magens (Kokain) oder die allgemeine (Brom) vermindern, Orexin usw. Der Zustand blieb unverändert. Wie früher trat Erbrechen nach jedem Bissen und Schluck auf, die Kachexie vergrößerte sich immer mehr, der Puls wurde immer schneller, obwohl gut gespannt, die Temperatur erreichte nur selten 37,2° C. Die Urinuntersuchung ergab weiterhin Eiweißspuren und nicht verminderte Chlorsalze. Es wurde nun eine Ernährung per anum eingeleitet und gleichzeitig Kochsalzlösung-Eingießungen auf demselben Wege gemacht. Mit dem Momente sistierte das Erbrechen, alle anderen Symptome blieben aber unverändert. Die Urinuntersuchung ergab am 20. VIII. 1904 einen vermehrten Eiweißgehalt und im Sedimente waren neben zahlreichen Blasen- und Nierenbeckenepithelien, einzelnen körnigen Zylindern und Leukozyten, auch Kristalle von Leuzin und Tyrosin und Fettsäurenadeln zu finden. In Angesicht dieses letzten Befundes — da ich das Erscheinen von Leuzin, Tyrosin und Fettsäure im Harnsediment für ein Signum mali ominis bei Hyperemesis halte — und der Tatsache, daß jede Probe einer Ernährung per os Erbrechen von neuem hervorgerufen hat, wurde ein Abort eingeleitet. Da eine dreimalige Tamponade der Cervix und des unteren Uterussegmentes keine Geburtstätigkeit hervorzurufen imstande war und der allgemeine Zustand sich schnell verschlimmerte, wurde am 24. VIII. 1904 der Halskanal mit dem Bossischen Dilatatorium erweitert und das Fruchtei mit Abortzange und Curette entfernt. Trotzdem und trotz reichlich angewandter Exzitantien kam die Frau ad exitum etwa 24 Stunden nach der Fehlgeburt.

Die Autopsie ergab neben allgemeiner Anämie eine hochgradige fettige Degeneration aller Organe. Am meisten ausgeprägt war sie in der Leber und den Nieren, in etwas geringerem Grade im Herzmuskel und auf der inneren Seite der Aorta. Im Genitalapparat war keine Veränderung, die man zur Deutung

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der Hyperemesis auf reflektorischem Wege heranziehen könnte, zu finden, im Darmtraktus kaum eine leichte chronische Gastritis.

2. Nr. 505, 1905/6. A. M., eine 42jährige Schullehrersfrau aus Rzeszow, am 10. IV. 1906 aufgenommen. Sie hat 4mal spontan geboren, zuletzt vor 5 Jahren. Die letzte Periode Anfang Januar 1906. Von nun an Erbrechen, ohne Unterschied, ob Nahrung genommen war, oder nicht. Die Untersuchung ergab eine hochgradige Kachexie, bei kleinem, frequenten Puls und normaler Temperatur und Urin. Die schwangere Gebärmutter reichte 3 Querfinger unterhalb des Nabels, und war, wie auch die Adnexe, vollkommen frei von Veränderungen. Zeichen irgendwelcher Neurose fehlten. Hartnäckiges Erbrechen gleich nach Einnahme irgendwelcher Nahrung oder Getränk.

Angesichts der hochgradigen Kachexie wurde in diesem Falle von Anfang an eine Ernährung per clysma eingeleitet und gleichzeitig Brom, anfangs per os, später per anum gegeben. Nach 2 Tagen hat das Erbrechen aufgehört. Trotzdem ging die Kachexie immer weiter, der schnelle, leicht zu unterdrückende und veränderliche Puls wurde immer kleiner, die Temperatur blieb dauernd unter 37° C, im Urin war nur ein normaler Befund zu konstatieren. Endlich erlag Pat. am 23. IV. 1906, 9 Uhr früh.

Der künstliche Abort wurde in diesem Falle nicht eingeleitet, weil das Erbrechen aufgehört hatte und der schlechte Allgemeinzustand das nicht erlaubte.

Die Autopsie ergab eine hochgradige fettige Degeneration aller Organe, vorwiegend des Herzmuskels und eine leichte ikterische Verfärbung. Nebenbei eine Herzdilatation und allgemeine Anämie. Dazu noch Gallensteine ohne irgendwelche Komplikation. In der Gebärmutter eine 5monatliche Schwangerschaft und sonst nichts, was zur Deutung der Hyperemesis auf reflektorischem Wege herangezogen werden könnte. Eine bakteriologische Untersuchung der Milz ergab negativen Befund.

3. Nr. 587, 1905/6. M. P., eine 21jährige Unteroffiziersfrau aus Lemberg, aufgenommen am 18. V. 1906. Eine Nullipara, die Ende Februar 1906 zum letzten Male menstruiert hat. Seit dieser Zeit schwanger, litt sie an unaufhörlichem Erbrechen. Die Untersuchung ergab eine schwach gebaute, sehr heruntergekommene Person, ohne irgendwelche nervöse Erscheinungen, mit normaler Körpertemperatur und sehr schwach gespanntem Puls 132. Die schwangere, sukkulente Gebärmutter lag in Hyperanteflexion und reichte mit ihrem Grunde bis 2 Querfinger oberhalb der Schamfuge. Sonst war sie sowohl wie die Adnexe frei von pathologischen Veränderungen. Im Urin 0,05% Eiweiß, Chlorsalze verschwunden, und im Sedimente zahlreiche hyaline, körnige und mit Epithelzellen bedeckte Zylinder, Scheiden-, Blasen- und Nierenbeckenepithelzellen nebst spärlichen Leukozyten. Außerdem viel Kreatinin und ein nicht näher zu bestimmender, stark reduzierender Körper (nicht Zucker). Erbrechen trat gleich nach jedem Schlucke von Nahrung oder Trank auf.

Es wurde gleich kalte Milch, Wein und Brom per os gereicht, da sich aber der Zustand nicht besserte, schon am nächsten Tage die Kranke per rectum ernährt neben gleichzeitiger Verabfolgung von Klysmen mit einer physiologischen Kochsalzlösung. Trotzdem hörte das Erbrechen nicht auf und ein geringer Ikterus trat auf. Erst am 22. V. 1906 war der Vomitus zu Ende und in dem gesättigten Urin traten Spuren von Chlorsalzen auf. Es schien als ob es der Kranken besser ginge, nur bei Nacht warf sie sich im Bette herum und phantasierte. Am nächsten Tage hat sie schon kalte Milch vertragen, verfiel aber am 24. V. 1906 abends in einen

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Kollaps, welcher nach einigen Stunden zum Tode führte. Durch diese ganze Zeit blieb die Temperatur unter 37°, und der Puls über 120.

Die Autopsie ergab eine parenchymatöse Degeneration des Herzmuskels und eine fettige der Leber, der Intima der Aorta, und der Nieren, wo nebenbei oder vielleicht aus diesem Grunde schon eine subakute Entzündung zustande gekommen war. Im Magen eine alte, chronische Gastritis, im Uterus ein 3monatliches Fruchtei und daneben, in der Höhe des inneren Muttermundes ein etwa haselnußgroßes, gegen die Höhle wachsendes Fibrom, von alten Blutgerinnseln umgeben. Sonst keine Veränderungen im Genitalapparat.

Ein Blick auf die Symptome und den Verlauf obiger Fälle bestätigt die klinische Diagnose: Hyperemesis gravidarum und der Sektionsbefund aller, die eigentümliche Ähnlichkeit untereinander, was die hochgradigen, parenchymatösen und fettigen Degenerationen aller Organe anbetrifft. Solche Degenerationen sind meistens nach Intoxikationen mit fertigen Giften oder Toxinen infektiöser Krankheiten zu finden, so daß in einem meiner Fälle von der Milz bakteriologische Kulturen, zwecks Konstatierung einer Infektion gemacht wurden, im anderen Falle viel diskutiert war, ob nicht eine Phosphorvergiftung vorliegt. Dies ist um so wichtiger, da in diesen zwei Fällen keine Veränderungen zu finden waren, die für das Entstehen des Erbrechens verantwortlich gemacht werden könnten. Im dritten Falle lag ein Fibrom in der Gegend des inneren Muttermundes vor, das man als Ursache beschuldigen konnte. Da dies aber zur Klärung der hochgradigen Degenerationen unzureichend ist und auch nicht als Todesursache gelten kann, bleibt es höchstwahrscheinlich, daß diesem Fibrom nur eine untergeordnete Bedeutung beikommt und nach der Krankheitsursache wo anders zu forschen ist.

Es weisen also die obigen Fälle auf eine Vergiftung, mit der Sektion bestätigt, und wenn man das früher Gesagte heranzieht, auf eine Vergiftung mit Stoffwechselprodukten des Kindes, d. i. auf eine Schwangerschaftsinfektion.

Ähnliche Sektionsbefunde sind auch mehrere bekannt. Seit der Zeit, wo Lindemann einen publiziert und, theoretisch erwägend, die Meinung aufgestellt hat, daß dieses Bild sehr einer Vergiftung ähnelt, waren schon mehrere Fälle veröffentlicht, so von Duncan, Stone, Eving, Williams zehn fremde und drei eigene Fälle und alle konstatieren Organdegenerationen in dem Grade, daß sie einen Verdacht auf Vergiftung erwecken.

Das sagen Fälle, wo ein Forschen nach der Krankheitsursache auf dem Wege der Autopsie möglich war. Die anderen Ansichten basieren ihre Beweise auf guten klinischen Erfolgen. Wer nicht iurare in verba magistri will, sondern sich selbst zu überzeugen trachtet, weiß gut, wie viel höher die erste Methode steht, und daß eigentlich

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nur sie allein zur Aufklärung der Krankheit und ihrer Ursache geschaffen ist. Nur Sektionen können beweisen und diese sprechen bei der Hyperemesis für eine Vergiftung, d. h. kommen zu derselben Schlußfolgerung, zu welcher ich früher auf theoretischem Wege gelangt.

Alles zusammenfassend komme ich zur Überzeugung, daß die Hyperemesis vera als ein Komplex von, für eine Form der Schwangerschaftsinfektion im Sinne Starzewskis charakteristischen Symptomen, alle anderen Formen auf anderer Ursache beruhend, d. i. die Hyperemesis spuria, als Symptomenkomplex verschiedener anderer mit der Schwangerschaft komplizierten Krankheiten anzusehen sind. So eine Definition ist erst imstande, die Hyperemesis zu einer einheitlichen Krankheitsform zu erheben, wovon nicht nur die Symptome und der Verlauf bekannt sind, wie dies bis jetzt der Fall war, sondern welche auch eine einheitliche pathologische Anatomie und Ätiologie besitzt, d. h. alles, was von einem kompletten Krankheitsbilde verlangt wird. Und wenn noch die folgenden Jahre einen anderen, nicht mehr ein Symptom, nur das Meritum der Krankheit bezeichnenden Namen bringen oder wenigstens einen, der nichts im voraus sagt, erst dann wird mit dem Worte: Hyperemesis auch das ganze Gewirr unserer Ansichten verschwinden und ein klares, vollkommenes Krankheitsbild hervorkommen.

Schon heute will ich versuchen, ein kurzes Bild dieses Leidens zu entfalten, die früher besprochene Ätiologie, und die aus Sektionsbefunden bekannte und mit Veränderungen bei chronischer Vergiftung identische pathologische Anatomie ausgenommen.

Symptome:

Bei Schwangeren, vorwiegend Primiparen, treten fast von Anfang der Schwangerschaft, Nausea und Erbrechen auf, nüchtern oder in einiger Zeit nach dem Essen. Nebenbei die mutmaßlichen Schwangerschaftszeichen, oft gesteigert, wie Appetitlosigkeit, Verlangen nach früher nicht genossenen Speisen oder auch nach nicht genießbaren, vorwiegend nach saueren Speisen, manchmal Speichelfluß, Depressionszustände, häufiges Urinieren, Anämie, Stuhlverhaltung usw. Das Erbrechen wird immer öfter, kommt in immer kürzerem Zeitraume nach dem Essen und Trinken, so daß endlich ein Zustand eintritt, wo jeder Bissen oder Schluck Erbrechen hervorruft. Zunächst werden genossene Speisen und Getränke, später Magenschleim, oft bei leerem Magen mit Galle, oder sogar Blut vermengt, erbrochen. Gleichzeitig ist eine rasche Abmagerung und ein Sinken des Körpergewichtes zu konstatieren.

Sehr bald kommt es zu Erbrechen, ohne Rücksicht aufs Essen. Es

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tritt viele Male, bei Tag und Nacht, auf, sogar bei Kranken, die gar nichts genießen, sogar bei solchen, die per rectum ernährt werden. Zu seiner Hervorrufung reicht der Geruch von Speisen, oder sogar ein Gedanke davon, aus. Ein peinlicher Durst kommt jetzt gewöhnlich vor, wenn auch der Hunger nicht die Kranke plagt. Die Zunge findet man in diesem Stadium trocken, dicht belegt, oft bilden sich Zahnfleischgeschwüre und ein Übelriechen aus dem Munde. Nebenbei Stuhlverhaltung und nicht getrübtes Bewußtsein. Zuletzt kommt ein Kollaps, Ohnmachten, Delirien, vorwiegend bei Nacht, Halluzinationen, Sopor und endlich auch der Tod. Das Erbrechen hört sub finem auf.

Die Untersuchung ergibt durch die ganze Zeit der Krankheit fast immer einen negativen Befund. Außer einer Schwangerschaft sind keine Krankheiten der Genitalorgane zu konstatieren, auch keine im Darmtraktus, keine Neurosen, keine Allgemeinkrankheiten. Erst am Höhepunkte der Erkrankung kann man eine Vergrößerung der Leber finden und ihren runden, weichen, schmerzhaften Rand palpieren, manchmal eine Vergrößerung der Milz und endlich sub finem einen leichten Ikterus. Dies alles sind Zeichen einer so vorgeschrittenen Krankheit, daß schon hochgradige Organdegenerationen bestehen, die aller Wahrscheinlichkeit nach nicht mehr behoben werden können.

Der Temperaturverlauf sagt nichts. Gewöhnlich bleibt die Temperatur unter 37° C, ausnahmsweise steigt sie um einige Zehntelgrad höher oder fällt sub finem unter 36° C herunter.

Der Puls wird gleich von Anfang an beschleunigt und kommt bald auf 100—140. Er ist leicht veränderlich und zwar nicht nur nach physischer Arbeit, sondern auch psychischen Reizen folgend. Anfangs gut gespannt, wird er immer kleiner und weicher, bis zum fadenförmigen.

Urin immer in geringer Menge, die der genossenen und erbrochenen Flüssigkeit entspricht. Oft wird nicht mehr als 200—300 cm in 24 Stunden abgesondert. Dementsprechend ist er auch saturiert, von hohem spezifischen Gewichte, sauer, oft mit einem Satze von Uraten. Seine chemische Zusammensetzung liefert nicht viel Wichtiges. Anfangs eiweißfrei, später mit einer geringen Eiweißmenge, die immer sich vermehrt. Chlorsalze bleiben lange Zeit unverändert, trotz Verlust erheblicher Salzmengen durch Erbrechen. Eine Verminderung oder gar ein Verschwinden von Chlorsalzen im Urin beweist eine hochgradige allgemeine Kachexie. Indikan gewöhnlich vermehrt, Zucker und Azeton abwesend, Kreatin und Kreatinin manchmal in großen Mengen. Die Diazoreaktion wurde von Walzer konstatiert; ich fand sie nie in meinen Fällen und fand auch in der Literatur

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keine weiteren Berichte darüber. Im Sediment findet man mit dem Momente, wo chemisch Eiweiß zu konstatieren ist, auch hyaline, körnige und mit Epithelien bedeckte Harnzylinder, die auf Degeneration der Nieren weisen. Daneben gewöhnliche Bestandteile des Harnsedimentes. Manchmal kommen Kristalle von Leuzin, Tyrosin und Fettsäurenadeln vor, wovon ich bisher nirgends gelesen und was ich für ein sehr schlimmes Zeichen hochgradiger Fettdegenerationen halte.

Die Diagnose basiert auf der Anamnese, der Feststellung von Erbrechen gleich nach dem Essen und Trinken, oder auch ohne dem, mehrmals täglich, und einer Konstatierung des Sinkens des Körpergewichtes. Nach meiner Meinung sind das die zwei wichtigsten Symptome.

Es müssen ferner alle Erkrankungen des Intestinaltraktus, der Genitalorgane und alle Neurosen ausgeschlossen werden, d. i. alle Krankheiten, die Erbrechen direkt oder indirekt hervorrufen könnten. Nur bei Kranken, wo als Ätiologie nur einzig und allein die Schwangerschaft beschuldigt werden kann, darf die Diagnose auf Hyperemesis gravidarum vera, d. h. eine spezielle Form der Schwangerschaftsinfektion, gestellt werden.

Die Prognose ist mindestens sehr zweifelhaft, wenn nicht direkt schlecht. In Publikationen, wo die Mortalität aller Fälle berechnet wurde, wo also bei der großen Seltenheit der Hyperemesis vera mindestens $^3/_4$ nicht dazu gehören, findet man meist hohe Zahlen der Sterblichkeit. Meine drei Fälle waren alle tödlich und ich glaube, daß alle anderen, wo eine entsprechende Therapie nicht gleich von Anfang an eingeleitet wurde, auch so enden werden. Das ist aber am öftesten der Fall. Jedenfalls wird die Prognose eine schlechte, wenn schon Zeichen von Degeneration der Leber, der Nieren und des Herzens zu finden sind und eine sehr schlechte, wenn im Harnsedimente Leuzin, Tyrosin und Fettsäuren erscheinen.

Therapie. Die angezeigte, ursächliche Therapie ist bis jetzt nicht möglich, da nicht nur Antitoxine gegen das Schwangerschaftsgift unbekannt sind, sondern auch das Gift selbst. Vorläufig müssen nur die allgemeinen Maßnahmen getroffen werden, wie bei jeder Vergiftung, es muß also das Gift möglichst eliminiert oder wenigstens verdünnt und die Tätigkeit der Ausscheidungsorgane gesteigert werden. Was das erste anbetrifft, kann ich nicht viel auf Venäsektion rechnen, da dabei zusammen mit dem Gifte auch das für so schwache Patienten kostbare Blut mit verloren geht. Ausnahmsweise kann sie aber auch in Frage kommen. Dagegen sind das Blut verdünnende, subkutane oder intravenöse Infusionen physiologischer Kochsalzlösung oder auch

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Eingießungen durch den Darm sehr am Platze und können viel Gutes leisten.

Zur Hebung der Tätigkeit aller Ausscheidungsorgane sind zahlreiche pharmazeutische Präparate angegeben, wovon nach individueller Ansicht des behandelnden Arztes alle subkutan oder per anum gereicht werden können. In den meisten Fällen bringt das aber keinen Nutzen, wegen der frühzeitigen Degenerationen der Leber, Nieren usw.

Sehr wichtig ist die symptomatische Behandlung, weil sie Zeit zu gewinnen erlaubt und die Patientin bis zu dem Momente erhalten kann, wo sie entweder fähig sein wird, genügend Antitoxin zu produzieren, oder auch, wo die Schwangerschaftsinfektion von selbst erlahmt. Das wichtigste ist hier eine entsprechende Diät bei ganz ruhiger Lagerung auf dem Rücken. Am besten vertragen die Kranken kalte Flüssigkeiten, oft und in kleinen Dosen gereicht, und zwar am liebsten kalte, süße Milch. In anderen Fällen wird ausgezeichnet kalte Limonade, Kognak, Wein oder Champagner vertragen. Ich sah einmal einen momentanen, obwohl vorübergehenden, Effekt, nach löffelweiser Darreichung einer Mischung von ausgestandenem Sodawasser mit saurem Wein. Nachhelfen kann man mit pharmazeutischen Präparaten wie Tra. amara, Tra. Chinae, Tra. nucis vomicae, Kokain in Tropfen usw., oder durch Einführung größerer Bromdosen per rectum. Ich wiederhole aber ausdrücklich, daß ich alle diese Mittel nicht als Heilmittel ansehe, sondern als Präparate, welche die Reizbarkeit des Magens oder die allgemeine vermindern, ohne auf die, als Grundursache bestehende, Schwangerschaftsinfektion einen Einfluß zu haben.

Es bleibt nur noch die Frage einer künstlichen Unterbrechung der Schwangerschaft zu besprechen. In der Überzeugung, daß die Hyperemesis Folge einer Vergiftung der Mutter durch Stoffwechselprodukte der Frucht ist, muß ich den künstlichen Abort als Regel aufstellen, weil es schwer anzunehmen wäre, daß entweder das Ei, welches so viel Gift produziert, daß dies eine schwere Erkrankung der Mutter hervorruft, diese Produktion einstellen könnte, oder daß die Patientin die nötige Menge von Antitoxinen bei ihrer Erschöpfung bilden könnte, wenn sie dies früher bei noch guten Kräften nicht imstande war. Dieser Behandlung steht aber die Tatsache im Wege, daß wir gegenwärtig nicht gleich die Hyperemesis vom physiologischen Erbrechen Schwangerer zu unterscheiden wissen, erst wenn das Leiden vollkommen ausgebildet ist. Und auch jetzt muß noch etwas Zeit verloren gehen, um zu erfahren, ob das Erbrechen nicht Folge einer Magen- oder Darmkrankheit, einer Neurose usw. ist, und um eine Reihe empirischer Mittel durchzuprobieren, da wir bei heutigem Stande

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der Wissenschaft noch nicht positiv die Schwangerschaftsinfektion beweisen können. Gewöhnlich klären das Leiden erst Zeichen von Organdegenerationen, wie Vergrößerung der Leber, Ikterus, Eiweiß und Zylinder im Urin, Herzschwäche usw. auf, leider schon zu spät. Ein Abort in diesem Stadium ist fast immer nutzlos, weil die Degenerationen wichtiger Organe schon selbst auch zum Tode führen. Die Sache verhält sich um so schlimmer, als die Frauen an Schwangerschaftserbrechen so gewöhnt sind, daß sie viel zu spät sich melden, wo eigentlich schon gar nichts mehr zu tun ist.

Wenn ich nun zum Schlusse meine Therapie der Hyperemesis, wie ich sie in der Praxis durchführe, angeben wollte, so möchte ich folgendes Schema aufstellen: Wenn irgendwelche Krankheiten verschiedener Organe, welche Symptome einer Hyperemesis spuria hervorrufen können, vorliegen, so müssen zunächst diese möglichst bald zum Schwinden gebracht werden, z. B. Erosionen durch Ausbrennen oder mit Tra. jodi fortior, Retroflexion durch Heben der Gebärmutter und deren Fixieren mit einem Pessar, Neurosen durch große Bromdosen usw. und gleichzeitig sehr strenge Diät, eventuell auch Ernährung per clysma verordnet werden. Hilft dies in kurzer Zeit nicht, oder sind keine Krankheiten im Körper zu finden, die eine Hyperemesis vera wahrscheinlich machen, so ist gleich eine Ernährung per rectum am Platze mit gleichzeitigen Eingießungen physiologischer Kochsalzlösung oder subkutanen Infusionen, und wenn dies ohne Erfolg und es die Kräfte der Patientin erlauben, ein künstlicher, möglichst rascher Abort. Es ist dies das letzte Mittel, das aber bei schon bestehenden Degenerationen auch sehr problematisch wirkt. Als ärgstes Zeichen betrachte ich die Konstatierung von Leuzin, Tyrosin und Fettsäuren im Urinsediment, weil dies für hochgradige Verfettung der Organe, wie bei Atrophia hepatis flava spricht.

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486.

(Chirurgie Nr. 141.)

Ergebnisse funktioneller Magenuntersuchungen bei Gastroenterostomierten hinsichtlich der Früh- und Spätresultate[1]).

Von

Stabsarzt Dr. Neuhaus,

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Die Fragen, ob nach einer Gastroenterostomie Galle allein oder zusammen mit Pankreassaft im Magen vorkommt, ob dieses Vorkommen ein dauerndes oder nur zeitweiliges ist, ob und in welchen Quantitäten die beiden Drüsensekrete im Magen vertragen werden, beantworten nicht alle Autoren in gleichem Sinne.

Wir haben deshalb an unserem ziemlich reichlichen Gastroenterostomie-Material versucht, einen Beitrag zur Klärung dieser Fragen zu liefern, und einen Teil der gastroenterostomierten Patienten nachuntersucht. Dabei kam es uns vor allem darauf an, nicht nur kurze Zeit nach Anlegung der Gastroenterostomie eine funktionelle Magenprüfung vorzunehmen, sondern gerade diejenigen Fälle erweckten unser besonderes Interesse, bei denen die Operation schon längere Zeit zurücklag. Daraus ergibt sich von selbst, daß wir unsere Untersuchungen mit wenigen Ausnahmen nur an solchen Patienten ausgeführt haben, welche wegen gutartiger Leiden gastroenterostomiert worden sind. Infolge der enormen Fluktuation des Gros der Berliner Bevölkerung ist es leider nicht immer gelungen, der früheren Patienten wieder habhaft zu werden; zum Teil bekamen wir auch von ihnen die Erklärung, daß sie sich seit der Operation völlig wohl fühlten und deshalb keinen Grund hätten, eine funktionelle Magenuntersuchung vornehmen zu lassen; sie scheuten sich eben vor der Einführung des Magenschlauches.

Wir haben infolgedessen von unserem reichen Gastroenterostomie-

1) Aus der chirurgischen Universitätsklinik der Königl. Charité zu Berlin Direktor: Geheimrat Professor Dr. O. Hildebrand.

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Material nur 17 Fälle nachuntersuchen können. 15 von diesen hatten ein benignes Magenleiden; in 2 Fällen bestand ein Carcinoma ventriculi. Der Zeitraum, welcher bei den einzelnen Patienten vom Tage der Anlegung der Gastroenterostomie ab bis zur Nachuntersuchung verstrichen war, differiert sehr. Das Maximum betrug 5 Jahre, das Minimum 3 Wochen. Entweder ist die Gastroenterostomia antecolica anterior mit Suspension der zuführenden Schlinge ausgeführt, oder eine Braunsche Enteroanastomose der Gastroenterostomie ohne Suspension hinzugefügt worden.

Um Wiederholungen zu vermeiden, und um eine Beurteilung und etwaige Nachprüfung der in den nachfolgenden Tabellen aufgeführten Ergebnisse zu ermöglichen, geben wir zunächst einige Erläuterungen, in welcher Weise bei den Untersuchungen verfahren worden ist.

Wir haben zwei Arten von Probemahlzeiten gegeben; die eine entspricht etwa der von Sahli angegebenen und enthält sehr viel Fett: 500 g Butter, 250 g Mehl, 300 g Wasser und etwas Salz wurden auf dem Herdfeuer miteinander vermengt und gekocht, so daß eine dünnflüssige, graubraune Suppe daraus entstand. Von dieser sehr fettreichen Suppe wurden etwa 400 ccm verabreicht; die andere Probemahlzeit entsprach der nach Ewald benannten: 1 Becher Tee, ca. 350 ccm, und 1 Schrippe. Die Probemahlzeit wurde entweder morgens gegen $8^1/_2$ Uhr nüchtern gegeben, nachdem am Abend vorher bei der Abendmahlzeit gegen 6 Uhr etwa 10—12 Backpflaumen gereicht worden waren. Zeigten sich nun morgens beim Aushebern, was ca. $1^1/_2$ Stunden nach der Probemahlzeit geschah, noch Reste von den Pflaumen, so wurde das Ergebnis dieser Probemahlzeit nicht verwertet, sondern an einem anderen Tage nochmals eine Probemahlzeit gereicht, aber nunmehr vorher der Magen ausgewaschen. Wir sind uns sehr wohl bewußt, daß eine absolut sichere Kontrolle, ob der Magen leer ist, lediglich dadurch geleistet werden kann, daß mit dem Magenschlauch ausgehebert und ausgewaschen wird. Da es uns aber im wesentlichen auf den Nachweis von Galle und Pankreassaft im Magen überhaupt ankam, und nicht auf eine ganz genaue Untersuchung des Magensaftes, so haben wir uns im Gebrauch der immerhin nicht angenehmen Schlundsonde im Interesse der Untersuchten auf das allernötigste beschränkt. Übrigens kann man wohl mit Recht für die meisten Fälle annehmen, daß der Magen leer ist, wenn man morgens von den abends vorher verabreichten Backpflaumen beim Aushebern nichts mehr findet, da erfahrungsgemäß Backpflaumen relativ schwer den Pylorus passieren. Für unsere Zwecke genügte die eingeschlagene Methode völlig; zum einwandsfreien Studium der Magensaftsekretion dürfte allerdings wohl eine Auswaschung des Magens vor dem Probefrühstück unerläßlich sein.

Zur Bestimmung der Motilität des Magens haben wir das von Matthieu angegebene Verfahren zu Hilfe genommen. Es besteht das bekanntlich darin, daß man nach dem Aushebern dem Patienten durch den Magenschlauch gleich nochmals 300 ccm Wasser in den Magen bringt, das Wasser im Magen möglichst verteilt und dann exprimieren läßt. Dann wird der Grad der Gesamtazidität sowohl des unverdünnten, wie des mit 300 ccm Wasser verdünnten Mageninhalts in der üblichen Weise mit $^1/_{10}$ Normalnatronlauge unter Anwendung von Phenolphtalein als Indikator bestimmt; darauf wird nach der Formel $x = \frac{300 \cdot b}{a - b}$ der im Magen

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verbliebene und durch Aushebern nicht gewonnene Rest der Probemahlzeit berechnet, wobei *a* den Aziditätsgrad des unverdünnten, *b* den des mit 300 ccm Wasser verdünnten Mageninhaltes bedeutet. Die Matthieusche Formel ist allerdings bezüglich ihrer absoluten Zuverlässigkeit angegriffen worden. Jedenfalls gibt sie die Möglichkeit einer annähernden Bestimmung des im Magen verbliebenen Restes der Probemahlzeit und ermöglicht somit einigermaßen die Beurteilung der Magenfunktion hinsichtlich der Motilität. Man kennt dann nämlich die Menge — in Kubikzentimetern ausgedrückt — der durch Aushebern direkt zurückgewonnenen Probemahlzeit und, nach der Matthieuschen Formel berechnet, auch den Rest — ebenfalls in Kubikzentimetern ausgedrückt — der nicht durch Aushebern zurückgewonnenen, aber noch im Magen vorhandenen Probemahlzeit. Beide Zahlen addiert man und zieht ihre Summen von der ebenfalls bekannten Menge der gereichten Probemahlzeit ab; die sich ergebende Zahl muß die Menge der in den Darm beförderten Probemahlzeit angeben.

Zum Nachweis des Pepsins und seiner Wirkung haben wir uns des Mettschen Verfahrens bedient. Dünne Glasröhrchen von 1—2 mm Dicke wurden mit flüssigem Hühnereiweiß durch Ansaugen möglichst gleichmäßig unter Vermeidung von Luftblasen gefüllt; in siedendem Wasser wurde das Eiweiß zum Gerinnen gebracht. Kleine Stücke von diesem mit Eiweiß gefüllten Röhrchen wurden dann in der auf Pepsin zu untersuchenden Flüssigkeit in den Brutschrank (37°) gestellt; nach 24 Stunden konnte man an den Röhrchen die Menge des verdauten Eiweiß ablesen. Wir haben fast immer 2 Schälchen mit Mageninhalt zur Untersuchung auf Pepsin angesetzt, eins ohne Salzsäurezusatz, eins mit einem Zusatz von Salzsäure, und zwar so viel, daß Congo deutlich positive Reaktion gab.

Mit Hilfe des von P. Bergell[1]) aus der Seidenraupe dargestellten Seidenpeptons waren wir in der Lage, in sehr exakter Weise den Mageninhalt auf Pankreassaft zu untersuchen. Die Methode hat sich uns als äußerst scharf und brauchbar erwiesen; sie hat mit absoluter Eindeutigkeit den Nachweis von Pankreassaft führen lassen, wo die sonst bekannten Verdauungsproben ein keineswegs sicheres und zweifelsfreies Resultat ergeben haben. Wir haben nämlich, als wir unsere Untersuchungen anfingen, neben der Probe mit Seidenpepton regelmäßig auch eine Probe mit Blutfibrin angesetzt. Dabei hat sich nach unseren Erfahrungen die Überlegenheit der Bergellschen Seidenpeptonprobe, was Exaktheit und Sicherheit in der Beurteilung anbetrifft, einwandsfrei ergeben. Bergell hat nämlich gefunden, daß das im Pankreassaft vorhandenes Trypsin, das Eiweiß verdauende Ferment des Pankreassaftes, mit dem Seidenpepton in alkalischer Lösung, am besten bei Brutschranktemperatur, Tyrosin bildet. Wenn man also zu einer auf Pankreassaft zu untersuchenden Flüssigkeit Seidenpepton zusetzt — etwa 5 ccm Untersuchungsflüssigkeit und 0,5 g Seidenpepton — und die Mischung nach Alkalisierung mit Ammoniak oder Natron bicarb. für 24—36 Stunden einer Temperatur von 37° C aussetzt, so ist beim Nachweis von Tyrosinkristallen der Beweis geliefert, daß die zu untersuchende Flüssigkeit Trypsin, also Pankreassaft, enthält. Gewöhnlich haben wir die Kristalle schon nach 12—16 Stunden nachweisen können. Mit dem im Magensaft vorhandenen, Eiweiß verdauenden Fermente bildet das Seidenpepton kein Tyrosin, auch dann nicht, wenn man den Magensaft stark alkalisch macht. Wir haben bei 15 Patienten, die nicht

1) Herr Professor Bergell war so liebenswürdig, mir eine Portion des von ihm dargestellten, im Handel schwer erhältlichen Fibrinseidenpeptons für diese Versuche zur Verfügung zu stellen, wofür ich auch an dieser Stelle nochmals bestens danke.

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wegen eines Magenleidens, sondern aus irgendeinem anderen Grunde sich auf der Klinik in Behandlung befanden, daraufhin Versuche angestellt, ohne jemals die Bildung der Tyrosinkristalle feststellen zu können. Die Untersuchung auf Tyrosinkristalle geschieht ohne Mühe mit Hilfe des Mikroskops. Man bringt einige Platinösen von dem Filtrat des in vorstehender Weise behandelten Mageninhalts auf einen Objektträger unter das Mikroskop und sieht meist auf den ersten Blick in sehr schöner, absolut einwandsfreier Weise die Tyrosinkristalle in ihrer typischen Form, deutlicher wie sie in irgendeinem Lehrbuch abgebildet sind. Am leichtesten waren sie meist am Rande des Deckglases zu finden. Es sind feine Nadeln, die einen gelblichen Schimmer haben; dieser tritt am deutlichsten in Erscheinung, wenn die Nadeln dicht beieinander liegen; dann sind sie in Büschel- oder Doppelbüschelform angeordnet; oft bilden sich auch Rosetten. Um uns vor Verwechselung mit Fettsäurenadeln zu schützen, ein Einwand, der ja in Anbetracht der reichlich Fett enthaltenden Sahlischen Probesuppe gemacht werden könnte, haben wir, wenn irgendwelche Zweifel auftauchten, durch Zusatz von Reagenzien die Sachlage geklärt. Tyrosin ist in heißem Wasser, Ammoniak und verdünnter Salz- oder Salpetersäure leicht löslich, unlöslich in Äther und Alkohol; Fettsäurenadeln werden aber durch Äther und erwärmten Alkohol aufgelöst, während Säuren sie nicht angreifen.

Der Nachweis von Galle war gewöhnlich schon aus dem bloßen Anblick des Ausgeheberten zu stellen, wir haben aber immer mittels der Gmelinschen Probe auf Gallenfarbstoffe eine genauere Kontrolle ausgeübt.

Wir lassen nun, in Gruppen angeordnet, Übersichtstabellen von den Ergebnissen unserer Untersuchungen folgen.

Die Gruppe A umfaßt 7 Patienten, bei denen eine Gastroenterostomia anterior antecolica gemacht worden ist. Der Zeitraum zwischen der Anlegung der Magendarmfistel und der funktionellen Magenuntersuchung variiert zwischen 5 Jahren und $1^1/_2$ Monaten. Wenn wir zunächst einen Blick auf die in der Tabelle stark umrahmten Spalten 8 und 9 werfen, welche das Untersuchungsergebnis über Beimengung von Galle und Pankreassaft zum Mageninhalt enthalten, so finden wir, daß sich bei diesen sämtlichen 7 Fällen Galle und auch Pankreassaft im ausgeheberten Mageninhalt hat nachweisen lassen. Wenn wir sagen „Pankreassaft", so ist das in gewissem Sinne nicht ganz exakt ausgedrückt, indem es richtiger Trypsin heißen müßte. Da aber das Trypsin einen wesentlichen, integrierenden Bestandteil des Pankreassaftes ausmacht, so dürfte mit dem Nachweis von Trypsin der Beweis für die Anwesenheit von Pankreassaft überhaupt als geliefert zu erachten sein. Bei den meisten dieser Patienten haben wir die Magenuntersuchung zweimal vorgenommen; es hat sich nie eine Differenz zwischen den beiden Untersuchungen hinsichtlich der Beimengung von Galle und Trypsin ergeben.

Ganz anders gestaltet sich das Untersuchungsresultat bei den unter Gruppe B untergebrachten 4 Patienten, bei denen auch eine einfache Gastroenterostomia anterior anticolica angelegt worden ist.

Wir sehen hier in den beiden stark umrahmten Spalten, welche

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den Befund über Galle und Trypsin im Mageninhalt enthalten, immer einen negativen Ausfall der Untersuchung verzeichnet; auch hier ist mit einer Ausnahme in jedem Falle zweimal untersucht worden; ein Unterschied zwischen den beiden Untersuchungen ist niemals vorhanden gewesen. Der Zeitraum zwischen Anlegung der Magendarmfistel und der Nachuntersuchung schwankt zwischen 2 Jahren 3 Monaten und 1 Jahr 4 Monaten.

Die dann folgende Gruppe C weicht hinsichtlich der Operation etwas von den beiden anderen ab; während bei Gruppe A und B nur eine einfache Gastroenterostomia anterior antecolica angelegt worden ist, haben wir bei den unter dieser Gruppe verzeichneten Fällen eine Braunsche Enteroanastomose hinzugefügt.

Betrachten wir zunächst den III. und IV. Fall auf Tabelle C. Beide haben in der Rubrik 8 und 9 ein positives Ergebnis zu verzeichnen; bei beiden ist also Galle und Pankreassaft im Magen nachweisbar gewesen. Wir möchten aber hervorheben, daß die Untersuchung relativ kurze Zeit nach der Operation vorgenommen worden ist (3 Wochen und $1^1/_2$ Monate post op.). Bei dem Fall II ist in Abständen von einem oder zwei Monaten im ganzen fünfmal untersucht worden; die erste Untersuchung fand einen Monat, die letzte acht Monate nach der Operation statt. Während nun bei den ersten vier Untersuchungen immer Galle und Trypsin im Mageninhalt nachweisbar waren, fehlte beides bei der letzten, acht Monate nach der Operation vorgenommenen Untersuchung. Beachtenswert ist auch bei Fall II die Rubrik 7; bei den ersten vier Untersuchungen fehlte stets freie Salzsäure, bei der fünften dagegen, wo Galle und Trypsin nicht nachweisbar waren, fand sich freie Salzsäure. Bei Fall I hat die Untersuchung alle vier Male keine Galle und kein Trypsin ergeben; die erste Untersuchung wurde drei Monate post op. ausgeführt.

In der letzten Gruppe D sind zwei Fälle untergebracht, bei denen der Pylorus völlig geschlossen ist; die einzige Verbindung zwischen Magen und Darm besteht also an der Gastroenterostomie-Stelle.

Bei dem Fall I handelte es sich um eine ausgedehnte Ätzstriktur am Pylorus. Die Patientin hatte ein Conamen suicidii mit roher Salzsäure begangen; sie hatte sich den Ösophagus und auch den Magen ganz erheblich verätzt. Während sie bei uns wegen ihres Ösophagus auf der Klinik behandelt wurde, entwickelte sich unter unseren Augen der völlige Verschluß des Pylorus. Der Magen nahm eine enorme Dilatation an; er stand schließlich mit seiner großen Kurvatur an der Symphyse und war wie eine große, die ganze Bauch-

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Gruppe

	1 Wann nachuntersucht?	2 Probemahlzeit	3 Farbe und Geruch
I. Arbeiter S. Vor 5 Jahren außerhalb wegen chronischer Magenbeschwerden mit Stauungszuständen GE. ant. antecol. mit Murphyknopf.	5 Jahre post op.	vor Relaparotomie Dezember 1907 Sahli	grünlich ranzig
Dezember 07 auf der Klinik. Relaparotomie wegen Ektasie des Magens. Stauungsbeschwerden. Murphyknopf im Magen. Anastomosenöffnung für kleinen Finger nicht durchgängig. Knopf entfernt.		nach Relaparotomie Februar 1908 Sahli	grünlich ranzig
II. Arbeiter G. Traumatische Pylorusstenose; dauernd Erbrechen, Ektasie des Magens ziemlich stark. 5. IV. 07 GE. ant. antecol.	2 Jahre 9 Monate post op.	16. XII. 07 Sahli	grünlich ranzig
III. Frau S. Wegen Dilatatio ventriculi mit Stauungsbeschwerden am 15. I. 06 GE. ant. antecol.	2 Jahre post op.	8. VII. 07 Sahli 17. I. 08 Sahli	grünlich ranzig grünlich ranzig
IV. Frau R. Dilatatio ventriculi mit chronischen Magenbeschwerden (Erbrechen). 19. I. 06 GE. ant. antecol.	2 Jahre post op.	15. I. 08 Sahli 10. II. 08 Sahli	grünlich ranzig grünlich ranzig
V. Frau Gl. Sanduhrmagen und Dilatatio ventriculi, chronische Magenbeschwerden auf dem Boden eines Ulcus. 12. IV. 07 GE. ant. antecol.	3 Monate post op.	15. VII. 07 Sahli 17. VII. 07 Ewald	grünlich ranzig grünbräunlich sauer
VI. Frl. H. Carc. pylori. 20. VII. 07 GE. ant. antecol. Zur Resektion ungeeignet.	$1^1/_2$ Monate post op.	4. IX. 07 Sahli	grünlich ranzig
VII. Frau G. Carc. pylori; zur Resektion ungeeignet, deshalb GE. ant. antecol. am 3. X. 07.	$1^1/_2$ Monate post op.	15. XII. 07 Sahli	grünlich ranzig

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A.

4 Motilität des Magens	5 Reaktion mit Lackmus	6 Gesamt-azidität	7 Freie HCl	8 Gmelinsche Probe	9 Trypsin-nachweis	10 Pepsinnachweis mit HCl-Zusatz	10 Pepsinnachweis ohne HCl-Zusatz
schlecht	sauer	30	+	+	+	+	+
besser	sauer	8	—	+	+	+	—
gut	neutral	10	—	+	+	+	—
schlecht	sauer	20	—	+	+	—	—
mäßig	sauer	8	—	+	+	+	—
mäßig	sauer	35	—	+	+	+	—
mäßig	sauer	30	—	+	+	+	—
gut	sauer	14	—	+	+	nicht geprüft	
gut	sauer	36	+	+	+	+	+
schlecht	schwach sauer	10	—	+	+	nicht geprüft	
schlecht	sauer	20	—	+	+	+	—

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Gruppe B.

	1 Wann nachuntersucht	2 Probemahlzeit?	3 Farbe und Geruch des Ausgeheberten?	4 Motilität	5 Reaktion mit Lackmus	6 Gesamtazidität	7 Freie HCl	8 Gmelinsche Probe	9 Trypsinnachweis	10 Pepsinnachweis mit HCl Zusatz	10 Pepsinnachweis ohne HCl Zusatz
I. Frau R. Stauungsbeschwerden Erbrechen infolge Enteroptose und Dilatation des Magens. 24. X. 05 GE. ant. antecol.	2 Jahre 3 Monate post op.	*15. I. 08* Sahli	bräunlich ranzig säuerlich	sehr gut	sauer	nicht bestimmbar, weil zu wenig Mageninhalt erhalten	—	—	—	+	—
		22. I. 08 Ewald	bräunlich sauer	sehr gut	sauer		+	—	—	+	+
II. Frau L. B. Chronische Ulcusbeschwerden, Schmerzen, wiederholte Blutungen. 19. IV. 05 GE. ant. antecol.	2 Jahre 2 Monate post op.	*7. VI. 07* Sahli	bräunlich ranzig säuerlich	sehr gut	sauer	nicht bestimmbar, da zu wenig Mageninhalt erhalten	—	—	—	nicht bestimmbar, da zu wenig Mageninhalt erhalten	
		12. VI. 07 Ewald	graubraun sauer	sehr gut	sauer		+	—	—		
III. Frau K. Chronische Magenbeschwerden mit Erbrechen. 12. II. 06 GE. ant. antecolic. Magen ektatisch und ptotisch Adhärenzen (Ulcusreste)	1 Jahr 5 Monate post op.	*6. VI. 07* Sahli	bräunlich ranzig säuerlich	mittelmäßig	sauer	44	—	—	—	+	+
		16. VII. 07 Ewald	braun sauer	mittelmäßig	sauer	50	+	—	—	+	+
IV. Frau E. Chronische Magenbeschwerden. 30. VII. 06 GE. ant. antecol. Sanduhrmagen; talergroße, harte mit Pankreas verwachsene Stelle an der hinteren Fläche Karz. oder Ulcustumor zunächst zweifelhaft; zur Zeit	1 Jahr 4 Monate post op.	Sahli	bräunlich ranzig sauer	gut	sauer	45	+	—	—	+	+

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	1 Wann nachuntersucht?	2 Probemahlzeit	3 Farbe und Geruch des Ausgeheberten	4 Motilität des Magens	5 Reaktion mit Lackmus	6 Gesamtazidität	7 Freie HCl	8 Gmelinsche Probe	9 Trypsinnachweis	10 Pepsinnachweis mit HCl-Zusatz	10 Pepsinnachweis ohne HCl-Zusatz
I. Frau Ri. Wegen chronischer Ulcusbeschwerden 8. III. 07 GE ant. antecolic.; chronischer Circulus vitiosus; Relaparotomie 23. IV. 07 Braunsche Anastomose.	11 Monate post op.	14. XII. 07 Sahli	graubraun ranzig	gut	sauer	20	—	—	—	+	+
		17. VII. 07 Ewald	braun sauer	gut	sauer	40	+	—	—	+	+
		18. VII. 07 Sahli	graubraun ranzig	gut	sauer	56	+	—	—	+	+
		17. II. 08 Sahli	graubraun ranzig	gut	sauer	50	+	—	—	+	+
II. Frl. Sch. Chronische Ulcusbeschwerden 3. VIII. 07 GE ant. antecol. mit Braunscher Anastomose	8 Monate post op.	3. IX. 07 Sahli	grünlich ranzig	mäßig	sauer	30	—	+	+		
		29. XII. 07 Sahli	grünlich ranzig	mäßig	sauer	28	—	+	+		
		3. I. 08 Sahli	grünlich ranzig	besser	sauer	nicht untersucht, da zu wenig Mageninhalt	—	+	+		
		4. II. 08 Sahli	grünlich ranzig	gut	sauer		—	+	+		
		2. V. 08 Sahli	graubraun ranzig	sehr gut	stark sauer		+	—	—		
III. Arbeiter H. Carc. pylori. GE ant. antecol. mit Braunscher Anastomose	$1^1/_2$ Monate post op.	Sahli	grünlich ranzig	schlecht	sauer	15	—	+	+	+	—
IV. Arbeiter J. Dilatio ventriculi GE ant. antecol. mit Braunscher Anastomose	3 Wochen post op.	Sahli	grünlich ranzig	gut	sauer	20	—	+	+	+	—

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Gruppe D.

	1 Wann nachuntersucht?	2 Probemahlzeit	3 Farbe und Geruch des Ausgeheberten	4 Motilität des Magens	5 Reaktion mit Lackmus	6 Gesamtazidität	7 Freie HCl	8 Gmelinsche Probe	9 Trypsinnachweis	10 Pepsinnachweis mit HCl-Zusatz	10 Pepsinnachweis ohne HCl-Zusatz
I. Frl. Ch. S. Völliger Verschluß des Pylorus durch Narbe nach HCl-Ätzung (Conamen suicidii). Enorme Dilatatio ventriculi; fast bis zur Symphyse. 6. V. 07 GE. ant. antecol.	9 Monate post op.	*6. VII. 07* Sahli	grünlich ranzig	schlecht	neutral	10	—	+	+		
		19. VII. 07 Sahli	grünlich ranzig	schlecht	schwach sauer	20	—	+	+		
		15. I. 08 Ewald	grünlich ranzig	gut	sauer	20	—	+	+		
		8. II. 08 Sahli	grünlich ranzig	gut	schwach sauer	11	—	+	+		
II. Arbeiter CH. Carc. pylori. Pylorusresektion u. GE. ant. antecol. (Billroth II.) 3. VII. 07.	6 Monate post op.	7. I. 08 Sahli	grünlich ranzig	gut	schwach sauer	11	—	+	+		

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höhle ausfüllende, ziemlich prall gespannte Zyste durch die dünnen Bauchdecken palpabel. Bei der Operation zeigte sich, daß der Pylorus völlig undurchgängig war. Die Magenwand selbst war sehr verändert; sie war enorm brüchig; die Fäden schnitten bei der Naht sehr leicht durch; die Serosa zeigte entzündliche Veränderungen; sie war verdickt und spiegelte nicht wie es normalerweise sein muß. Es wurde in diesem Falle eine besonders große Anastomosenöffnung angelegt. In dem II. Fall ist der Pylorus wegen Karzinoms reseziert worden. Die Resektionsstelle wurde völlig geschlossen und eine Gastrojejunostomie angelegt, also nach Billroth II operiert. Die einzige Passage zwischen Magen und Darm bestand also wiederum nur an der Anastomosenöffnung. Bei beiden Patienten hat die funktionelle Magenuntersuchung dauernd Galle und Trypsin im Mageninhalt ergeben. Die Untersuchungen sind nicht etwa gleich nach der Operation vorgenommen worden, sondern haben bis zu 9 Monaten nach dem operativen Eingriff stattgefunden.

Was lehren uns nun unsere Untersuchungsergebnisse?

Wir haben bei unseren Fällen in den ersten Wochen und Monaten nach der Gastroenterostomie stets Galle und Pankreassaft — wenn wir den Nachweis des Trypsins für identisch mit dem Nachweis des Pankreassaftes ansehen — im Magen gefunden, das heißt bei der Gastrojejunostomia anterior antecolica mit und ohne Anastomose Brauns. Hinsichtlich der Gastrojejunostomia retrocolica posterior stehen uns eigene Erfahrungen nicht zur Seite, da wir diese Methode nur selten üben; es ist aber nicht einzusehen, warum die Verhältnisse bei dieser Methode anders liegen sollten als bei der anterior. Auch über die sogenannten Y-Methoden haben wir keine eigenen Erfahrungen. Wir machen für gewöhnlich weder die von Roux[1]) besonders geübte Gastrojejunostomia ypsiliformis retrocolica posterior, noch die von Rotgans[2]) vor einiger Zeit wieder empfohlene Gastrojejunostomia ypsiliformis antecolica anterior, eine Operation, welche übrigens schon 1883 von Wölfler[3]), allerdings nur an Hunden, ausprobiert und für bestimmte Fälle angegeben worden ist. Die Doyensche Methode[4]) der Gastroenterostomie, welche darin besteht, daß in einer Sitzung eine Magenjejunumfistel, eine Enteroanastomose zwischen zu- und abführendem Schenkel und schließlich noch eine Resektion der zuführen-

1) Kocher, Chirurg. Operationslehre. 1907. S. 859.

2) Rotgans, Gastroenterostomia ypsiliformis antecolica anterior. Wien. klin. Rundschau 1904 und Nederl. Tijdschrift vor Genesk. 1902, I, no. 23. Ref. Zentralbl. 1902, S. 1100.

3) Wölfler, Vortrag auf dem Chirurgenkongreß 1883.

4) Doyen, Revue de chirurgie 1897, p. 1030.

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den Darmschlinge gemacht wird, schafft dieselben Zirkulationsverhältnisse für den Darminhalt wie die Y-Methoden und dürfte infolgedessen, was das Einfließen von Galle und Pankreassaft in den Magen anbetrifft, denselben Effekt haben wie die Y-Methoden. Wegen ihrer Kompliziertheit und der zeitraubenden Resektion des zuführenden Schenkels ist sie aber nicht zu empfehlen. Bei der Gastroduodenostomia wird sich ebenfalls kein Darmsaft im Mageninhalt finden lassen, es sei denn, daß Doyen mit seiner Behauptung, daß der Duodenalsaft auch rückläufig in den Magen fließen kann, recht hat.

Daß bei den Y-Methoden das Einfließen von Galle und Pankreassaft in den Magen fortfällt, ist durchaus plausibel. Wenn sich, wie in einem von Kaiser[1]) mitgeteilten Falle, dabei doch Galle im Mageninhalt finden sollte, so ist das nicht anders zu erklären, als daß durch antiperistaltische Bewegungen des Darmes sein Inhalt in den Magen getrieben wird. Für gewöhnlich bilden die Y-Methoden ein Hindernis für das Einfließen von Darminhalt in den Magen. Das beweisen auch die in der Literatur niedergelegten klinischen Beobachtungen. Sehr instruktiv ist in dieser Hinsicht die Leidensgeschichte eines fünfmal laparotomierten Kollegen, welche Tavel[2]) mitteilt. Im November 1896 wurde von Sick bei dem Herrn wegen Hyperchlorhydrie zunächst eine Gastroenterostomia antecolica isoperistaltica nach Kocher an der großen Kurvatur in der Pars pylorica angelegt. Sehr bald stellten sich Erscheinungen ein, welche auf ein mangelhaftes Funktionieren der Magenfistel schließen ließen. Es bestand Anorexie, dauernd fader, pappiger Geschmack, Aufstoßen und Übelkeiten. Es wurde deshalb im Januar 1897 von Mikulicz konsultiert, welcher relaparotomierte und zur Beseitigung der beschriebenen Beschwerden eine Braunsche Anastomose anlegte; nebenbei machte er eine Gastroenterostomoplastik, da die Magendarmfistel nur noch bleistiftdick war. Es trat jedoch danach keine Besserung des Zustandes ein.

Kausch[3]) erwähnt übrigens diesen Patienten in seiner Arbeit aus der Mikuliczschen Klinik als Beweis für die allerdings nur selten, aber doch mitunter vorkommenden Fälle, in denen das Einfließen von Galle in den Magen schwere Schädigungen mit sich bringt, und in denen es auch durch weitere Eingriffe nicht gelingt, den Gallenfluß in den Magen zu beseitigen.

Die anfängliche Besserung nach der von v. Mikulicz vorgenom-

1) O. Kaiser, Über die Erfolge der Gastroenterostomie. Deutsch. Zeitschr. für Chirurgie Bd. 61.

2) Tavel, Le reflux dans la gastroentérostomie. Revue de chirurg. 1901, no. 12.

3) Kausch, Über funktionelle Ergebnisse nach Operationen am Magen. Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie. 4. Bd. 1899.

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menen Operation hielt nämlich nur kurze Zeit an, so daß im Juni 1897 Kocher wegen der genannten Beschwerden zu Rate gezogen wurde. Es wurde wiederum die Abdominalhöhle geöffnet. Man fand die zuführende Schlinge, welche unter ziemlich spitzem Winkel in den Magen einmündet, als etwas zu kurz; ihr Mesenterium komprimierte das Colon transversum. Infolgedessen wurde das Mesenterium der zuführenden Schlinge gedehnt und eine Fältelung und Einengung der letzteren nach v. Hacker vorgenommen, um auf diese Weise das Einfließen von Darmsaft in den Magen zu erschweren. Die Besserung der alten Beschwerden war nur sehr gering, so daß schon zwei Monate später zum vierten Male zur Laparotomie geschritten werden mußte. Dieses Mal trennte Tavel den Pylorus von dem Duodenum, und als hiernach noch immer keine Beseitigung der geschilderten Magenbeschwerden eintrat, resezierte Tavel in einer fünften Sitzung die zuführende Jejunumschlinge. Nunmehr verschwanden die Beschwerden endgültig, nachdem also ein Zustand geschaffen war, welcher dem nach einer Doyenschen Gastroenterostomie entspricht.

Wenn wir uns nun weiter in der Literatur bezüglich des Vorkommens von Galle und Pankreassaft im Magen nach Gastroenterostomien umsehen, so finden wir ein nicht unbeträchtliches Variieren in den diesbezüglichen Mitteilungen. Obalinski und Jaworski[1]) haben anscheinend zuerst Untersuchungen über den Einfluß von Operationen am Magen hinsichtlich der Wiederherstellung der gestörten Funktion dieses Organs angestellt und zwar bei einer Patientin, die wegen Pyloruskarzinom in „typischer Weise" pylorektomiert worden war; 2½ Monate post op. ist noch mehrfach Galle im ausgeheberten oder erbrochenen Mageninhalt gefunden worden. Heinsheimer[2]) hat an zwei Patienten, bei denen wegen Ulcusnarben mit konsekutiver Stenose eine Gastrojejunostomie gemacht worden war, eingehendere Stoffwechseluntersuchungen angestellt. Bei dem einen Patienten war die Nachuntersuchung einen Monat post op. vorgenommen worden. Hier ergab sich normale Verdauungskraft des Darmes für alle Nahrungsstoffe, insbesondere eine vorzügliche Fettausnutzung. Der andere Patient war zwei Jahre vor der Stoffwechseluntersuchung operiert worden; bei ihm fand Heinsheimer eine extrem gute Eiweißausnutzung, aber schlechte Resorption von Fett. Für beide Fälle findet sich jedoch keine Angabe, ob Galle oder Pankreassaft im Magen war. Die Arbeit bringt also eigentlich keinen Beitrag zu der uns momentan

1) Obalinski und Jaworski, Wien. klin. Wochenschr. 1889, Nr. 5 und 17.

2) Heinsheimer, Stoffwechseluntersuchungen bei zwei Fällen von Gastroenterostomie. Mitteilungen aus dem Grenzgebiete 1896, 2. Bd., S. 348.

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interessierenden Frage; sie wird auch von uns nur aus dem Grunde zitiert, weil in der einschlägigen Literatur diese Mitteilung Heinsheimers mehrfach Berücksichtigung gefunden hat. Für die Frage, ob Galle oder Pankreassaft im Mageninhalt sich befanden, kann sie natürlich nicht in Betracht kommen. Ebenso verhält es sich mit der stellenweise zitierten Arbeit Siegels[1]) über funktionelle Erfolge nach Operationen am Magen. In derselben wird zwar hervorgehoben, daß in allen Fällen „Rückfluß von Galle in den Magen" nicht beobachtet sei. Es ist aber damit offenbar gemeint, daß es nicht zum Circulus vitiosus gekommen sei. Von einer Ausheberung und Untersuchung des Mageninhaltes post op. ist nichts vermerkt, also auch wohl nicht vorgenommen. Infolgedessen hat auch die Siegelsche Arbeit für die Beantwortung unserer Frage keine Bedeutung. Ferner hat Kaenschke[2]) 1892 aus der Mikuliczschen Klinik über drei Fälle berichtet, bei denen er Untersuchungen über das funktionelle Resultat von Operationen am Magen angestellt hat; 2 Magenresektionen und 1 Gastroenterostomie nach v. Hacker unter Anwendung von einer ca. 50 cm langen Jejunalschlinge. Von der Gastroenterostomie wird mitgeteilt, daß zunächst der Erfolg der Operation zufriedenstellend gewesen sei, dann aber sei es sehr oft zu galligem Erbrechen gekommen, oftmals fünf- bis sechsmal in der Nacht; bei der Magenspülung sei auch das Spülwasser anfangs meist grünlich verfärbt gewesen und habe die Gallenfarbstoffreaktion gegeben. Offenbar ist also Galle im Magen gewesen; allem Anschein nach hat aber auch die Anastomose nicht ordentlich funktioniert; die Entleerung nach dem aboralen Darmteil hin scheint Schwierigkeiten gemacht zu haben. Bezüglich der beiden Resektionsfälle findet sich keine spezielle Angabe hinsichtlich des Befundes von Galle im Magen. Es wird nur allgemein bemerkt, daß das sekretorische Verhalten bei allen Patienten in übereinstimmender Weise durch die Operation nicht beeinflußt worden sei. Leider findet sich in der Arbeit auch nicht vermerkt, welche Methode der Pylorusresektion angewandt worden ist. Nur die II. Billrothsche Methode hat natürlich infolge Benutzung einer Jejunumschlinge zur Bildung einer Kommunikation zwischen Magen und Darm nach völligem Verschluß der Resektionsstelle dieselben Verhältnisse wie eine Gastroenterostomie, während bei der I. Billrothschen Methode durch das Einpflanzen des Duodenums in den Magen wesentlich andere Wechselbeziehungen zwischen

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1) Siegel, Über die funktionellen Erfolge nach Operationen am Magen. Mitteilung aus dem Grenzgebiet 1896, 1. Bd. S. 328.

2) Kaenschke, Untersuchungen über das funktionelle Resultat von Operationen am Magen. Deutsch. med. Wochenschr. 1892, Nr. 49.

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Magen und Darm geschaffen werden. Bei der I. Billrothschen Methode ist nur dann Galle im Magen zu erwarten, wenn antiperistaltisch der Darminhalt bewegt wird, während bei normaler Peristaltik kein Grund für den Eintritt von Darmsäften in den Magen ersichtlich ist. Würde also die I. Billrothsche Methode angewandt worden sein, so würde der von Kaenschke mitgeteilte Befund (dauerndes Fehlen von Galle im Magen) u. E. durchaus erklärlich sein. Nun sagen Mikulicz und Kausch im Handbuch der praktischen Chirurgie 1903 S. 175 Anmerkung, daß „früher" in der Breslauer Klinik „ausschließlich die I. Billrothsche Methode" angewandt worden sei. Man ist also wohl in Anbetracht des Umstandes, daß die Kaenschkesche Veröffentlichung aus der Mikuliczschen Klinik im Jahre 1892 erschienen ist, berechtigt zu schließen, daß in den beiden in Rede stehenden Fällen auch die I. Billrothsche Methode angewandt worden ist. Damit verlieren aber die beiden Kaenschkeschen Resektionsfälle hinsichtlich der Frage, ob Galle nach einer Gastroenterostomie im Magen zu erwarten sei, ihre Bedeutung.

Grundzach und Mintz[1]) und Mintz[2]) teilen einen Fall mit, bei welchem wegen narbiger Pylorusstenose, Hypertrophie des Pylorus und konsekutiver Magendilatation eine Gastroenterostomie nach Wölfler ausgeführt wurde. Neun Monate später stellte sich die Patientin mit einer Gewichtszunahme von 54 Pfund wieder vor; Magenbeschwerden bestanden nicht mehr; Magendimensionen waren normal; chemische und motorische Tätigkeit war ebenfalls normal; nüchtern fand sich im Magen etwas alkalisch reagierender, speisefreier Inhalt. Von der Anwesenheit von Galle oder Pankreassaft im Magen ist nichts vermerkt.

Rosenheim[3]) demonstrierte in der Berliner med. Gesellschaft Oktober 1894 3 Patienten, bei denen Hahn wegen Karzinom (2mal) resp. wegen gutartiger Pylorusstenose (1 mal) Gastroenterostomie gemacht hatte. Die Operation lag 4 resp. 9 Monate zurück. Rosenheim gab an, daß bei den beiden Karzinomfällen eine befriedigende Besserung hinsichtlich Sekretion und Motilität des Magens nach der Operation zu verzeichnen gewesen sei; bei den Patienten mit gutartiger Pylorusstenose sei die sekretorische und motorische Funktion des Magens durchaus normal geworden. Angaben, ob jemals Galle oder Pankreassaft im Magen gewesen sind oder ob daraufhin untersucht worden ist, finden sich nicht in der Arbeit.

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1) Grundzach und Mintz, Medycyna 1893 und Revue de méd. 1893, no. 11.

2) Mintz, Operative Behandlung der Magenkrankheiten. Zeitschr. f. klin. Med. 1894, Bd. 25.

3) Rosenheim, Über das Verhalten der Magenfunktion nach Ausführung der Gastroenterostomie. Berl. klin. Wochenschr. 1894, Nr. 50.

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Dumin[1]) hat 3 Fälle mitgeteilt, bei denen wegen gutartiger Stenose des Pylorus Gastroenterostomie gemacht worden ist. Die letzten funktionellen Magenuntersuchungen sind relativ kurze Zeit post op. gemacht worden (bis zu 5 Monaten). Dumin weist am Schluß seiner Arbeit ausdrücklich nochmals darauf hin, daß „kein einziges Mal im Mageninhalt die Anwesenheit einer ‚größeren Quantität' Galle beobachtet worden sei". Demgemäß muß sich also doch etwas Galle im Magen befunden haben. Auf Pankreassaft ist nicht untersucht worden.

Carle und Fantino[2]) haben bei allen ihren Operierten bald nach der Operation sowohl im nüchternen Zustand als verschiedene Stunden nach der Mahlzeit Galle im Magen beobachtet. Die Beimengung von Galle ist bei den nach der Wölflerschen Methode Operierten meist reichlicher gewesen als nach der v. Hackerschen Operation; allerdings ist auch bei den nach v. Hacker Operierten verschiedentlich recht reichliche Beimengung von Galle zum Mageninhalt gefunden worden. Carle und Fantino bemerken dann ausdrücklich, daß der Rückfluß nach und nach seltener wurde, bis er bei vielen Operierten verschwand; sie heben ferner hervor: „bei vielen, nicht bei allen". „Dies geschah in einem bis zu vielen Monaten wechselnden Zeitraum, was auf Rechnung eines um die neue Öffnung gebildeten Sphinkters zu setzen ist." Sie erklären sich den Rückfluß in der ersten Periode in der Weise, daß sie zunächst eine mangelhafte oder noch ganz fehlende Funktion eines an der Gastroenterostomieöffnung neu zu bildenden Sphinkters annehmen; später, wenn sich der Sphinkter gebildet haben soll, bleibt der Rückfluß der Galle aus.

Kausch[3]) ist der Ansicht, daß nach der Gastroenterostomie in allen Fällen meist geringe Mengen Galle im Magen sich befinden. Dieses Verhalten soll das ganze Leben hindurch bestehen bleiben, indessen nur selten Beschwerden verursachen.

Hartmann et Soupault,[4]) welche ziemlich eingehend und genau über die funktionellen Resultate ihrer Magenoperationen (Pylorus-

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1) Dumin, Über die Resultate der Gastroenterostomie bei narbiger Verengerung des Pylorus. Berl. klin. Wochenschr. 1894, Nr. 4, S. 90.

2) Carle und Fantino, Beitrag zur Pathologie und Therapie des Magens. Arch. f. klin. Chir. Bd. 56.

3) Kausch, Über funktionelle Ergebnisse nach Operationen am Magen. Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie 1899, 4. Bd., S. 463.

4) Hartmann et Soupault, Les résultats éloignés de la gastroentérostomie. Revue de chirurgie 1899, p. 137.

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resektionen und Gastroenterostomien) berichten, teilen mit, daß sie bei allen ihren Gastroenterostomien, einerlei ob nach der Wölflerschen oder v. Hackerschen Methode verfahren worden sei, mit Ausnahme eines einzigen Falles, stets Galle im Magen gefunden hätten, oft in ganz beträchtlichen Quantitäten. Dabei ist zu bemerken, daß alle diese Untersuchungen, welche einen positiven Gallenbefund ergeben haben, relativ kurze Zeit post operationem (im Maximum ein halbes Jahr) vorgenommen sind. Nur in dem einen Falle (Obs. VII), bei welchem keine Galle im Magen gefunden wurde, fand die Untersuchung $1^1/_4$ Jahr post op. statt. Hier wurde nämlich bei einer 56jährigen Frau am 27. Juli 1897 wegen eines Ulcus ventriculi eine Gastroenterostomia anterior antecolica mit gutem Erfolge angelegt. Am 16. Dezember 1897 fand sich noch bei einer funktionellen Magenuntersuchung Galle im Magen. Die nächste Untersuchung fand am 17. Oktober 1898 statt und hierbei wurde keine Galle mehr im Magen gefunden. Bemerkenswert sind aus der Arbeit von Hartmann et Soupault auch noch zwei andere Fälle, bei denen wegen dauernden, galligen Erbrechens eine Braunsche Anastomose zur Gastroenterostomie hinzugefügt werden mußte (Obs. V u. IX). Der eine von ihnen (Obs. V) war am 25. Mai 1897 wegen Gastritis nach Wölfler gastroenterostomosiert worden. Wegen galligen, oft rezidivierenden Erbrechens mußte zu Anfang März 1898 eine Enteroanastomose hinzugefügt werden. Die Beschwerden hörten sofort auf; eine 6 Wochen nach der Anlegung der Enteroanastomose vorgenommene funktionelle Untersuchung ergab aber noch Galle im Magen. Die nächste funktionelle Magenuntersuchung wurde am 20. Oktober 1898, also 1 Jahr 5 Monate nach der Gastroenterostomie, gemacht, und nun fand sich keine Galle mehr im Magen. Bei dem anderen Falle (Obs. IX) wurde wegen Carcinoma pylori eine Gastroenterostomie angelegt; es ergab sich gleich bei der Operation die Notwendigkeit, eine Braunsche Anastomose hinzufügen zu müssen. Eine funktionelle Magenuntersuchung wurde in diesem Falle kaum 4 Wochen post op. gemacht. Es fand sich Galle im Magen. Später konnte nicht mehr untersucht werden; der Patient erlag seinem Leiden.

Auch Terrier und Hartmann[1]) haben in der ersten Zeit nach der Gastroenterostomie stets die Anwesenheit von Galle im Magen konstatiert; andererseits werden mehrere Fälle aufgeführt, bei denen später keine Galle mehr im Magen gefunden wurde. In einem Falle (Obs. XVI) fanden sie schon 5 Monate post op. keine Galle im Magen mehr. Im übrigen ist das Material, auf welches Terrier und Hart-

1) Terrier et Hartmann, Chirurgie de l'éstomac. Paris, Steinheil 1899.

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mann sich stützen, ziemlich dasselbe, welches Hartmann und Soupault in ihrer vorstehend zitierten Arbeit benutzt haben.

Kelling[1]) hält das Durchströmen von Galle und Pankreassaft durch den Magen mit dem Wohlbefinden für vereinbar.

Kappeler ist anderer Ansicht; er meint, die Galle sei gleichgültig für den Magen, der Pankreassaft aber nicht, und Tavel[2]) äußert sich in ähnlichem Sinne, wenn er sagt: si la pénétration de la bile est indifferente à la digestion stomacale, il n'est pas toujours de même de la pénétration du suc pancréatique.

Krönlein[3]) faßte sein Resümee auf dem Chirurgenkongreß 1906 auf Grund der Ergebnisse seiner Zusammenstellung über den Erfolg der Gastroenterostomie bei Ulcus ventriculi dahin, daß „der häufig auftretende Rückfluß von Galle in den Magen keine ausgesprochenen Beschwerden zur Folge habe und nach längerer Zeit zu verschwinden scheine; der Rückfluß von Pankreassaft sei relativ selten nachweisbar".

Schließlich ist Katzenstein[4]) durch experimentelle Untersuchungen an Hunden zu der Ansicht gekommen, daß der Einfluß von Galle und Pankreassaft in den Magen nach einer Gastroenterostomie (anterior und posterior) nicht nur nicht schade, sondern zur Heilung eines Ulcus ventriculi direkt beitrage infolge Neutralisation des aziden resp. superaziden Magensaftes. Das Einfließen soll in der ersten Zeit nach der Operation dauernd, später periodisch erfolgen; und zwar soll der Fettgehalt der Nahrung infolge reflektorischer Anregung der Absonderung von Galle und Pankreassaft in quantitativer Hinsicht von Bedeutung sein.

Eine Bestätigung der Katzensteinschen Theorie hat an drei gastroenterostomierten Patienten Desiderius Balás[5]) gefunden. Wir möchten aber hervorheben, daß Katzenstein und Balás ihre Untersuchungen immer sehr bald nach Anlegung der Gastroenterostomie angestellt haben.

Es ergibt sich also, daß die Mitteilungen und Ansichten der Autoren nicht unbeträchtlich differieren. Die einen haben dauernd Galle im

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1) Kelling, Studien zur Chirurgie des Magens. Langenbecks Archiv 1900, S. 308.

2) Tavel, Le reflux dans la gastroentérostomie. Revue de chirurg. 1901, no. 12.

3) Krönlein, Selbstbericht über Chirurgenkongreß 1906. Zentralbl. f. Chir. 1906, S. 94.

4) Katzenstein, Über die Änderung des Magenchemismus nach Gastroenterostomie und den Einfluß dieser Operation auf das Ulcus und Carcinoma ventriculi. Deutsch. med. Wochenschr. 1907, Nr. 3 und 4.

5) Desiderius Balás, Beiträge zur Lehre der Hyperazidität. Deutsche med. Wochenschr. 1908, Nr. 1.

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Magen gefunden, die anderen nicht; die einen glauben an eine Unschädlichkeit des Gallenzuflusses zum Magen, die anderen nicht; die einen sind der Ansicht, daß Galle allein für den Magen indifferent sei, der Pankreassaft aber nicht; die anderen halten beide Sekrete für nicht nur nicht indifferent, sondern bei Ulcus ventriculi für sogar heilsam und erwünscht.

Beschäftigen wir uns nun zunächst mit der Frage, ob Galle allein im Magen vertragen wird, so kann es keinem Zweifel mehr unterliegen, daß dieselbe sich auch in größeren Mengen jahrelang im menschlichen Magen finden kann, ohne irgendwelche Beschwerden zu verursachen. Freilich werden in der Literatur vereinzelte Fälle mitgeteilt, wo die Anwesenheit von Galle im Magen nach einer Gastroenterostomie Erbrechen, pappigen Geschmack, Unbehagen usw. hervorgerufen haben soll. Ob aber in diesen Fällen tatsächlich die Galle schuld an den geklagten Beschwerden gewesen ist, oder ob sie nur, weil nach der Gastroenterostomie im Magen vorhanden, mit dem Erbrechen herausbefördert worden ist, während der Grund des Erbrechens ein ganz anderer war, das ist keineswegs mit Sicherheit klargestellt. Jedenfalls gibt es doch zu denken, daß diesen wenigen Fällen, wo angeblich die Galle im Magen das den Brechreiz auslösende Moment gewesen sein soll, so viele Beobachtungen entgegenstehen, wo die Galle unbeanstandet jahrelang vertragen wird. Sollte nicht mit weit größerer Wahrscheinlichkeit angesichts der letzterwähnten Tatsache die Annahme berechtigt sein, daß bei jenen wenigen Ausnahmefällen irgendeine andere Schädlichkeit der Grund der Beschwerden gewesen ist, z. B. chronischer Circulus vitiosus infolge nicht ganz funktionierender Anastomose, Fortbestehen der vor der Operation vorhandenen Sekretionsanomalien, nervöse Beschwerden usw.? Wir möchten in Übereinstimmung mit Kelling[1]) und Hartmann et Soupault[2]) besonders letzteren Punkt als Grund für die Beschwerden annehmen, vorausgesetzt natürlich, daß ein organischer Fehler, z. B. ein chronischer Circulus vitiosus, fehlt.

Den besten Beweis für die Unschädlichkeit der Galle im Magen liefern diejenigen Fälle, bei denen mit Sicherheit nur die Galle allein in den Magen gelangt; in dieser Hinsicht mangelt es uns nicht an experimentellen und klinischen Beobachtungen an Hunden und an Menschen: die Unschädlichkeit großer Quantitäten Galle für den Magen beim Tiere ist experimentell zuerst von M. Dastre[3]) nach-

1) Kelling, Studien zur Chirurgie des Magens. Langenbecks Archiv 1900, S. 306.

2) Hartmann et Soupault, Presse médic. 1899, 14.

3) Dastre, Recherches sur l'action de la bile sur la digestion gastrique. Archives de physiologie Paris 1890, t. II, p. 316.

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gewiesen worden. Dastre legte bei Hunden Magenfisteln an und führte durch dieselben Ochsengalle in verschieden großen Quantitäten und zu verschiedenen Stadien der Verdauung in den Magen ein, ohne jemals irgendwelchen Nachteil davon zu sehen. Ruggero Oddi[1]) hat dann bei Hunden den Ductus choledochus unterbunden und eine Cholezystgastroanastomose angelegt; die gesamte produzierte Galle mußte also in den Magen gehen; irgendeine schädliche Folge hat Oddi davon nicht gesehen.

Masse[2]) hat dieses Experiment bei einer größeren Anzahl von Hunden nachgeprüft; das Ergebnis war dasselbe wie bei Oddi; die Tiere haben ein Jahr und länger nach der Operation gelebt und an Gewicht zugenommen. Bei der Sektion, die bei einigen Tieren $^3/_4$ Jahr nach der Operation vorgenommen wurde, erwies sich der Verschluß des Choledochus als vollkommen intakt. Chlumski[3]) hat durch seine experimentellen Untersuchungen an Hunden ebenfalls die Ungefährlichkeit der Beimengung von Galle zu Mageninhalt bestätigt.

Aber nicht allein für Tiere, auch für Menschen liegen bereits in der Literatur Mitteilungen vor, die eine Bestätigung der durch Tierexperiment gewonnenen Erfahrung bringen. Daß die diesbezüglichen Mitteilungen nicht zahlreicher sind, erklärt sich aus der Tatsache, daß für gewöhnlich, falls sich die Notwendigkeit der Anlegung einer Anastomose zwischen Gallenblase und dem Magendarmtraktus ergibt, der Darm zur Anastomosenbildung benutzt wird. So hat z. B. im Jahre 1902 Radsiewski[4]) 56 Fälle von künstlicher Gallenblase-Darmfistel — teils mit dem Dünndarm, teils mit dem Dickdarm — zusammengestellt. Hinter dieser Zahl bleiben allerdings die Fälle von Gallenblasen-Magenfistel zurück.

Wickhoff und Angelberger[5]) waren die ersten, welche eine Cholecystgastrostomie an Menschen machten wegen starker Verwachsung nach Cholelithiasis und Kompression des Choledochus, welche zu einem völligen Passagehindernis für die Galle geführt hatte. Der Erfolg der Operation war durchaus zufriedenstellend. Selbst in den ersten Tagen nach der Operation trat kein galliges Erbrechen ein. Der Patient verließ 3 Monate nach der Operation geheilt das Krankenhaus, ohne irgendwelche Beschwerden. Er hatte an

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1) Ruggero Oddi, Azione della bile sulla digestione gastrica. Perugia 1887.

2) Masse, De la cholécystogastrostomie. Congrès français de chirurgie, Paris 1898, p. 300.

3) Chlumski, Beitr. z. klin. Chir. Tübingen, Bd. 20.

4) Radsiewski, Mitteilungen a. d. Grenzgebieten der Mediz. u. Chir. 1902, Bd. 9, S. 659.

5) M. Wickhof und Fr. Angelberger, Wien. klin. Wochenschr. 1893, S. 325.

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Körpergewicht während der Behandlung zugenommen. Terrier[1]) machte wegen einer Pankreaskopfgeschwulst dieselbe Operation mit gleich gutem Effekt. Monod[2]) legte bei einem infolge Karzinom des Pankreas ikterisch gewordenen Menschen eine Cholecystgastrostomie an; der Patient starb allerdings schon am Tage nach der Operation. Quénu[3]) und Jaboulay[4]) machten wegen Tumor resp. Verhärtung des Pankreaskopfes je eine Anastomose zwischen Gallenblase und Magen. Die vor der Operation bestehenden Erscheinungen von Gallenstauung gingen prompt zurück; die völlige Ableitung von Galle nach dem Magen hin machte sich in keiner Weise unangenehm bemerkbar. Krumm[5]) legte wegen Neubildung im Pankreaskopf mit Ektasie der Gallenblase infolge Verschlusses des Choledochus bei einem stark Ikterischen ebenfalls eine Cholecystgastrostomie an. Das Duodenum war wegen zahlreicher Verwachsungen nicht geeignet zur Anastomose. Auch hier wurde die Entleerung der gesamten Galle in den Magen ohne Erbrechen hervorzurufen und unter allmählicher Entfärbung der Haut gut vertragen. Ferner hat O. Hildebrand während seiner Baseler Tätigkeit 4mal eine Anastomose zwischen Gallenblase und Magen angelegt. Im Jahre 1902 veröffentlichte Hildebrand[6]) einen Fall, bei welchem er mit gutem Erfolg die in Rede stehende Anastomose wegen dauernden Gallenflusses aus einer nach einer Cholelithiasisoperation zurückgebliebenen Fistel vorgenommen hatte. Die Galle im Magen wurde dauernd gut vertragen; die Fistel schloß sich. Eine Anastomose mit dem Dünndarm war unmöglich gewesen wegen ausgedehnter Verwachsungen. Ein Jahr später machte Hildebrand[7]) Mitteilung von 3 anderen Fällen, bei welchen er die Fistelbildung zwischen Magen und Gallenblase mit demselben guten Erfolge ausgeführt hatte. 2mal handelte es sich um Karzinom des Pankreas, einmal um ein Gallenblasenkarzinom mit sekundärer Geschwulstbildung. Die anatomischen Verhältnisse lagen ähnlich so, wie in dem zuerst beschriebenen Falle. Kürzlich haben wir auf der Klinik in einem

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1) Terrier, Remarques sur deux cas l'un de cholécysto-duodénostomie l'autre de cholécysto-gastrostomie. Revue de chirurg. 1896, p. 169.

2) Monod, Bull. et mém. de la soc. de chirurg. de Paris 1896, p. 546.

3) Quénu, Bull. et mém. de la soc. de chirurg. de Paris 1896, no. 18, p. 558.

4) Jaboulay, Lyon médical 1898, no. 47.

5) Krumm, Münch. med. Wochenschr. 1901, Nr. 2.

6) O. Hildebrand, Beiträge zur operativen Chirurgie. Langenbecks Archiv 66 p. 347.

7) O. Hildebrand, Ein weiterer Beitrag zur Cholocystogastrostomie. Deutsche Zeitschr. für Chir. B. 66 S. 379.

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Falle von inoperabelem Karzinom an der Papilla Vateri, welches erhebliche Gallenstauung mit hochgradigem Ikterus verursacht hatte, wiederum eine Cholecystgastrostomie gemacht. Der Erfolg war derselbe gute wie in den früheren Fällen. Auch Kehr[1]) teilte auf dem Chirurgenkongreß 1903 mit, daß er solche Cystogastrostomie in 12 Fällen gemacht habe, ohne daß die Galle die Magenverdauung irgendwie gestört habe.

Wir kommen nun zu der Frage, ob und wieviel Pankreassaft sich nach einer Gastroenterostomie im Magen befindet und wie es mit der Toleranz des Magens gegen das Sekret der Bauchspeicheldrüse steht? Wie wir gesehen haben, finden sich zwar in der Literatur zahlreiche Mitteilungen über das Vorhandensein von Galle im Mageninhalt nach einer Gastroenterostomie; von Pankreassaft ist aber relativ selten die Rede. Und doch ist es eigentlich selbstverständlich, daß nicht nur Galle, sondern auch Pankreassaft in den Magen kommen muß, wenn einmal eine Anastomose zwischen Magen und Jejunum existiert. Man hat aber offenbar seltener bisher darauf geachtet. Es erklärt sich das auch zwanglos daraus, daß die Beimengung von Galle zum Mageninhalt infolge ihrer Farbe viel leichter sich manifestiert, als das Pankreassekret. Katzenstein[2]) hat nun, wie gesagt, durch seine Experimente an Hunden nachgewiesen, daß sich regulär bei der Gastroenterostomia anterior antecolica und posterior retrocolica einige Monate nach Operation nicht nur Galle, sondern auch Pankreassaft im Magen findet, ohne irgendwelche Störungen zu machen. Es kann sich jedoch dabei immer nur um mehr oder minder große Beimengungen von Pankreassaft zum Mageninhalt handeln. Wenn es auch für die Galle erwiesen ist, daß das gesamte produzierte Lebersekret seinen Weg durch den Magen in den Darm nehmen kann, ohne Schädigung des Organismus und anscheinend auch ohne selbst eine wesentliche Alteration seiner Wirksamkeit zu erleiden. — die Stühle sind bei Cholecystgastrostomien nicht acholisch —, so ist das für den Pankreassaft in seiner Totalität anscheinend nicht der Fall. Wenigstens scheinen die bisher vorliegenden Experimente und klinischen Erfahrungen dagegen zu sprechen. Chlumskiy[3]) hat an Hunden das Duodenum 10 cm unterhalb der Papilla Vateri durchschnitten, die zuführende Schlinge nahe der Kardia in den Magen eingenäht und das abführende Darmstück mit dem Pylorusteil verbunden. cfr. Fig. a.

1) Kehr, Chirurgenkongreß 1903, S. 76.

2) Katzenstein, l. c.

3) Chlumskig: Über die Gastroenterostomie. Beiträge zur Klin. Chirurgie 1898 und Weitere Erfahrungen über die Gastroenterostomie. Beiträge zur Klin. Chirurgie 1900.

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Alle drei in dieser Weise operierten Hunde, bei denen also das gesamte Sekret von Leber und Pankreas in den Magen gelangte, starben unter den Symptomen hochgradigen Durstgefühls, der Appetitlosigkeit und der Abmagerung. Bei zwei anderen Hunden durchschnitt Chlumskiy, um nur den Pankreassaft in den Magen zu leiten, den Dünndarm nahe dem Duodenum; das orale Ende wurde unterbunden, das aborale in die vordere Magenwand eingepflanzt, der Ductus choledochus durchschnitten und unterbunden, und schließlich eine Verbindung zwischen Gallenblase und dem abführenden Darmstück hergestellt. cfr. Fig. b.

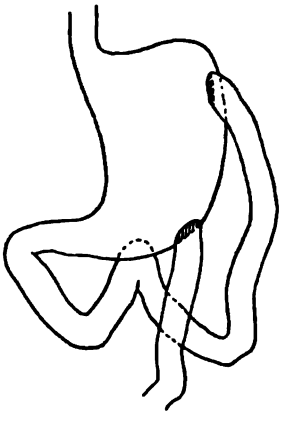

Fig. a.

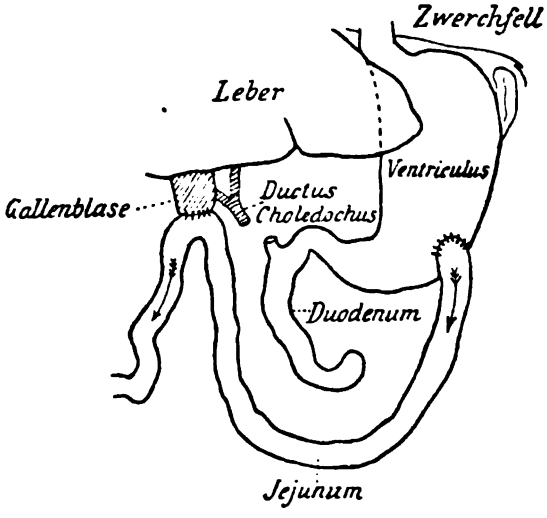

Fig. b.

Beide Hunde starben unter denselben Symptomen wie oben. Somit schien der Beweis gebracht zu sein, daß das Tier zugrunde geht, wenn der Pankreassaft vollständig in den Magen eingeleitet wird.

Steudel gelang es aber, einen Hund, bei dem er das Jejunum durchschnitten, den oralen Schenkel durch eine Okklusionsnaht verschlossen und den aboralen Schenkel in den Magen eingenäht hatte, 18 Tage bei normaler Freßlust am Leben zu halten. cfr. Fig. c. Das Tier starb an einem Nahtdefekt. Es mußte also Galle und Pankreassaft vollständig in den Magen durch den Pylorus gegangen sein. Dieses Ergebnis schien gegen die Chlumskiyschen Versuche zu sprechen. Chlumskiy selbst, der die Steudelsche Versuchsanordnung nachgeprüft und auch bei dieser Modifikation einen Hund durchgebracht hat, erklärt die Differenz zwischen den Resultaten seiner und Steudels Versuchen dadurch, daß er das schädigende Moment bei seinen Versuchen in dem beständigen Zufluß von Galle und Pan-

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kreassaft zum Magen sieht, während bei den Steudelschen Versuchen sich Galle und Pankreassaft während der Verdauung im Duodenum sammle, und der zunächst geschlossene Pylorus erst dann sich öffne und den Zufluß von Galle und Pankreassaft in den Magen gestatte, wenn die Ansammlung von Flüssigkeiten im Duodenum sehr groß und die Magenverdauung schon beendet sei. Das Einfließen der Sekrete der beiden großen Unterleibsdrüsen finde also bei Steudels Versuchsanordnung nicht kontinuierlich, sondern periodisch statt, und hierin liege die Erklärung der Differenz der Versuchsergebnisse. Eine Stütze seiner Anschauung bekam Chlumskiy durch den Fall, den Ledderhose[1]) auf dem Chirurgenkongreß 1899 vorstellte. Bei einer Duodenalstenose unterhalb der Papilla Vateri durch Tumor mit kopiösem, galligem Erbrechen hat Ledderhose eine Gastroenterostomia anterior antecolica mit gutem Resultat gemacht. Da das Hindernis an der Papilla Vateri saß, so wurde sicherlich neben der Galle auch Pankreassaft in den Magen entleert. Der Patient erbrach nach der Operation nicht mehr. Ledderhose nahm an, daß die Hauptmenge der Galle und des Pankreassaftes durch den Pylorus in den Magen und dann in den Darm fließe, jedoch infolge der vorhandenen normalen Verschlußfähigkeit des Pylorus nicht kontinuierlich, sondern nur zeitweise, hauptsächlich wohl gegen Ende der Verdauung.

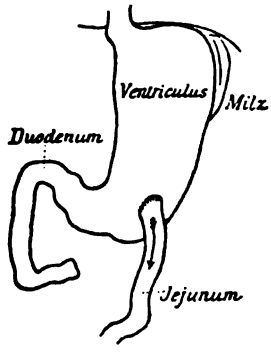

Fig. c.

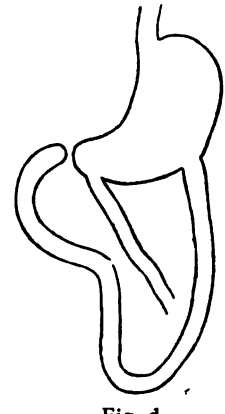

Fig. d.

Kelling[2]) ist jedoch nicht der Ansicht, daß der Pankreassaft eine so unheilvolle Wirkung ausübt, wie Chlumskiy annimmt. Er teilt mit, daß es ihm gelungen sei, durch Experiment einem Hunde sämtliche Galle und sämtlichen Pankreassaft durch den Magen fließen zu

1) Ledderhose, Ein Fall von Gastroenterostomie wegen Stenose des unteren Duodenums. Chirurgenkongreß 1899.

2) Kelling, Studien zur Chirurgie des Magens. Langenbecks Archiv 1900, S. 310 u. 313.

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lassen. Der Hund sei ganz munter gewesen, habe Appetit gehabt, habe normalen Stuhl abgesetzt und sei 8 Wochen nach der Operation genau so schwer gewesen wie vorher. Die Art des Kellingschen Versuches demonstriert nebenstehende Fig. d.

Das Jejunum wurde dabei unterhalb des Durchtrittes durch die Radix mesenterii durchschnitten, das obere vom Duodenum kommende Ende wurde in den Fundus zirkulär eingenäht; der Pylorus wurde verschlossen; das abführende Ende des Jejunums wurde in die hintere Wand des Pylorusteiles direkt oberhalb der Pylorusunterbindung zirkulär eingenäht. Kelling sucht die Todesursache der Chlumskiyschen Hunde nicht in dem Zufluß von Pankreassaft zum Magen und der sich daraus ergebenden Verdauungsstörungen, sondern in motorischen Störungen, welche dadurch bedingt sein sollen, daß das gefüllte Duodenum eine Hemmung auf den Magen ausübt. Seine Experimente haben ihm ergeben, „daß Füllung des Duodenums die Entleerung des Magens erheblich verzögert", ein Resultat, welches mit der v. Meringschen[1]) Behauptung im Einklang steht, „daß durch die Füllung des Duodenums eine Sistierung der Magenentleerung statt hat". Der Unterschied zwischen den Chlumskiyschen und Kellingschen Versuchen besteht eben darin, daß Kelling der Chlumskiyschen Versuchsanordnung noch einen Verschluß des Pylorus hinzugefügt hat. Dadurch ist die Füllung des Duodenums vom Magen aus unmöglich geworden; mithin ist nach Kellings Auffassung die Hemmung weggefallen, welche das gefüllte Duodenum auf den Magen ausüben kann, damit ist die Ursache beseitigt, daß der Magen nicht die Kraft entwickeln kann, welche er braucht, um seinen Inhalt durch die Jejunalfistel auszutreiben.

Wir haben nun die Versuche nach Chlumskiys Anordnung an drei Hunden nachgeprüft und sind zu demselben Resultat gekommen wie Chlumskiy; die Hunde sind sämtlich innerhalb 8 Tagen gestorben und zwar nicht an Peritonitis; beide Darmschlingen, die also an verschiedenen Stellen in den Magen (Kardia- resp. Pylorusnähe) eingenäht waren, zeigten keinerlei Differenz in ihrem Füllungsgrade; sie waren beide ziemlich leer; dagegen war der Magen außerordentlich gedehnt und schlaff mit schwärzlichen, dünnflüssigen Massen gefüllt. Das Bild war bei allen drei Hunden dasselbe.

Auch die Kellingsche Versuchsanordnung — also noch eine Unterbindung des Pylorus nach dem Einpflanzen der beiden Darmenden in den Magen — haben wir an zwei Hunden nachgeprüft. Die

1) v. Mering, 15. Kongreß für innere Medizin 1897. Zur Funktion des Magens.

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Tiere sind ebenfalls beide nach 5—6 Tagen eingegangen; das Sektionsergebnis war dasselbe wie bei den nach Chlumskiy operierten Hunden.

Unseres Erachtens scheint die Frage nach der Anwesenheit und Schädlichkeit des Pankreassaftes im Magen folgendermaßen zu beantworten zu sein. Sicher ist, daß Pankreassaft, und oft in nicht geringen Mengen, wenn man ein einigermaßen proportionales Verhalten zwischen Leber und Pankreasabsonderung annimmt, im Magen nach einer Gastrojejunostomie vorkommt. Allem Anschein nach hat aber das periodische Einfließen des Darmsaftes in den Magen, wie das ja bei einer Gastrojejunostomie ganz zweifellos entsprechend dem periodischen Verhalten beim Austreten des Sekretes aus den beiden Drüsen in den Darm der Fall ist, doch eine gewisse Bedeutung. Außerdem fließt sicherlich bei einem Gastroenterostomierten nicht aller Pankreassaft in den Magen. Ob der Magen sämtlichen Pankreassaft verträgt, scheint noch nicht absolut einwandsfrei klar gestellt zu sein. Praktisch ist diese Frage hinsichtlich der Gastroenterostomie aber auch von untergeordneter Bedeutung; in dieser Hinsicht ist mit der Erkenntnis völlig Genüge geschehen, daß der menschliche Magen diejenigen Mengen von Pankreassaft, welche durch eine Gastroenterostomieöffnung eintreten, ohne Schädigung enthalten kann. Die Ansicht der Autoren, welche das Einfließen von Pankreassaft in den Magen überhaupt als gefährlich ansehen, ist demgemäß als irrtümlich und widerlegt zu bezeichnen.

Wie haben wir uns nun zu erklären, daß wir nicht bei allen unseren Gastroenterostomierten Galle und Pankreassaft im Mageninhalt haben nachweisen können? Z. B. hat sich bei den unter Gruppe B untergebrachten 4 Patienten der Nachweis niemals führen lassen; auch bei Gruppe C (Gastrojejunostomie mit Braunscher Anastomose) hat sich bei Fall I (4mal untersucht) überhaupt nicht, bei Fall II (6mal untersucht) nur in der ersten Zeit post op. Galle und Pankreassaft im Magen gefunden. Der Grund dafür kann unseres Erachtens nur darin gesucht werden, daß bei denjenigen Fällen, wo wir Galle und Pankreassaft im Magen nicht nachweisen konnten, die Anastomosenöffnung nicht mehr funktioniert hat. Es is sonst gar nicht zu verstehen, warum bei gutem Funktionieren der Fistel zwischen Magen und Darm kein Darmsaft in den Magen eintreten sollte. Wir müssen uns den Vorgang etwa folgender Art vorstellen: In der ersten Zeit nach der Gastroenterostomie findet bei allen Patienten ein Einfließen von Galle und Pankreassaft in den Magen statt, und zwar ist es gleichgültig, ob die vordere oder hintere Gastrojejunostomie gemacht worden ist; das Hinzufügen der Braunschen Anastomose scheint an diesem

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Befunde zunächst nichts zu ändern. Bei den Y-Methoden halten wir es für wahrscheinlich, daß Galle und Pankreassaft von vornherein im Magen fehlen können; eigene Erfahrungen stehen uns in dieser Hinsicht, wie gesagt, nicht zur Seite.

Im Laufe der Zeit scheint sich aber unter gewissen Umständen ein Verschluß des Lumens der Anastomose mehr und mehr auszubilden. Es kann sich allerdings nur um einen Verschluß in funktionellem, nicht in anatomischem Sinne handeln; im Effekt können aber beide gleich sein. Daß es zu einem anatomischen Verschluß, also zu einer völligen Verwachsung der Anastomosenränder für gewöhnlich nicht kommen kann, ist durchaus plausibel, da ja die Ränder der Anastomosenöffnung mit Schleimhaut oder Narbe bedeckt sind und nachträglich ohne blutige Anfrischung nicht verwachsen können.

Ein funktioneller Verschluß der Anastomose ist aber sehr wohl denkbar. Drei Faktoren sind es, die u. E. den funktionellen Verschluß herbeiführen. Erstens die Narbenschrumpfung der Anastomosenränder. Daß dieselbe eintritt und daß sie besonders dann, wenn nicht ganz exakt die Schleimhauträder miteinander vernäht sind, ziemlich erheblich sein kann, ist außer Frage. Tavel[1]) hat erst kürzlich wieder auf die Möglichkeit aufmerksam gemacht, daß die Anastomosenränder bei nicht ganz exakter Schleimhautnaht sich bald nach der Operation sogar wieder völlig vereinigen können, so daß die ganze Anastomose illusorisch wird. Eine schlechte Mukosanaht disponiere enorm zu narbiger Kontraktion; es könne sogar eine völlige Stenose entstehen. Graser[2]) hat sich in ähnlichem Sinne ausgesprochen: besonders beim Murphyknopf sah er mehrmals nachträgliche Verklebung der Anastomosenstelle.

Diese unerwünschten Verengungen und völligen Verklebungen der Anastomose haben auch mit dazu beigetragen, den Murphyknopf zu diskreditieren und vor allem eine exakte Schleimhautnaht zu verlangen. Rotgans[3]) hat zur Vermeidung dieses Mißerfolges empfohlen, ein ovales Stück aus der Magenwand zu exzidieren. Sicherlich wird man dadurch die Gefahr einer Stenose der Anastomoseöffnung vermindern.

Der zweite zur Verengerung führende Faktor liegt in der Änderung, die der Magen in der Art seiner Entleerung nach der Operation durchmacht. Wenn nämlich die Gastroenterostomie, wie es sehr oft der Fall ist, wegen Stauungsbeschwerden mit Dilatation und Atonie der

1) Tavel, l. c.
2) Graser, Chirurgenkongreß 1906.
3) Rotgans, l. c.

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Magenmuskulatur gemacht ist, so wird zunächst nach Anlegung der neuen Öffnung eine gute Entleerung des Magens durch die Anastomose stattfinden. Zunächst steht, da doch die Gastroenterostomie bei einem dilatierten, atonischen Magen am tiefsten Punkte der Curvatura major angelegt wird, die Anastomosenstelle am tiefsten und wird infolgedessen auch als hauptsächlichste Austrittsöffnung vom Mageninhalt benutzt. Sobald sich aber unter den neuen, günstigen Entleerungsbedingungen der Magen von seiner Dilatation und Atonie erholt hat, d. h. also sich verkleinert und seine Muskulatur wieder gekräftigt hat, wählt der Mageninhalt nicht mehr, wie zu Anfang, die künstliche Anastomose als Austrittsstelle, sondern wieder den normalen Pylorus. Es hängt das zum Teil davon ab, weil bei nicht dilatiertem Magen der Druck im Pylorusteil am größten ist. Cannon und Blake[1]) haben das durch ihre Experimente an mit Wismutbrei gefütterten Katzen, bei denen sie den Gang der Nahrung röntgenographisch kontrollierten, in Übereinstimmung mit den von Moritz und v. Pfungen mitgeteilten Beobachtungen nachgewiesen. Auch Power[2]) und Rogers[3]) sind der Ansicht, daß der Mageninhalt nicht längere Zeit nach Anlegung der Gastroenterostomose die künstliche Anastomosenöffnung benutzt, sondern bald wieder den alten Weg durch den Pylorus nimmt.

Als drittes Moment für die Verengerung des Anastomosenlumens kommt dann noch folgendes in Betracht: Wir wissen, daß die Schleimhaut im Magen und Darm sehr reichlich bemessen ist, bedeutend reichlicher als z. B. die Serosa. Daher kommt es auch, daß bei Füllung eines Magens oder Darmes bis zum Platzen zunächst nur Serosarisse entstehen. Die Schleimhaut liegt im Darm und Magen nicht glatt, sondern bildet zahlreiche Falten und Erhebungen; außerdem ist sie gegen ihre Unterlage sehr verschieblich. Wir können uns davon bei jeder Magendarmoperation überzeugen, haben wir doch dabei sehr oft mit dem lästigen und störenden Schleimhautprolaps zu kämpfen. Diese Vorstülpung kann so bedeutend sein, daß bei mäßiger Füllung des Magens ein nicht zu großer Schnitt in die Magenwand sogar für Flüssigkeiten völlig verstopft wird. Kelling[4]) hat bei Hunden nach Eröffnung der Bauchhöhle ein Stück des Magens an

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1) W. B. Cannon and J. B. Blake (Boston), Gastroenterostomie and pyloroplastik, an experimental study by means of the Röntgen rays. Division of surgery of the med. school of Harward university. Boston.

2) D'Arcy Power, A years gastrojejunostomies. St. Bartholomews hospital reports 1905, vol. XLI.

3) Rogers, On the present status of the operation of gastroenterostomie. Annales of surgery 1904, no. 4.

4) Kelling, Studien zur Chirurgie des Magens. Langenbecks Archiv 1900 S. 9.

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das Peritoneum parietale angenäht, wie bei einer zweizeitigen Gastrostomie. Wurde dann der Schnitt im Magenfundus angelegt und fielen die Schleimhauträuder so vor, daß sie sich in den Schnitt der Muskularis einklemmten, so entstand daraus ein ziemlich wasserdichter Verschluß. Auf derselben physiologischen Eigenschaft beruht auch zum Teil die Erfahrungstatsache, daß ein glatter Magen- oder Darmschuß mit einem geringkalibrigen Projektil nicht immer an Peritonitis zugrunde geht.

Wenn wir also resümieren, so sind es u. E. drei Momente, welche zu diesem funktionellen Verschluß der Anastomosenöffnung führen. Erstens die mehr oder minder hochgradige Narbenschrumpfung an der Anastomosenstelle, zweitens die Verkleinerung der Magendarmfistel, welche mit der Verkleinerung des ganzen dilatierten Magens durch Beseitigung der Dilatation Hand in Hand geht, und drittens der bei der mobilen Schleimhaut so leicht mögliche Schleimhautprolaps in Verbindung mit der das ganze Hohlorgan verkleinernden Muskelkontraktion beim Verdauungsakte, und zwar kommen dabei für die Anastomose im wesentlichen die Längsmuskeln in Betracht.

Freilich ist auch die Ansicht vertreten worden, daß ein Verschluß an der Anastomose aus dem Grunde einträte, weil sich dort ein neuer Sphinkter gebildet habe. Carle und Fantino[1]) haben z. B. diese Ansicht geäußert. Das ist aber sicherlich nicht der Fall. Einmal ist es von vornherein u. E. nicht recht einleuchtend, wie es kommen soll, daß sich an der Anastomosenstelle ein neuer Schließmuskel, der doch immer einen ziemlich komplizierten Apparat darstellt, bilden soll, und zweitens kann man sich, wie wir es an mehreren, mit Gastroenterostomose versehenen Hundemägen getan haben, an mikroskopischen Schnitten keineswegs davon überzeugen, daß an der Anastomosenstelle eine besondere Anhäufung von Muskulatur vorhanden ist. Man sieht im mikroskopischen Bilde die vom Darm resp. Magen herrührenden Gewebsschichten zu einem mehr oder minder starken Wulst vereinigt. Der Wulst wird, je länger die Anastomose bestanden hat, um so flacher; man sieht auch an der früheren Nahtstelle einige Bindegewebszüge, aber von einer besonderen Anhäufung von muskulären Elementen haben wir nichts finden können.

Mit dieser unserer Auffassung hinsichtlich der Anastomosenstelle steht die Tatsache im Einklang, daß bei all den Fällen unter Gruppe B, die also keine Galle und keinen Pankreassaft im Magen nachweisbar hatten, die Anlegung der Anastomose bereits längere Zeit — Minimum

1) Carle und Fantino, Beitrag zur Pathologie und Therapie des Magens. Langenbecks Archiv Bd. 56.

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1 Jahr 4 Monate — zurücklag. Daß während dieses Zeitraumes eine Reparation der Dilatation und Atonie stattfinden kann, dürfte außer allem Zweifel sein.

Sehr nahe liegt natürlich der Einwurf, wie es denn kommt, daß aber bei den unter Gruppe A untergebrachten 7 Patienten Galle und Pankreassaft im Magen nachweisbar waren. Diese Fälle mit positivem Ergebnis von Galle und Pankreassaft im Magen scheiden sich in zwei Klassen. Die erste Klasse umfaßt diejenigen Fälle, welche sehr bald nach der Operation nachuntersucht worden sind, und bei denen sich naturgemäß Galle und Pankreassaft im Magen finden mußten, weil sich in so kurzer Zeit eine etwa vorhandene Dilatation und Atonie des Magens nicht hat zurückbilden können. In diese Klasse gehören die Fälle V, VI, VII der Gruppe A. Bei allen diesen hat die Zeitdauer von der Operation bis zur Nachuntersuchung im Maximum drei Monate betragen. Für die Fälle I—IV, die auch Galle und Pankreassaft im Magen haben, kann eine befriedigende Erklärung nur dadurch gegeben werden, daß man annimmt, entweder hat sich die Dilatation und Atonie aus irgendwelchen Gründen, die oftmals nicht völlig klar zutage liegen, nicht zurückgebildet oder es hat von vornherein keine erhebliche Dilatation und Atonie vorgelegen, so daß ein Schrumpfen des Magens und damit auch der Anastomosenöffnung nicht eintreten konnte. Sehr bemerkenswert und instruktiv ist in dieser Hinsicht der Fall I unter Gruppe A. Der Patient ist vor fünf Jahren auswärts wegen dauernder Magenbeschwerden gastroenterostomiert worden. Es wurde eine Gastroenterostomia anterior antecolica mit Hilfe des Murphyknopfes gemacht. Das Befinden besserte sich nicht wesentlich und der Patient schleppte sich fünf Jahre lang in ziemlich desolatem Zustande weiter. Schließlich ließ er sich auf unsere Klinik wegen seiner dauernden Magenbeschwerden aufnehmen. Wir fanden bei der funktionellen Magenuntersuchung einen enorm dilatierten Magen, dessen untere Grenze etwa handbreit unterhalb des Nabels stand; im Mageninhalt war sehr viel Galle und Pankreassaft. Die daraufhin vorgenommene Relaparotomie bestätigte unseren Befund hinsichtlich der enormen Dilatation; wir fanden außerdem im Magen den $2^1/_2$ cm im Durchmesser großen Murphyknopf. Die Anastomosenstelle war kaum für die Kuppe des kleinen Fingers durchgängig, also nicht 1 cm im Durchmesser groß. Es ist klar, daß in diesem Falle die Verengerung der anfänglich doch mindestens $2^1/_2$ cm betragenden Anastomosenstelle lediglich durch Narbenschrumpfung stattgefunden haben muß, denn eine Verengerung der Anastomose infolge Rückgang der Magendilatation im allgemeinen war angesichts des noch vorliegenden, enorm dilatierten Magens naturgemäß ausgeschlossen.

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Der im Magen liegen gebliebene Murphyknopf hat u. E. den Grund für das Fortbestehen der Dilatation und Atonie abgegeben; es ist sehr wohl denkbar, daß der dicke Knopf sich gegen den Pylorus gelegt und die Entleerung des Magens beeinträchtigt hat. Wir haben den Knopf entfernt, sonst aber an der Anastomosenstelle, die ja, wenn auch nur sehr gering, doch noch durchgängig war, nichts gemacht. Der Patient hat sich nach der Operation sehr erholt und seine bisherigen Beschwerden verloren. Er hat nicht mehr, wie vor der Operation, an kopiösem Erbrechen gelitten, dagegen an Körpergewicht zugenommen. Leider war die Zeit, während der wir ihn auf der Abteilung hatten, zu kurz, um die Möglichkeit einer Rückbildung der Dilatation des Magens und damit eine Ausschaltung der Anastomosenöffnung zu beobachten.

In ähnlicher Weise müssen alle die Fälle erklärt werden, welche nach längerer Zeit noch Galle und Pankreassaft im Magen aufweisen. Die Anastomose ist offen geblieben, weil sich aus irgendeinem Grunde die Dilatatio ventriculi nicht zurückgebildet hat. So ist z. B. auch bei Fall II unter Gruppe A das Verhalten des Magens völlig klar. Der Patient hatte eine Verschüttung mit starker Quetschung des Abdomens erlitten und etwa 4—5 Monate nach dem Unfall, allmählich einsetzend, mehr und mehr stärker werdende Stauungsbeschwerden von seiten seines Magens bekommen. Die chemische Untersuchung seines Mageninhalts ergab die typischen, oftmals für Karzinom verwerteten Resultate einer Magenstauung: keine freie Salzsäure, reichlich Michsäure, schlecht angedautes Probefrühstück, lange Bazillen usw. Wir konnten unter diesen Umständen ein Pyloruskarzinom nicht mit Sicherheit ausschließen, machten die Laparotomie und fanden am Pylorus zahlreiche, bindegewebige Verwachsungen, welche eine Stenosierung des Pylorus und anschließenden Duodenums bewirkt hatten. Wir lösten die Verwachsungen, fügten aber sofort in Erinnerung der Tatsache, daß sich solche Verwachsungen fast immer wieder bilden, gleich eine Gastrojejunostomie hinzu. Seit der Operation ist der Mann wieder arbeitsfähig, ohne Magenbeschwerden zu haben. Er ist, wie die Tabelle zeigt, 2 Jahre 9 Monate post op. von uns nachuntersucht worden. Hier ist der Pylorus ziemlich sicher fast ganz oder ganz geschlossen. Der Mageninhalt ist also auf einen anderen Ausgang angewiesen. Den bildet die Anastomose, die sich unter diesen Umständen naturgemäß nicht schließen kann. Für die Fälle III und IV sind wir nicht in der Lage eine detaillierte Erklärung geben zu können, warum sich bei ihnen nicht wieder der alte Entleerungsmodus durch den Pylorus mit sekundärer Unwegsamkeit der Anastomosenöffnung eingestellt hat.

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Daß bei völlig geschlossenem Pylorus die Magendarmfistel sich nicht schließen kann, ist selbstverständlich. Dementsprechend findet man auch bei solchen Fällen immer Galle und Pankreassaft im Magen. Wir verweisen in dieser Hinsicht auf die beiden Fälle unter Gruppe D. Fall I hatte ein Conamen suicidii mit roher Salzsäure begangen; es bestand eine schwere Ösophagusstriktur, derentwegen die Patientin auf der Klinik behandelt wurde; es entwickelte sich unter unseren Augen eine völlige Stenose des Pylorus. Der Magen wurde ad maximum dilatiert; er reichte schließlich nach 2—3 Tagen, in denen die Stenose manifest wurde, fast bis zur Symphyse. Wir machten die Laparotomie und fanden den Pylorus völlig undurchgängig. Eine Gastrojejunostomie half dem bedrohlichen Zustande ab. Diese Patientin hat natürlich, da bei völlig geschlossenem Pylorus der Mageninhalt lediglich auf die neue Anastomosenöffnung angewiesen ist, dauernd Galle und Pankreassaft im Magen, da ja die Magendarmfistel unter diesen Umständen naturgemäß offen bleiben muß. Genau so liegen die Verhältnisse bei dem anderen Fall unter Gruppe D. Ihm ist wegen Pyloruskarzinom eine Pylorusresektion nach Billroth II, also völliger Pylorusverschluß und Anastomose zwischen Magen und Jejunum, gemacht worden. Diese Öffnung ist nunmehr die einzig abführende am Magen; infolgedessen schrumpft sie nicht wegen des dauernden Gebrauches; der Magen enthält aber auch dauernd Galle und Pankreassaft.

Bei den Gastroenterostomien mit Braunscher Enteroanastomose scheint zunächst stets Galle und Pankreassaft im Magen zu sein, später jedoch nicht mehr. Wir haben vier derartige Fälle nachuntersucht, welche unter Gruppe C untergebracht sind; zwei, Fall III und IV, bei denen die Zeit zwischen Operation und Nachuntersuchung allerdings erst sehr kurz war (bis zu $1^1/_2$ Monaten), hatten Galle und Pankreassaft im Magen. Bei Fall I (4mal nachuntersucht) hat sich vom 4. Monate ab niemals mehr Galle und Pankreassaft im Magen nachweisen lassen. Der Fall II war besonders bemerkenswert. Hier war bis zum 6. Monate post op. Galle und Pankreassaft im Magen nachweisbar; die Patientin, eine stark nervöse, um nicht zu sagen hysterische Person, welche wegen eines Ulcus ventriculi operiert worden war, litt nach der Operation angeblich noch dauernd unter Magenbeschwerden. Erst vom 7. Monate ab fielen die Nachuntersuchungen hinsichtlich Galle und Pankreassaft im Magen negativ aus; gleichzeitig damit ging es der Patientin auch objektiv besser. Es scheint, als ob sich bei Gastroenterostomien mit Braunscher Anastomose im allgemeinen relativ bald nach der Operation Zirkulationsverhältnisse herausbilden, die ein Einfließen von Galle und Pankreas-

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saft in den Magen zu verhindern vermögen. Daß das auch bei diesen Fällen lediglich durch Verengerung resp. Verschluß der Anastomosenöffnung bedingt wird, glauben wir jedoch nicht. Die Art der Darmschaltung ist wohl hier das schwerwiegende Moment, wenngleich es feststeht, daß die Braunsche Anastomose keineswegs das Einfließen von Galle und Pankreassaft in den Magen zu inhibieren vermag. Naturgemäß wird auch die Lage der Stelle, an welcher die Enteroanastomose gemacht worden ist, in dieser Hinsicht von Bedeutung sein. Liegt sie nahe an der Gastroenterostomie, so ist die Möglichkeit des Einfließens von Darminhalt aus dem zuführenden Schenkel in den Magen größer, als wenn sie weiter davon entfernt liegt.

Es würde nicht in der Absicht dieser Arbeit liegen und auch ihren Rahmen bei weitem überschreiten, wenn wir die in den Tabellen dieser Arbeit niedergelegten Befunde, welche sich auf den Chemismus des Magens nach einer Gastroenterostomie beziehen, genügend würdigen wollten. Wir behalten uns deshalb eine eingehendere Besprechung dieses Teiles unserer Untersuchungen vor.

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(Gynäkologie Nr. 177.)

Im Kampfe gegen das Kindbettfieber.

Ein Mahnwort an Ärzte.

Von

Otto von Herff,

Basel.

M. H.! „Sollte, was Gott verhüten möge, mir nicht gegönnt sein, diese glückliche Zeit, in welcher in und außerhalb der Gebärhäuser der ganzen Welt nur Fälle von Selbstinfektion vorkommen, mit eigenen Augen zu schauen, so wird die Überzeugung, daß diese Zeit früher oder später nach mir unaufhaltsam kommen muß, noch meine Todesstunde erheitern" so schrieb dereinst Semmelweis in wehmütiger Stimmung.

Die großen Arbeiten Pasteurs, Robert Kochs und Listers, die Antisepsis und die Asepsia verbreiteten sich überall, aber außerhalb der Gebärhäuser will das Kindbettfieber noch immer nicht verschwinden. Zwar ist die Zahl dieser Fälle auf etwa ein Drittel der früheren vermindert worden, aber seit Jahren ist ein Stillstand eingetreten, da und dort steigt die Ziffer der Todesfälle dieser sicher vermeidbaren Erkrankung — so z. B. in den großen Städten Preußens, in Bayern um nur einiges zu erwähnen. Ist diese nicht wegzuleugnende Tatsache nicht im höchsten Grade beschämend für die Ärztewelt, wird sie nicht um so trauriger, wenn man bedenkt, wie alljährlich zahllose Menschenleben, vieles Familienglück einer unbegreiflichen Gleichgültigkeit auf diesem Gebiete, nicht selten einem Schlendrian in den Vorbeugungsmaßregeln gegen das Kindbettfieber geopfert werden?

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Viele Tausende von Wöchnerinnen erliegen jährlich einer nahezu sicher vermeidbaren Erkrankung, trotzdem rührt sich fast niemand hierin nachdrücklichst Wandel zu schaffen, das Übel mit der Wurzel auszureißen.[1])

So groß die Verdienste der chirurgischen Ära der Geburtshilfe für den engen Bezirk der operativen Eingriffe sein mögen, so hat sie leider vielfach die Aufmerksamkeit von diesen Fragen abgelenkt. Gilt es doch gegenwärtig da oder dort als „antiquiert" sich mit diesen für die Allgemeinheit, den Staat, der Familie so überaus wichtigen Fragen zu beschäftigen! Trotz alledem, die Frage der Vorbeugung des Kindbettfiebers muß immer wieder auf die Tagesordnung gesetzt werden, das Gewissen so manchen Arztes muß immer wieder und wieder aufgerüttelt werden, schließlich werden diese Tropfen die Gleichgültigkeit aushöhlen, das von Semmelweis so sehnlich erwartete Zeitalter heranbrechen lassen! Hierzu sind Sie, meine Herren Kollegen, die berufensten Vertreter! Helfen Sie in erster Linie nach besten Kräften die Ursachen der betrübenden Häufigkeit des Kindbettfiebers freizulegen, um sie endgültig zu beseitigen! In Ihren Händen liegt die Zukunft der Vorbeugung des Kindbettfiebers, nicht in denen der Gebäranstalten, die mit diesem Feinde vielfach schon fertig geworden sind!

Soll ein Feind bekämpft werden, so müssen alle seine Schliche und Angriffsseiten bekannt werden, man muß die eigenen Schwächen klaren Auges erkennen, um dem Angriff sicher begegnen zu können. Hierzu sollen meine Ausführungen einen kleinen Beitrag geben, in der Hoffnung, daß Sie sich, meine Herren, auch rühren und Ihrerseits zu diesen doch gewiß für die Allgemeinheit und die Familie ungemein wichtigen Fragen Stellung nehmen, daß Sie Ihre wertvolle Erfahrung der Allgemeinheit nicht vorenthalten!

Kindbettfieber ist eine Wundinfektionskrankheit, die in ihrer schweren Form fast nur durch Übertragung von Lebewesen, d. h. Spaltpilzen, sehr selten Sproßpilzen, die aus dritter Quelle stammen, d. h. durch Fremdkeime, die nicht in den Geschlechtsteilen der Frau hausen, entsteht. Eine solche Übertragung kann durch das ärztliche Personal — Arzt, Hebamme, Vorgängerin — stattfinden, oder sie erfolgt durch den Ehemann beim Beischlaf, gelegentlich auch durch

1) Übertrifft doch z. B. in Preußen die Zahl der im Kindbette gestorbenen Frauen — darunter befinden sich die Opfer des Kindbettfiebers — die Verluste an Typhus ganz erheblich. Es starben an Typhus 1905 und 1906 je 0,73 und 0,62, im Kindbett aber 2,13 und 1,97 auf 10000 lebende Frauen berechnet. Wie würden sich die Verhältnisse gestaltet haben, wenn Männer in gleicher Zahl diesen Leiden ausgesetzt wären? Würde nicht alles in Bewegung gesetzt werden?

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die Wöchnerin durch Selbstuntersuchen. Selten sind ein Bad oder andere Eingriffe, Katheterisation, Ausspülung mit schmutzigem oder zerbrochenem Mutterrohre usw. die Ursache. Zieht man die alltäglichen kurzen Wundfieber (Resorptionsfieber) ab, so werden die Eigenkeime, jene Spaltpilze, die in den Geschlechtsteilen der Kreißenden oder Wöchnerinnen hausen, ihrer Trägerin nur sehr selten von sich aus gefährlich. Diese Eigentümlichkeit der Eigenkeime, sie findet sich ja an vielen andern Stellen des Körpers vor, läßt sich vielleicht dadurch erklären, daß auch die Spaltpilze, wie so viele andere Lebewesen, die Eigenschaft besitzen, sich ihnen irgendwie schädlichen und ungünstigen Lebensbedingungen anzupassen. Sie gewöhnen sich und schützen sich gegen die Schutzvorrichtungen des Körpers, so daß sie diesen nicht erliegen, kurz, sie werden immunfest, serumfest. Auf der andern Seite gewöhnt sich der Körper an die Anwesenheit dieser seiner Feinde, indem er an Ort und Stelle die Gewebe örtlich schützt, so daß diese den Angriffen der Spaltpilze nicht erliegen.

Auf der Hand liegt es, daß durch solche Verhältnisse eine Harmlosigkeit der Spaltpilze, eine Verminderung ihrer Angriffskraft (Virulenz) vorgetäuscht wird, die in Wirklichkeit nicht vorhanden ist. Aber diese anscheinend so harmlosen Eigenkeime werden bei einem andern Menschen als gerade bei dem Wirt die schlimmste Erkrankung auslösen, sie werden sich als sehr bösartig erweisen, wenn sie in Geweben des Wirtes eingepflanzt werden, die durch irgendeinen Umstand in ihrer Widerstandskraft irgendwie geschwächt oder nicht mehr wie vorher geschützt sind. Als solche Umstände sind bekannt: mechanische Schädigungen der Gewebe durch Quetschung, Zerreißung, allgemeine Schwächung des Körpers durch Blutverluste, durch Krankheit wie Diabetes usw.

Die Eigenkeime der Geschlechtsteile der Frau, insbesondere Streptokokken, die ja so oft in der Scheide ganz gesunder Frauen hausen, sind fast nur dann zu fürchten, wenn sie unmittelbar in frische, zumal gequetschte Wunden, die begreiflicherweise noch nicht geschützt sein können, eingepflanzt werden. Am ehesten wird diese Bedingung durch ein Trauma gesetzt. Diese Gefahr ist also fast nur bei Operationen, die Verletzungen irgendwelcher Art mit sich bringen, zu fürchten. Unter diesen Umständen kann die Gefahr seitens dieser Eigenkeime, dieser Art von Selbstinfektion, vermittelt durch ein Trauma sehr groß werden. Diese „Selbstinfektion“ lernt man beim Gebrauch der Gummihandschuhe in ihrer ganzen Wichtigkeit kennen. Ihr Vorkommen ist übrigens nicht auf die Frau und ihre Geschlechtsteile beschränkt, sondern jeder Chirurg lernt sie in allen Körperhöhlen

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gründlichst kennen — an ihrem Vorhandensein ist nicht im mindesten zu zweifeln, es sei denn, daß man eine Vogel-Strauß Politik treiben will.

Die Möglichkeit dieser Selbstansteckung vermittelt durch Gewebsschädigung kennen zu lernen ist außerordentlich wichtig. Aus solcher Tatsache folgt, daß ein Kindbettfieber nach irgendeinem Eingriff, nach einer Untersuchung noch lange nicht auf einen notwendigen, ursächlichen Zusammenhang mit irgendeiner Infektion mit Fremdkeimen schließen läßt — es sei denn daß Eigenkeime mit an Sicherheit grenzender Wahrscheinlichkeit als Urheber ausgeschlossen werden können. Das gilt ganz besonders bei Anklagen gegen Ärzte und vor allem gegen Hebammen, den Sündenböcken für so viele ärztliche Verstöße.

Alle Vorbeugungsmaßregeln gegen Kindbettfieber müssen den Hauptwert auf das Fernhalten von Fremdkeimen legen, doch darf die Gefahr der Eigenkeime durchaus nicht unterschätzt oder gar ganz außer acht gelassen werden. Auch dieser Möglichkeit, insbesondere der Selbstinfektion vermittelt durch ein Trauma muß nachdrücklichst begegnet werden.

Diesen beiden wichtigsten Aufgaben einer wirksamen Vorbeugung des Kindbettfiebers in der Theorie gerecht zu werden, erscheint sehr leicht, in der Praxis aber begegnet deren Lösung den größten Schwierigkeiten, wie alljährlich die zahllosen Opfer des Kindbettfiebers nur zu beredt predigen.

Eine Übertragung von Fremdkeimen durch das ärztliche Personal — Arzt, Hebamme — läßt sich mit allergrößter Sicherheit vermeiden, wenn für Keimfreiheit aller benutzten Gegenstände, insbesondere auch der Hände, gesorgt wird.

Ungemein leicht, auch in der allgemeinen Praxis, läßt sich dieser wichtigsten Forderung durch Kochen der Instrumente, Bürsten, Schutztücher, eines Teiles des Nahtmateriales nachkommen. Zudem werden Verbandstoffe, die zuverlässig in Dampf sterilisiert sind, überall zu billigen Preisen abgegeben — nur darf allerdings eine angebrochene Büchse nicht ohne erneute Sterilisation wieder benutzt werden. Seide, noch besser und billiger Zelloidinzwirn, Silk, Draht lassen sich leicht kochen. Catgut, das bei Dammnähten den Vorzug verdient, läßt sich ebenso leicht durch Jodlösungen keimfrei machen oder als trockenes Jodcatgut kaufen.[1]) Kochen und Dämpfen sterilisieren am besten. Beides

1) Auch an dieser Stelle sei darauf hingewiesen, daß trockenes jodiertes Catgut in vorzüglicher Art von der Firma Billmann in Mannheim hergestellt und vertrieben wird.

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ist überall leicht zu haben, ihre Anwendung verursacht nirgends nennenswerte Schwierigkeiten.

Nach Große kann jederzeit auf jedem beliebigen Feuer eine genügende Dampfsterilisation wie folgt vorgenommen werden. Auf dem Boden eines großen Gefäßes, etwa eines Topfes wird ein kleineres gestellt, das die zu reinigenden Sachen aufnimmt. Gießt man in das größere Gefäß etwa 200 ccm Wasser (ein großes Wasserglas) und deckt das Ganze zu, so genügen 10 Minuten des Kochens, um eine genügende Keimfreiheit zu sichern.

Nur die Reinigung der Haut und der Hände begegnet den allergrößten Schwierigkeiten. Am leichtesten lassen sich die Hände reinigen, am schwersten die äußere Scham. Die Desinfektion dieses Teiles ist so schwierig, daß von einer befriedigenden Lösung dieser Aufgabe zurzeit nicht die Rede sein kann.

An der Hand läßt sich mit Sicherheit eine solche Keimverarmung, die an Keimfreiheit grenzt und die die längste geburtshilfliche Operation überdauert, erzielen. Allerdings jene wenigen Keime, die in der Tiefe der Haut vergraben liegen, entziehen sich allen Schädigungen der Desinfizientien. Sie können aber nach und nach auf die Hautoberfläche auswandern, wenn sie nicht irgendwie zuverlässig in ihrer tiefen verborgenen Lage festgehalten werden — solches ist aber heutzutage sehr gut zu erreichen.

Alle Methoden, die die Haut aufweichen, arbeiten einer Keimfreiheit entgegen, darunter auch die Seifenwasserwaschung. Eine Keimauswanderung wird eingeschränkt und selbst völlig gehemmt, je trockener die Haut ist, je mehr sie einschrumpft.

Mit Hilfe dieser beiden wichtigsten Prinzipien läßt sich mit einer an Sicherheit grenzenden Wahrscheinlichkeit zum mindesten eine Keimverarmung, die an Keimfreiheit grenzt, wenn nicht von vornherein eine Keimfreiheit erzielen. Die Mittel hierzu sind folgende:

Mechanische Beseitigung jener Spaltpilze, die auf der Hautoberfläche hausen, durch Reiben und Scheuern bei gleichzeitiger Auflösung der Schmutz- und Fettschichten mit nachfolgender Einsperrung der Keime, die in den zahllosen Fältchen der Haut, in den Haarbälgen, in den Drüsen, zwischen lockern Hornzellen der mechanischen Entfernung unzugänglich geblieben sind. Lebhaft zu begrüßen ist es natürlich, wenn zugleich mit dieser Maßnahme eine chemische Abtötung der zugänglichen Keime erfolgt, soweit solches in den wenigen Minuten, die eine solche Reinigung praktisch dauern kann, überhaupt möglich ist.

Alle Desinfektionsmethoden, die dieser Forderung nicht gerecht werden, müssen ungünstige Ergebnisse zeitigen, in erster Linie Wasser und Seife wie alle jene Maßnahmen, die mit einer Wasserwaschung

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schließen. Alle diese arbeiten den mechanischen Prinzipien der Keimabsperrung geradezu entgegen, sie erleichtern den Hautkeimen der Tiefe das Auswandern anstatt ihnen dieses zu erschweren — und das ist im Prinzip gewiß falsch.

Das mechanische Prinzip des Wegscheuerns der Oberflächenkeime verbunden mit einer Einsperrung der Tiefenkeime (Schleich), meine Herren, wird in Zukunft die Technik der Hautreinigung beherrschen.

Der Glaube an die rasche Wirksamkeit auch der stärksten Antiseptika bei der Hautreinigung — Halogene, Sublimat, Sublamin, Hydrargyrumoxycyanat — ist leider noch sehr verbreitet, er verschuldet so manches Unglück. Alle diese Mittel brauchen für ihre Wirkung in reinen Lösungen längere Zeit, es ist unmöglich, daß sie in wenigen Minuten Keime, die in Schmutz und Fett irgendwie eingehüllt liegen, abtöten — diese allbekannte Tatsache wird vielfach übersehen. Es ist ja so viel bequemer, einige Pastillen in eine Schüssel Wasser zu werfen und sein Gewissen damit zu beruhigen. Das ist nicht nur gedankenlos, sondern oft genug auch gewissenlos.

Keime, die in Flüssigkeiten frei schweben oder die der Oberfläche irgendwelcher Gegenstände — Glasperlen, Seidenfäden — anhaften, sind durch Antiseptika bei entsprechendem Zeitaufwand leicht und sicher zu töten. Liegen Spaltpilze im Fett oder Schmutz wohl verborgen, so versagt die keimtötende Kraft der Antiseptika während der kurzen Zeit einer noch so sehr verlängerten Desinfektion vollständig, auch die stärksten Mittel zerschellen an einer Umhüllung, die an der Tageshand aus Staub, Eiweiß und Fett gebildet wird. Das sind gewiß Binsenwahrheiten, Tatsachen, meine Herren, aber wie viele Ärzte, geschweige denn Hebammen, die allerdings an ihre Vorschriften gebunden sind, beachten sie Tag für Tag nicht?

Das an sich logisch richtige und zutreffende Prinzip der chemischen Abtötung der Hautkeime, die bestenfalls nur die Oberflächen- aber nicht die Tiefenkeime sicher treffen kann, hat die Hautdesinfektion in eine Sackgasse geführt, aus der sich viele, weil sie gestützt wird durch die herrschende Lehrmeinung und ihre einleuchtende Folgerichtigkeit, nicht mehr herausfinden werden.

Die Alkoholwaschung vereinigt in sich die Prinzipien der mechanischen und der chemischen Reinigung, allerdings besonders stark das erstere in glücklichster Weise. Der Weingeist muß daher vollständig in den Vordergrund treten — bis etwas Besseres mit der Zeit gefunden werden wird.

Heißwasser und Seife allein ergibt schlechte Ergebnisse, weil die Haut aufgelockert und der Schmutz aufgewühlt wird. Vielfach erhält

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man den Eindruck, daß durch eine solche Reinigung erst recht die Menge der Keime vermehrt wird. Nur, wenn gleichzeitig eine nachdrückliche mechanische Scheuerung der Haut mit Sand, Marmor, vor allem mit Kieselgur (Saposilikseife), gefolgt von einem festen Abreiben mit einem rauhen trocknen Handtuche stattfindet, kann die Zahl der Keime einigermaßen vermindert werden. Durch das trockne Abreiben werden die losgelösten Spaltpilze gründlicher entfernt. Nicht selten läßt sich eine weitgehende, aber leider noch nicht genügende Keimverarmung herbeiführen. Dieser Seifenwaschung noch eine Reinigung mit irgendeinem Desinfizienz in Wasser folgen zu lassen, kann die Verhältnisse nicht wesentlich verbessern, weil in so kurzer Zeit kein Desinfizienz die Keime töten, jene der Tiefe überhaupt schädigen kann. [1])

Heißwasser und Seife plus Desinfiziens in Wasser ist nicht besser als eine Heißwasserreinigung mit Sand, Saposilikseife plus Abreiben mit einem rauhen Tuche. Diese Methode muß als unzuverlässig fallen gelassen werden.

Die noch so weit verbreitete Anwendung dieser „Desinfektion" ist zweifellos mit in erster Reihe daran schuld, daß das Kindbettfieber nicht abnehmen will, sondern im Gegenteil vielfach noch im Zunehmen begriffen ist. Diese Art der Reinigung wäre auch schon lange vollständig verlassen worden, wenn nicht glücklicherweise die gewöhnlichen Hautkeime einer Tageshand im ganzen genommen eine geringe An-

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1) Gewiß bewirkt die Seife eine gute mechanische Reinigung, da sie durch ihr freies Alkali Schmutzteile auf- und loslöst, diese Teile durch den Seifenschaum, der durch saure Fettsalze erzeugt wird, lockert und sie einhüllt. Diese mechanische Reinigung reiner Seife wird in der Saposilikseife durch feine Kieselgurkristalle ganz außerordentlich gesteigert — die Haut wird sichtlich geglättet durch Abscheuern der lockeren Hornzellen. Zu dieser allbekannten mechanischen Wirkung gesellt sich, jedoch nur freiliegenden Keimen gegenüber eine nicht unbeträchtliche keimtötende Kraft. Diese ist nach Raß am bedeutendsten bei einer heißen 20prozentigen Seifenlösung, sofern sie mit destilliertem Wasser hergestellt wird. Auch kann die Wirkung durch Zusatz von Kresol oder Phenol zu gleichen Teilen beträchtlich gesteigert werden, so daß 3prozentige Phenolseifenlösungen dieser Art genügen. Jeder Gehalt an Kalk im Wasser vermindert jedoch die Wirkung. Auf Grund dieser Untersuchungen versuchte ich 20—50prozentige Seifenlösungen, ich erreichte jedoch keine Keimverarmung, da die keimtötende Kraft der Seife offenbar für die Keime der Tiefe, die geborgen liegen, nicht ausreicht.

Wird nach der Seifenwaschung ein Quecksilbersalz angewandt, z. B. Sublimat, Sublamin, so liegt die Gefahr nahe, daß durch Reste der Seife, oder durch einen etwaigen Kalkgehalt des Wassers diese Verbindungen gebunden oder ausgefällt und in ihren antiseptischen Wirkungen geschwächt werden. Quecksilberoxycyanat macht allerdings eine Ausnahme, es müßte daher den obigen Stoffen weit vorgezogen werden.

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griffskraft (Virulenz) besäßen, und daher nur selten schadeten. Wurde und wird doch noch auf Erden in unzähligen Fällen Geburtshilfe mit ungereinigten Händen getrieben und dennoch erkranken und sterben die wenigsten dieser Frauen! Weil nicht jedesmal nach einer solchen Reinigung, die, wie wir jetzt sicher wissen, ungenügend ist, eine Infektion erfolgt, wird bei so manchem Arzte der Trugschluß begünstigt, daß diese Methode für die Bedürfnisse der Praxis völlig ausreiche. Wer bürgt aber, meine Herren, dafür, daß sich nicht gelegentlich, vielleicht ausnahmsweise auf der Tageshand höchst angriffskräftige Spaltpilze z. B. Streptokokken eingenistet haben? Eine Möglichkeit, die aus naheliegenden Gründen sicherlich am ehesten und am häufigsten an den Händen der Ärzte, vielleicht seltener an den Händen der Hebammen eintreten kann.

Die Hand eines Arztes, der sich nicht streng von infektiösen Stoffen fernhält, ist erheblich gefährlicher als eine noch so schmutzige Tageshand einer entarteten Hebamme. An ersterer Hand werden öfters gefährliche Keime haften, an letzterer brauchen sie nicht vorhanden zu sein.

Tausendfältig sind die Beweise für diese Tatsache. Immerhin möge hier darauf hingewiesen werden, daß Stöckel (Münch. med. Wochenschr. 1908, S. 45) jüngsthin wieder ein solch warnendes Beispiel aufführte. Ein Arzt, dessen Hand mit Anginaeiter beschmutzt wurde, untersuchte seine eigene Frau nach gründlichster Desinfektion — trotzdem starb sie am Kindbettfieber. Der Fall beweist, daß entweder diese gründliche Desinfektion, deren Methode leider nicht erwähnt wird, trotz der besten Absicht doch nicht gründlich gewesen ist, weil sie es an sich nicht sein konnte, oder daß ein anderer ursächlicher Zusammenhang vorgelegen haben kann, der Anginaeiter unschuldig gewesen ist. Im ersten Falle wäre das Unglück mit einer der modernen Alkoholmethoden vermieden worden.

Ganz ungezwungen erklärt es sich, warum so manches Kindbettfieber durch das ärztliche Personal, insbesondere durch den Arzt veranlaßt wird trotz tatsächlich gründlichster und gewissenhaftester Desinfektion — allerdings etwa nach der ungenügenden Heißwasserreinigung plus einem Desinfizienz in Wasser. Man zerbricht sich den Kopf, man forscht nach allen möglichen entfernten Ursachen — aber an das Nächstliegende, an die angewandte, notwendig ungenügende Reinigung, meine Herren, wird nicht gedacht. Hat doch diese in so und so vielen Fällen genützt, freilich nur, und das wird übersehen, weil die Keime der Tageshand verhältnismäßig harmlos sind und sich zufälligerweise keine angriffskräftigen darunter befunden haben. Eine Selbsttäuschung, die schon sehr viel Unheil angerichtet hat und leider noch weiterhin Unglück herbeiführen wird!

Die großen Opfer, die das Kindbettfieber alljährlich fordert, müssen das ärztliche Gewissen schärfen, sie müssen dazu führen, die

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Methode der Heißwasserseifenreinigung plus einem Desinfizienz gelöst in Wasser vollständig zu verwerfen.

Asepsis gewährleistet durch mechanische Maßnahmen, meine Herren, ist das Ziel das erstrebt werden muß — darin hat Schleich durchaus recht!

Das mechanische Prinzip der Entfernung der Oberflächenkeime neben einer Einsperrung der Tiefenkeime durch Einschrumpfung und Entwässerung der Haut muß in der Händereinigung den breitesten Raum einnehmen. Eine solche Reinigung kann einer Heißwasserseifenwaschung folgen (Ahlfeld). Noch besser freilich ist es diese vollständig fallen zu lassen, weil sie dem Prinzipe der Eintrocknung der Haut entgegenarbeitet. Die Desinfektionsmethode wird denkbar vereinfacht und abgekürzt, sie wird zu einer *Schnelldesinfektion* (Schumburg), ohne irgendwie dabei an Zuverlässigkeit einzubüßen.

Der nächstliegende und überall erreichbare Stoff ist der Weingeist. *Ohne Alkohol keine irgendwie genügende Keimarmut oder Keimfreiheit* heißt es überall und mit vollem Recht meine Herren! Folgt aber der Anwendung des Alkohols, der die Haut eintrocknen und einschrumpfen läßt, überdies Keime tötet und weithin in die Tiefe der Haut eindringt, eine Wasserwaschung plus Desinfiziens in Wasser — Fürbringersche Methode — so wird die Haut wieder aufgeweicht. Das Schlußergebnis wird verschlechtert, weil das wichtigste mechanische Prinzip einer Keimverarmung der Haut und damit auch der Alkoholanwendung vernichtet oder doch ganz wesentlich abgeschwächt wird. Viel zweckmäßiger und vernünftiger wäre es doch, wenn jene, die sich nun einmal von der Vorstellung durchaus nicht freimachen können, als ob z. B. Sublimat oder gar Lysol in wenigen Minuten Spaltpilze, die in Eiweiß- oder Fetthüllen oder sonstwie geschützt liegen, töten oder irgendwie unschädlich machen können, die Alkoholwaschung am Schlusse setzen, also Heißwasser, Seife plus Desinfiziens in Wasser plus Alkohol anwenden. Oder, was kürzer und weit bequemer wäre, dem Weingeist gleich das gewünschte Desinfiziens zufügen, somit sich nach Engels Methode reinigen, d. h. Heißwasser, Seife plus Desinfiziens in Alkohol gelöst z. B. 2 % Bazillol, Lysoform oder 5 % Formizin (Füth) od. dgl. m. anwenden.

Die Heißwasser-Alkoholdesinfektion mit oder ohne Zusatz eines Desinfizienz — *Ahlfeld, Engel*, ist für alle, die Heißwasser und Seife nicht fallen lassen mögen, die zurzeit beste. Sie gewährleistet die größtmögliche Zuverlässigkeit und Sicherheit ihrer Wirkung. Nicht selten wird

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eine Keimfreiheit erzielt, *unter allen Umständen* aber eine Keimverarmung, die an Keimfreiheit grenzt, die daher praktisch einer solchen gleichkommt.

Ahlfelds Reinigung, wie übrigens die nach Engel empfiehlt sich auch aus dem Grunde ganz besonders für den Hausarzt und die Hebamme, weil sie weit bequemer und einfacher ist als die Fürbringersche Methode.

Seife wie Wasser weichen die Haut auf, sie befördern das Auswandern der Tiefenkeime ohne sie zu vernichten. Sollte es daher nicht zweckmäßiger sein, diese beiden Mittel in der Händereinigung vollständig fallen zu lassen?

Wasser schaltet die Seifenspiritusreinigung nach Mikulicz und Vollbrecht aus. Die Endergebnisse sind leider ungleich, wohl infolge der verschieden starken Aufweichung der Haut durch den alkalischen Seifenzusatz. Neben weitgehendster Keimverarmung findet man Fälle mit ungenügender Wirkung, ohne daß der Grund ersichtlich wäre. Immerhin ist die Methode praktisch brauchbar und wird auch zurzeit von vorzüglichen Chirurgen durchgehendst angewandt.

Wasser und Seife vermeidet Schumburg, dem unzweifelhaft das Verdienst zukommt, die Frage der sichern Schnelldesinfektion untersucht und der Lösung näher gebracht zu haben. Schumburgs Ergebnisse werden unter anderen von Kolle und Tavel in Bern, denen ich mich anschließe, bestätigt. Nach Reinigung der Nägel wird die Tageshand unter Verzicht auf Wasser und Seife mittelst Watte, Gaze oder Flanell, was ich vorziehe, um die so gefährlichen ungekochten Bürsten auszuschalten, mit einer Lösung von zwei Teilen Alkohol auf einen Teil Äther bei einem Zusatz von $^1/_2$ % Salpetersäure abgerieben. MeineVersuche wurden mit und ohne vorgängige Reinigung der Nägel — letzteres soll natürlich nicht als Vorbild dienen — teils mit einer Lösung von zwei Teilen Alkohol und einem Teil Azeton, teils mit Alkohol und Azeton zu gleichen Teilen ausgeführt. Die Tageshand wurde mit dieser Lösung und einem Flanellappen, der einmal gewechselt wurde, kräftig abgerieben. Die Ergebnisse sind in jeder Beziehung zufriedenstellend. Von beiden Händen zugleich gingen in der Regel nur 2—3—4 Keime, die sehr wohl aus der Luft stammen konnten, auf, ab und zu blieben die Platten steril — d. h. es wurde eine Keimverarmung, die an Keimfreiheit grenzt, die praktisch einer Keimfreiheit gleichkommt, erzielt. Diese Schnelldesinfektion, die nicht mehr wie 4—5 Minuten in Anspruch nimmt und nur eine einzige Maßnahme erfordert, ist denkbar kurz und bequem. Durch Eintrocknen und Entwässern der Haut wird den Spaltpilzen, die noch an der Hand haften geblieben sind, die wesentlichsten Bedingungen für ein

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Auskeimen entzogen, sie können sich während der Dauer einer Operation nicht vermehren. Alkohol-Azeton wirkt daher außerordentlich entwicklungshemmend auf Keime der Tiefe wie der Oberfläche.

Das auffallendste an dieser Desinfektionsmethode ist aber die Dauerwirkung. Bei einer ganzen Anzahl langdauernder Operationen, die nach dieser Schnelldesinfektion und ohne Handschuhe ausgeführt wurden, bei denen also genug Luftkeime auf die Hände fallen konnten, fanden sich bei der Schlußentnahme, selbst wenn dieser ein 8—10 Minuten langes Aufweichen der Haut mit $^1/_{10}$ % Natronlauge vorausgegangen war, nur spärliche Keime an beiden Händen zugleich vor. Die Zahlen schwankten zwischen 0 und 18, nach einer Natronlaugeaufweichung zwischen 1—36 — d. h. weniger als Luftkeime auf die Hände während der Operationszeit gefallen sein konnten. Die Dauerwirkung erstreckt sich auf 1—2 Stunden nach der Desinfektion. Sie wird nicht durch häufiges Abwaschen der Hände in sterilem Kochsalzwasser oder durch Aszites oder durch Blut und sonstige Körpersäfte vermindert. Die Desinfektion ist so sicher, daß Handschuhe überflüssig erscheinen können.[1])

Kein Zweifel, daß die Schumburgsche Methode, namentlich in meiner Abänderung mit Azeton, eine ganz ausgezeichnete und zuverlässige Schnelldesinfektion ist, die, insbesondere letztere, alles Wünschbare leistet.

Schumburg und neuerdings v. Brunn haben auch Versuche mit reinem Weingeist, mit dem gewöhnlichen denaturierten Brennspiritus angestellt, die recht zufriedenstellend ausgefallen sind. Eigene Versuche habe ich damit noch nicht ausführen können, auch ist mir über eine Dauerwirkung, die doch die Hauptsache bei allen Desinfektionsmethoden ist, noch nichts bekannt geworden.

Azeton habe ich statt Äther gewählt, einmal weil es billiger als dieser ist, sodann und in erster Linie weil es vermöge seiner fettlösenden Eigenschaft ein ausgezeichnetes Putzmittel für die Haut ist, weil es ein vorzüglicher Ersatz für Seife ist, das entgegengesetzt der Seife die Haut nicht aufweicht, sondern diese, gleichzeitig in vorzüglicher Weise eintrocknet und härtet. Die Tiefenkeime werden derart eingesperrt, daß die $^1/_{10}$ prozentige Natronlaugenaufweichung nicht genügt, sie zu befreien, geschweige denn andere Flüssigkeiten, wie steriles Kochsalzwasser, Aszites, Blut usw. Wenig angenehm ist der Geruch des Azetons; immerhin ist er geringer als jener des Äthers. Leider ist Azeton feuergefährlich, so daß Vorsicht bei Licht durchaus geboten ist. Meiner Erfahrung nach reizt Azeton die Haut nicht, im Gegenteil beobachte ich, daß ein eingewurzeltes Alkoholekzem meiner Hände sich bessert. Die Ketone sollen eine geringe keimtötende Eigenschaft besitzen, doch scheint diese bei dem Azeton sehr schwach zu sein, indes habe ich über diesen Punkt nur geringe Erfahrung.

Wederhake (Deutsch. med. Wochenschr. 1907, Nr. 15) empfiehlt eine Jodtetrachlormethanlösung, besonders auch, weil sie sehr tief in die Haut eindränge. Wasser und Seife sind ebenfalls nicht erforderlich. Tetrachlormethan, CCl_4, ist

1) Herr Dr. Oeri wird demnächst diese Versuche genauer mitteilen.

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nicht feuergefährlich, es löst mit Leichtigkeit Öle, Paraffin, Kautschuk — ich habe bisher keine Gelegenheit gehabt, mich mit dieser Methode näher zu befassen.

Die Kenntnis, daß es mit keiner der früheren Desinfektionsmethoden, insbesondere der Fürbringerschen, gelingt, eine an Keimfreiheit grenzende Keimverarmung der Hände auf die Dauer zu erzielen, führte, da sich die Zwirnshandschuhe Halsteds (1891) nicht bewährten, zur Einführung der Gummihandschuhe durch Zoege von Manteuffel (1895). Diese wurden von Friedrich wesentlich verbessert. Als Vorläufer der Handschuhe kann die Empfehlung Murphys (1894)[1]) gelten, die Hände mit einer 4prozentigen Guttaperchabenzinlösung, das Operationsfeld mit dem rascher trocknenden Guttaperchaazeton zu bestreichen — auf welche Vorschläge ich noch später zu reden kommen werde. Gummihandschuhe lassen sich durch Dampf, weniger zweckmäßig durch Kochen, im Notfalle auch durch Heißwasserseife und Alkohol sicher keimfrei machen — das ist nicht zu bezweifeln, doch dürfen sie keine Löcher haben, das ist unbedingte Voraussetzung. Der Vorteil der sicheren Keimfreiheit ist so groß, daß manche in deren Gebrauch das beste Prophylaktikum gegen Kindbettfieber sehen (Döderlein). Leider haften den Handschuhen mancherlei Nachteile an, die damit nicht aus der Welt geschafft werden, daß sie als nicht stichhaltig kurzweg erklärt werden. Auch verfügen wir jetzt über Methoden der Händereinigung, die eine solche Keimarmut in wenigen Minuten in der bequemsten Weise gewährleisten, daß man sich füglich fragen muß, ob die Handschuhe tatsächlich eine solch größere Sicherheit gewähren, daß ihre nicht unbeträchtlichen Nachteile gerne in den Kauf zu nehmen wären. Besonders zu erwähnen sind: die Glätte, ferner die Herabsetzung des Tastgefühles, die geringe Haltbarkeit und Kostspieligkeit, nicht zu vergessen die Zerreißlichkeit. Ich bin seit vielen Jahren an Handschuhe und Fingerlinge gewöhnt, aber noch heute ist mir bei gewissen geburtshilflichen Eingriffen die Glätte und die Herabsetzung des feinen Tastgefühls höchst unangenehm. So wird mir das Sprengen der Eihäute außerordentlich erschwert, die Ablösung der Placenta unsicherer, das Fühlen einzelner Eihautfetzen fast unmöglich gemacht. Habe ich doch vor wenigen Wochen erlebt, daß ein sehr tüchtiger und gewandter Arzt, der das Fehlen eines Plazentarlappens erkannte, diesen trotz sorgsamster Abtastung der Uterushöhle nicht finden konnte, schließlich glaubte sich geirrt zu haben. Aber einige Tage später wurde ein handtellergroßes Plazentarstück ausgestoßen! Grund? Die Austastung war wegen Lues mit Handschuhen

1) Journal of American Association 1904, no. 12.

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vorgenommen worden. Welche Schwierigkeiten mag drum ein Arzt, der nicht so wie ich an den täglichen Gebrauch von Handschuhen und Fingerlingen gewöhnt ist, zu überwinden haben, muß nicht sein Eingreifen unsicher werden? Man erkundige sich nur einmal danach! Und was die Zerreißlichkeit anbelangt, so sah ich erst neulich auf meinem Kreißsaale, wie bei einer allerdings etwas schwierigen Extraktion nach Deventer-Müller ein neuer Handschuh quer einriß und in Fetzen ging! Das hatte im vorliegenden Falle ja nichts zu sagen, da zuvor die Hand nach Ahlfeld zum mindesten keimarm gemacht worden war, und diese Keimarmut sich unter einem trockenen Handschuhe nachweislich stundenlang erhält. Wie oft, meine Herren, entstehen kleine Risse, wie oft haben neue Handschuhe ganz kleine und kleinste Defekte? Man achte doch nur einmal darauf! Ist die Hand nicht vollkommen trocken, bildet sich ein „Handschuhsaft", der oft genug voll Spaltpilzen ist, so quillt er nach außen und kann infizieren. Auf der andern Seite können Flüssigkeiten von außen unter den Gummi eindringen und den Arzt erheblich gefährden. *Unter allen Umständen zwingt die Zerreißbarkeit der Handschuhe die Hand zuvor auf das allersorgfältigste zu reinigen.* Geschieht solches mit einer der besprochenen Alkoholmethoden, die ja eine anhaltende, weitestgehende Keimarmut, die an Keimfreiheit grenzt, die unter allen Umständen für die Dauer einer geburtshilflichen Operation weit ausreicht, sicher gewährleisten, so sind Handschuhe eigentlich überflüssig, da sie praktisch kaum einen größeren Schutz verleihen können.[1]) Das sind Tatsachen, die sich durch noch so volltönende Worte der eigenen Überzeugung des Gegenteils nicht aus der Welt schaffen lassen und die keinen anderen logischen Schluß gestatten als den eben geführten. Handschuhe haben auch in der Tat die Ergebnisse der Kliniken nicht im geringsten zu bessern vermocht, was doch unbedingt der Fall sein müßte, wenn wirklich den Handschuhen ein so großer Vorteil zukommen würde. Um diesen Punkt gehen die Anhänger der Handschuhe herum — aber damit wird diese wichtige Tatsache nicht ausgeschaltet! Der Nichtgebrauch der Handschuhe bedeutet daher noch lange nicht einen Mangel an Gewissenhaftigkeit (Jung) oder gar einen Leichtsinn, wie kurzsichtige Kritik, der die Wertigkeit

1) So schreibt Durlacher in der Ärztlichen Rundschau 1907, Nr. 33 aus der Praxis heraus: Den geübten Praktiker kann zwar hier (d. h. bei zuvor mit Eiter beschmutzten Händen) das Operieren mit Gummihandschuhen große Vorteile bieten; der Anfänger wird aber durch die Tastsinnesbeschränkung für die Handschuhe weniger Sympathie bekunden. — Jedenfalls kommen die ganz vereinzelten Keime, die sich von einer Hand ohne Handschuhe entfernen lassen, nicht in Betracht, gegenüber der Menge Keime der Scham und Scheide, die mit verschleppt werden.

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der neueren Desinfektionsmethoden offenbar vollständig unbekannt geblieben ist, behaupten könnte.

Der außerordentlich unschätzbare Wert der Handschuhe liegt auf einem anderen Gebiete:

Schutz der eigenen Hand gegen Beschmutzung mit gefährlichen Fremdkörpern aus anderen Quellen, Schutz gegen eigene Erkrankung, Ermöglichung der eigenen Tätigkeit, wenn die Hand nicht ganz frei von kleinsten Rillen, Schrunden und Wunden aller Art ist. Gerade diese unscheinbaren Hautverletzungen beherbergen Spaltpilze, darunter gewiß nicht selten angriffskräftige Formen, die, als Fremdkeime in frische Geburtswunden eingepflanzt, die schwersten Erkrankungen auszulösen vermögen.

In der Ermöglichung des Eigenschutzes, der sogenannten Noninfektion, liegt der wahre Wert der Handschuhe, der selbst ein unersetzlicher wäre, wenn dieser Eigenschutz sich nicht auf dem Wege Murphys in billigerer Weise ermöglichen ließe! Eine tiefe Wahrheit liegt in Kochers Anschauung, gewiß eines der maßgebendsten Chirurgen der Jetztzeit, dem nicht Mangel an Gewissenhaftigkeit oder gar Leichtsinn vorgeworfen werden kann, daß man nicht während einer Operation, sondern in der Zwischenzeit Handschuhe tragen solle!

Betont muß werden, daß ein Selbstschutz nur bei unverletzten Handschuhen vorhanden ist. Zweimal habe ich trotz Handschuh Wundrose an meinem linken Arm davongetragen, der Gummi mußte kleinste Löcher besessen haben. Daß in solchen Fällen die Gefahr bei Handschuhen größer ist als ohne diese liegt auf der Hand. Immerhin scheinen diese persönlichen Erfahrungen Ausnahmen zu sein.

Der richtige und sachgemäße Gebrauch von Handschuhen vereinfacht nicht die Desinfektion, sondern erschwert und verteuert sie namentlich nicht unerheblich. Höhere Ansprüche an die Gewissenhaftigkeit, an die Bequemlichkeit und an die Zeit des Arztes werden gestellt, als es irgendeine der bewährten Alkoholmethoden nach Ahlfeld, Engel, oder Schumburgs Schnelldesinfektion fordern. Das sind Gründe genug, um danach zu streben, die Handschuhe für die Praxis überflüssig zu machen.

Die unverkennbaren Nachteile der Handschuhe, das Bestreben, deren Vorteile auch auf andere Weise zu erreichen, insbesondere das mechanische Prinzip der Keimeinsperrung in der Händereinigung (Schleich) zu vervollkommnen, führte zu aussichtsreichen Versuchen, die Hautkeime in ihren Schlupfwinkeln fest zu bannen. Der Versuche Murphys, eine Art Gummihandschuh zu schaffen, habe ich soeben gedacht, ich selbst besitze darüber keine Erfahrung.

In einfacher, allerdings in wenig sicherer Weise läßt sich der Forderung der Keimabsperrung nachkommen durch das von alters

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her bekannte Einreiben der Haut mit Öl, überhaupt mit Fett oder Vaseline. Ich selbst habe für das Operationsfeld mit gutem Erfolge längere Zeit Jodipin verwandt. Alle diese Mittel verschmieren, außerdem kann man sich leicht davon überzeugen, daß sie nicht sicher sind, weil sie sich zusamt den Hautkeimen, die nicht abgetötet wurden, abstreifen lassen. Durchaus brauchbar ist die Desinfektionsmethode Heußners: 5—10 Minuten langes Abreiben der trockenen Hand mit 1‰ Jodbenzinlösung, — statt dessen habe ich Jodazeton mit gleicher Wirkung versucht, — welcher etwa 10% Paraffinöl zur Schonung und Deckung der Haut zuzusetzen ist. Verlangt muß aber werden, daß solche Schutzmittel eintrocknen und der Haut fest anhaften, wenn sie Gummihandschuhe ersetzen sollen. Döderlein verwendet daher gelöstes Guttapercha, Gaudanin im Handel genannt, doch ist dieses Mittel, abgesehen von der Umständlichkeit seiner Anwendung, auf einem Operationsfeld sehr brauchbar, aber nicht für die Hände geeignet.

Viel zweckmäßiger erscheint das Wichsen (Bohnen) der Haut mittelst Chirosoter, einer Lösung verschiedener Wachsarten in Tetrachlorkohlenstoff, die gefärbt und ungefärbt in den Handel kommt.[1]) Diese Wachslösung, die besonders von Klapp empfohlen wird, gewährleistet, selbst wenn sie auf eine völlig unvorbereitete Tageshand aufgetragen wird, eine solche weitgehende Keimarmut, die an Keimfreiheit grenzt, wie sie nur mit den Alkoholmethoden erreicht werden kann. Diese ist zudem dauernd, weil Tiefenkeime nicht mehr auswandern können und der Wachsüberzug nicht so bald abblättert. Zur Anwendung eignen sich nach den Versuchen Meißners, dem ich hier folge, sowie nach meinen eigenen Erfahrungen — ich verwende seit langem Chirosoter zur Abdeckung aller Operationsfelder, einschließlich des der Scham mit allerbestem Erfolge — eine möglichst trockene Haut oder eine solche, die mit Seifenspiritus oder nach Ahlfelds Methode, oder, noch besser, mittelst der Schumburg-Schnelldesinfektion oder mit Alkohol-Azeton gereinigt wurde. Je trockener die Haut, die Hände sind, desto besser haftet der Chirosoterüberzug, desto sicherer und dauernder werden die Spaltpilze auf und in der Haut festgeleimt. Sollen Unternagelräume, deren Bedeutung übrigens übertrieben wird, gewichst werden, so müssen die Nägel so kurz wie nur möglich geschnitten werden, eine Forderung, die auch sonst gestellt

1) Chirosoter, der leicht flüchtig aber nicht feuergefährlich ist, wird von der Firma Krewel u. Ko., Köln a. Rhein hergestellt. Die mit Sudanrot gefärbte Lösung ist besonders für das Operationsfeld zu empfehlen, weil dadurch ein Zuviel und somit unangenehme Hautreizungen leicht vermieden werden.

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werden muß. Gigerl- und Chinesennägel bei einem Operateur weisen auf höchst üble Gewohnheiten in der Desinfektion hin. Der Chirosoter ist erheblich billiger als Gummihandschuhe, steht jederzeit bereit, läßt sich leicht mitführen und bedarf als nicht feuergefährlich keiner besonderen Vorsicht. Ferner ist dieses Mittel ein gutes Hautkosmetikum und erleichtert den Selbstschutz. Als Nachteil teilt der Chirosoterüberzug die Schlüpfrigkeit mit den Gummihandschuhen, auch vertragen einzelne Personen den Tetrachlorkohlenstoff nicht. Ich wenigstens bekomme eine stark juckende Hautentzündung, die mehrere Tage anhält, doch bin ich in dieser Beziehung ausnahmsweise besonders empfindlich. In den recht zahlreichen Fällen, in denen ich bei Scheidenoperationen Chirosoter auf die doch so empfindliche äußere Scham angewandt habe, sind bisher keine besonderen Reizerscheinungen aufgetreten. Mit einem solchen Wachshandschuh läßt sich das Prinzip des Selbstschutzes, d. h. der Noninfektion, in weitestgehender Weise durchführen. Ich freue mich hier sagen zu können, daß Kollegen sich dieses Schutzmittels mit bestem Erfolge z. B. bei Sektionen bedienen und damit sehr zufrieden sind. Diese ziehen den Chirosoter entschieden den so leicht verletzbaren und teuren Handschuhen vor. Meine Herren, ich stehe daher nicht an zu erklären:

Der Chirosoterüberzug, das Wichsen der Hand, besonders aber der Haut ist eine Methode, die der Beachtung der Ärzte in jeder Beziehung wert ist, die namentlich auf dem Gebiete des Selbstschutzes die so wichtige Handschuhfrage in glücklichster Weise zu lösen scheint. Die Handschuhe erscheinen entbehrlich. Chirosoter und Schumburgsche Schnelldesinfektion neben Ahlfelds oder Engels Methode leisten überdies auf dem Operationsfeld das zurzeit Erreichbare.

Die Anwendung des Chirosoters ist erheblich einfacher als die des Gaudanins und daher ist es diesem vorzuziehen. Nach Reinigung der Unternagelräume wird auf die möglichst trockene Hand der Chirosoter unter Berücksichtigung der Nagelräume aufgesprüht oder auch einfach aufgegossen und in die Haut fest eingerieben. Will man die Hand oder die Haut eines Operationsfeldes zuvor reinigen, was doch gewiß nur von größten Vorteil sein kann, so geschehe dieses nach einer der Alkoholmethoden, insbesondere mit Alkohol-Azeton, um eine möglichste Trockenheit zu erzielen. Jodlösungen erübrigen sich von selbst, ein Punkt, der wegen etwaiger Hautreizungen nicht ganz ohne Bedeutung ist.

Die Scham einer Frau keimfrei zu machen, ist eine noch ungelöste Aufgabe, da die zarte Haut dieser Teile sehr empfindlich ist. Und doch muß bei dem ungeheuren Reichtum an Spaltpilzen, der gerade dieser Gegend eigentümlich ist, diese Frage erledigt werden, weil bei einem operativen Eingriff jederzeit die Gefahr der Keimverschleppung in die Tiefe der Teile besteht.

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Die wohl allgemein übliche und außer mir ohne Narkose befolgte Reinigung mit Heißwasser-Seife plus Desinfiziens (Sublimat) in Wasser hat nicht mehr Wert als irgendeine gewöhnliche Seifenwaschung, welch letztere ja als völlig ungenügend genugsam bekannt ist. Man untersuche nur einmal diese Gegend auf ihren Keimgehalt und man wird überrascht sein von dem geringen Ergebnis dieser „gründlichen Desinfektion". Man begreift aber auch, wie so manche durch scharfe Logik ausgezeichnete Geburtshelfer von einer solchen Scheindesinfektion völlig absehen wollen, die Scham unberührt lassen, um deren Keime nicht noch mehr aufzurühren und in Bewegung zu setzen zumal auch ein sorgfältig enthaarter Haarboden sich mit solchen Mitteln nicht im geringsten keimarm machen läßt.

Die besten Ergebnisse an der Scham erhielt ich mit einer Alkohol-Azetonreinigung. Mehrfach gelang es mir, eine sehr weitgehende Keimverarmung zu erzielen, niemals aber Keimfreiheit wie an der Hand. Bei gynäkologischen Eingriffen suche ich sie mir mit einem Chirosoterüberzug zu sichern. Aber eine solche Reinigung läßt sich ohne Narkose nicht durchführen, sie ist somit für die gewöhnliche Geburtshilfe ausgeschlossen. Gegenwärtig wende ich vor einem geburtshilflichen Eingriffe, sofern er Narkose erfordert, Ahlfelds Methode, auch Mikulicz' Seifenspirituswaschung an. Vielleicht gehe ich aber zum Alkohol-Azeton über, ob mit Chirosoterüberzug, das weiß ich allerdings noch nicht. Ich halte diese Frage übrigens für ziemlich nebensächlich.

Die zurzeit bestehende Unmöglichkeit, die weibliche Scham ohne Narkose irgendwie keimfrei zu machen, verbunden mit der täglichen Erfahrung, daß die hier hausenden Spaltpilzarten in der Regel verhältnismäßig unschuldig für ihre Trägerin sind, sofern sie nicht in frische Geburtswunden durch ein Trauma eingepflanzt werden, hat, wie gesagt, einzelne Geburtshelfer, so Krönig, dazu geführt, auf jedwelche Reinigung der Schamgegend zu verzichten. Ich gehe nicht so weit, weil ich der Ansicht bin, daß man stets das zurzeit Erreichbare leisten soll, wenn es auch zunächst noch so unvollkommen ist. Immerhin muß ich nach besserer Erkenntnis der Sachlage den scharfen Ausdruck, den ich in meiner Abhandlung über das Kindbettfieber gebraucht habe, nämlich, daß die Unterlassung der Reinigung der Scham ein Kunstfehler sei, als viel zu weitgehend zurücknehmen. Ich betone aber, daß durch Rasieren der Scham und ihre Reinigung mit einer der Alkoholmethoden eine recht beträchtliche Keimverarmung erzielt wird, die so ganz ohne Wert doch nicht sein dürfte, daß bei Anwendung des Chirosoters eine Keimverarmung, die an Keimfreiheit grenzt, zu erreichen ist.

Die Forderung, die Scheide von ihren Eigenkeimen zu befreien, läßt sich theoretisch wohl begründen, praktisch stößt aber eine solche Maßregel auf große Schwierigkeiten, sicherlich ist sie aber in vielen Fällen unnötig. Durch Scheidenspülungen läßt sich eine Keimfreiheit gar nicht oder doch sehr schwer erzielen, hingegen ist eine weit-

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gehende Keimverarmung, bedingt durch Abschwemmen der lose sitzenden Spaltpilze, eher zu erreichen.

Bei normaler Geburt mit geringstem Geburtstrauma sind die Scheidenkeime kaum zu fürchten, zumal viele von ihnen unter der Geburt durch das Fruchtwasser, nachhaltiger jedoch durch den Fruchtkörper und die Nachgeburt ausgewischt werden. Die übriggebliebenen Spaltpilze steigen erst zu einer Zeit im Verlaufe des Wochenbettes in die Gebärmutter, zu der die Geburtswunden bereits geschützt sind, schwere Erkrankungen kaum noch zustande kommen können. Von Vorteil sind daher nach meiner Erfahrung und Überzeugung — und darin fühle ich mich eins mit Fehling — Scheidespülungen vor einer jeden geburtshilflichen Operation. Unbedingt notwendig sind sie bei mangelhaftem Schluß des Scheidenmundes infolge Dammriß, bei Vorfällen aller Art, zumal wenn gleichzeitig Cervixrisse vorhanden sind. Auch bei einer Infektion, kenntlich an Fieber unter der Geburt oder an einem übelriechenden Ausfluß, sind sie am Platze, ferner wenn zwischen dem Eisacke und der Uteruswand operiert werden muß, z. B. vor und nach Nachgeburtslösungen. Für diese letzteren beiden Fälle müssen als weitere Vorbeugungsmaßregel neben den Scheidenspülungen noch Gebärmutterausspülungen ausgeführt werden. Die Überlegung ist gewiß nicht falsch, die Möglichkeit muß zugegeben werden, daß mit der Verminderung der Zahl der Keime die Wahrscheinlichkeit, daß solche in Geburtswunden verschleppt werden oder selbständig einwandern, entsprechend geringer wird. Überdies ist die Zahl der eigentlichen Krankheitserreger, deren Angriffskraft infolge ungenügender Ernährung zunächst noch geschwächt ist, klein. Jedenfalls können solche Ausspülungen nicht schaden, sondern nur nützen.

Die Berechtigung, ja den Nutzen vorbeugender Scheiden- und Gebärmutterausspülungen glaube ich, durch meine und anderer klinische Erfahrung in jeder Beziehung sicher gestützt zu sehen.

Immer und immer wird gegen Scheidenspülungen der Vorwurf erhoben, daß solche schaden, selbst von Verteidigern der Spülungen vor Operationen, wiewohl ein etwaiger Schaden sich in solchen Fällen ganz besonders geltend machen müßte, was aber hierbei direkt verneint wird! Die glänzenden Ergebnisse der Kliniken, bei denen grundsätzlich vorbeugende Scheidenspülungen gemacht werden, lehren laut das Gegenteil, sie werden daher bei Erörterung dieser Fragen gern als unbequem beiseite geschoben! Insbesonders die Ziffern Würzburgs unter Hofmeier[1]) mit nunmehr über 9000 Geburten, bei denen Sublimat-

1) Weitere Beispiele siehe Das Kindbettfieber in v. Winckels Handbuch der Geburtshilfe Bd. 3, 2. Teil.

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ausspülungen gemacht worden sind, beweisen in einwandfreier Weise, weil es sich um konstante Ergebnisse handelt, — die Fehlergrenze liegt unter 1 — daß die Scheidenspülungen keinerlei Schaden angerichtet haben. Beobachtet wurden, bei 11,5% Gesamtmorbidität, 7,3% Kindbettfieber, worunter nur 2,3% schwere Formen gewesen sind. Einen Nutzen mag man, wenn man durchaus will, weil Vergleichszahlen fehlen, trotz dieser glänzenden Ergebnisse bestreiten, aber einen Schaden zu behaupten erscheint widersinnig.

In solchen heiklen Fragen können kleine Zahlen nichts beweisen. Ich habe solches bereits Baisch gegenüber an anderer Stelle rechnerisch nachgewiesen und bin noch nicht widerlegt worden.[1]) Baisch wiederholt (Medizinische Klinik 1907, S. 281), daß Scheidenspülungen schadeten, da bei seinen Gespülten um das Doppelte mehr Fieber als bei den Nichtgespülten beobachtet wurde. Urteilen Sie selbst, meine Herren! Mit der Zuverlässigkeit eines wissenschaftlichen Experimentes soll bewiesen worden sein, daß Scheidenspülungen vor der Geburt immer schädlich seien. Denn von

500 Nichtgespülten fieberten insgesamt 8%, davon genital 5,2%
500 Gespülten „ „ 12,8%, „ „ 10,0%

d.h. es fanden sich Unterschiede zuungunsten der Nichtgespülten von 4,8%. Die Wahrscheinlichkeitsrechnung, die Baisch freilich außer acht läßt, lehrt nun, daß für gleiche Reihen von 500 Nichtgespülten der Prozentsatz der Genitalfiebernden zwischen rund 2,4% und 7,9% schwanken kann, bei den Gespülten zwischen 6,2% und 13,7%, bei den Insgesamtfiebernden bei den Nichtgespülten zwischen rund 3,3% und 12,7% und bei den Gespülten von 7,5% und 18,5% = d. h. beide Reihen der äußersten Möglichkeiten berühren sich. Bei 500 Fällen beträgt die Fehlergrenze $\pm$ 0,0276, wenn $\frac{h}{g} = 0{,}05 = 5\%$ ist und bei 10% $\left(\frac{h}{g} = 0{,}10\right) \pm 0{,}0379$! Baisch hat trotz des Unterschiedes von 4,8%, der geringer ist als der wahrscheinliche Fehler, daher kein Recht mit der Sicherheit eines Experimentes zu behaupten, daß ein Schaden durch die Ausspülungen entstanden ist, daß das Spiel des Zufalles in seinen Reihen sicher ausgeschlossen werden kann. Würzburgs Erfahrungen müssen doch zur größten Vorsicht auffordern! Meine eigenen Untersuchungen auf diesem Gebiete, die freilich noch nicht abgeschlossen sind, ergaben bei 3% Therapogenscheidenspülungen und bei einer Fiebergrenze von 37,9° Achselmessung, daß unter 1800 Gespülten rund 15% fieberten, darunter vom Genital aus 8%, gegenüber dem früheren Durchschnitt von 11,03% unter 4735 Geburten der Jahre 1902/1906. Es läßt sich daher eine Besserung von 3% bei den Gespülten ausrechnen. Trotzdem würde es einen sehr groben Fehler meinerseits, der ich von der Notwendigkeit der Wahrscheinlichkeitsrechnung überzeugt bin, bedeuten, wenn ich diese Besserung schon jetzt als eine sichere Folge der Therapogenausspülungen ausposaunen wollte, denn bei 1000 beträgt der mögliche Fehler bei 10% noch immer $\pm$ 0,0270, bei 2000 $\pm$ 0,019! Man bedenke nur, daß in dem ersten Viertel dieser Fälle 6,7%, im zweiten 9,8%, im dritten 8,5% und im vierten 6,5% Genitalfieber gezählt wurden, Zahlen, die untereinander natürlich gleichwertig sind. Nur durch Heranziehung der Wahrscheinlichkeitsrechnung wird die vergleichende Statistik

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1) Baisch, Der Einfluß der Scheidendesinfektion auf die Morbidität im Wochenbett. Arch. f. Gyn. 1906, Bd. 19, Heft 2.

2) Jahresbericht des Frauenspitals Basel Stadt für das Jahr 1906, S. 46.

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wissenschaftlich verwertbare Ergebnisse erzielen — diese Erkenntnis muß sich auch auf dem Gebiete der Medizin unbedingt Bahn brechen!

Unrichtig ist die stets wiederkehrende Behauptung, daß schwere Erkrankungen oder gar Todesfälle nach Eigenansteckungen nicht vorkommen können, daß Kreißende, die ausnahmsweise auf diese Art erkranken, nur leichte Fieber bekommen. Die Ergebnisse aller Kliniken, die ausschließlich mit Handschuhen untersuchen, lehren eindringlichst, daß trotzdem schwere Erkrankungen, wenn auch vielleicht nur auf dem Umwege eines Geburtstraumas vorkommen. Auch diese Kliniken haben über Verluste an Anstaltsinfektionen zu klagen so gut wie die anderen. Zeigten doch auch in Tübingen unter Döderleins Leitung jene Kreißenden, die während der Geburt innerlich nicht untersucht wurden, d. h. die leichtesten und günstigsten aller Geburten, die an sich die geringsten Aussichten auf eine Infektion darbieten, noch immer rund 5%, wenn auch zumeist leichtes Fieber (Baisch). Ganz harmlos sind die Eigenkeime der Frau durchaus nicht. Es kommt nur auf die Bedingungen an, die sie vorfinden, diese können gelegentlich auch so beschaffen sein, daß die schlummernde Angriffskraft gesteigert wird. Unter anderen hat ja Natvig das Aufsteigen eines Streptokokkus der äußeren Scham in die Tiefe der Geschlechtsteile und eine dadurch bewirkte Infektion nachgewiesen, die leicht hätte tödlich werden können.

Keinem Zweifel kann es unterliegen, daß eine zweckmäßige wirklich zuverlässige Desinfektion des ärztlichen Personales — Arzt, Hebamme — aller Gegenstände, die mit der Kreißenden in Berührung kommen, sowie der Gebärenden selbst, soweit dieses zurzeit erreichbar ist, sofort und dauernd die Zahl der schweren oder tödlich endigenden Kindbettfieber auf eine Mindestzahl sinken lassen wird. Aussicht ist aber nur dann vorhanden, wenn das Geburtstrauma möglichst klein ausfällt, wenn es nicht durch ein Operationstrauma vergrößert wird. Eine Bedingung, die in ihrer Wichtigkeit gleich hinter der Forderung einer zuverlässigen Desinfektion kommt.

Betrachten wir einmal, meine Herren, diese wichtige Bedingung näher!

Eine ganz besonders hervorragende Rolle in der Vorbeugung des Kindbettfiebers spielt nicht allein die Einschränkung der inneren Untersuchungen, sondern weit mehr die Vermeidung aller nicht unbedingt notwendigen Operationen, d. h. die Herabsetzung des Geburtstrauma auf das physiologisch unvermeidbare Minimum.

Eine bekannte, von Friedrich bewiesene Tatsache ist, daß ein großer Unterschied darin liegt, ob Krankheitserreger nur locker einer Wundoberfläche, wie nach einer gewöhnlichen Geburt aufliegen, oder ob sie mit Kraft in eröffnete Gewebsspalten eingeimpft werden, wie es bei Operationen leicht geschieht. Das Trauma ist nicht die einzige, aber eine der wichtigsten Bedingungen für das Entstehen eines Kindbettfiebers!

Eine uns Ärzte tief beschämende Tatsache ist es leider, daß in

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allen Ländern in der Vorgeschichte und damit auch als Ursache schwerer Kindbettfieberformen Operationen häufig erwähnt werden. Von vielen Ärzten werden unnötige Eingriffe — Luxus-, Erlösungs- vorbeugende Zangen — ausgeführt. Diese bevorzugen merkwürdigerweise auch die für eine Infektion gefährlichste Operation, die manuelle Plazentarlösung. Alle diese Eingriffe nehmen mit der Häufigkeit der Ärzte zu. Eine merkwürdige Erscheinung, die durch Furcht vor unlauteren Wettbewerb, die durch eine gewisse Abhängigkeit einflußreicher Hebammen erklärt, aber nicht entschuldigt wird. Die Narkose, ein ungerechtfertigtes Vertrauen in eine oft obendrein völlig ungenügende, wenn auch sonst gewiß sehr oft sehr gewissenhaft durchgeführte Desinfektion — Heißwasser, Seife plus Desinfiziens in Wasser — verleitet gar zu leicht zu häufigeren Eingriffen als dieses bei unseren Voreltern, die das Kindbettfieber zu fürchten alle Ursache hatten, üblich gewesen zu sein scheint!

Freilich, eine rasche, dabei schonende Untersuchung mit kurz geschnittenen Nägeln, die nicht verletzt, erhöht die Gefahr nicht. Wenn es sich gezeigt hat, daß bei behandschuhten Fingern die Zahl der Untersuchungen ohne wesentlichen Einfluß auf das Vorkommen von Fieber ist (Baisch), so liegt die Ursache nicht darin, daß die Keime der Scheide oder der Scham harmlos sind, sondern darin, daß Schüler, Studenten und Hebammen, mit Handschuhen keine oder doch nur selten Verletzungen machen können. Die Handschuhe vermindern auf diese Weise die Möglichkeit der Einpflanzung von Eigenkeimen in die Gewebe — das ist zuzugeben. Weiß doch ein jeder Lehrer, wie viel Mühe es kostet, bis die Hände der Anfänger in den Tuschierkursen weich geworden sind — ein Ziel, das zu den vornehmsten Aufgaben eines Lehrers auf diesem Gebiete gehört.

Gestatten Handschuhe eine ausgebreitetere Anwendung der Operation, ermöglichen sie die Anbahnung einer ungefährlichen echten chirurgischen Ära der Geburtshilfe? Das sind, meine Herren, wichtige Fragen, die das Wohl der werdenden Mutter tief berühren. Nein, und abermals nein, muß die Antwort lauten. Eines nämlich kann keine noch so sorgfältig gereinigte Hand, kein Gummihandschuh, kein Chirosoter, nichts, nämlich die Vermeidung einer Verschleppung von Keimen aus den äußeren Geschlechtsteilen, aus der Scheide, selbst aus der Cervix in die Gebärmutterhöhle, gegebenenfalls an und in die Plazentarstelle, solange es nicht gelingt, eine weitgehende Keimverarmung der Geburtswege herzustellen! Freilich sind das Keime, die bei spontanen Geburten sehr selten schwerste Erkrankungen auslösen, weil sie bei diesen in die Gebärmutterhöhle nicht verschleppt werden, weil sie in selbständiger Einwanderung

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an die große Wunde des Endometriums erst dann gelangen, wenn diese Breschen in den gesunden Geweben schon längst geschützt sind. Die Spaltpilze finden keine günstigen Bedingungen, keine besondere Gelegenheit zur Entfaltung und Wirkung ihrer Angriffskraft. Werden aber die gleichen Keime in einem Einzelfalle in eben entstandene, oft genug noch gequetschte oder sonstwie geschädigte Wunden, etwa bei Gelegenheit eines operativen Eingriffes — trotz sonst völlig keimfreier Hand — Handschuh! — unmittelbar eingeimpft, so werden diese Spaltpilze, die sonst harmlos erscheinen, ihre unheilvolle Tätigkeit unbehindert durch Schutzvorrichtungen des Körpers, die noch nicht zur Stelle sein können, entfalten. Das kann nicht oft genug wiederholt und gepredigt werden.

So erklärt es sich ungezwungen, wie ein Trauma, insbesonders die Setzung gequetschter, zerrissener Wunden unter der Geburt, noch weit gefährlicher allerdings im Wochenbett und bei Eingriffen an der Plazentarstelle, die allergünstigsten Bedingungen für den Ausbruch einer schweren Erkrankung abgeben kann.

Die Schwere des Geburtstraumas spielt eine wesentliche Rolle in der Entstehung des Kindbettfiebers; das Trauma verschuldet, daß nicht nur Fremdkeime, sondern auch harmlose Eigenkeime der Wöchnerin gefährlich werden.[1]) Je geringer das Geburtstrauma ausfällt, desto ungünstigere Bedingungen für eine Infektion liegen vor, je ausgedehnter ein Operationstrauma ist, je näher dieses an der Plazentarstelle liegt, desto größer die Gefahr für die Wöchnerin — das sind Sätze, die ein jeder Geburtshelfer sich fest einprägen muß, nach denen er sein Handeln einzurichten hat. Zweifellos läßt sich der ungünstige Einfluß des Traumas durch eine tadellose Asepsis und Antisepsis paralysieren, aber wehe, wenn sie irgendwie ungenügend sind, wie leider so oft in der Hauspraxis.

Das Übersehen, das Geringschätzen des Traumas, meine

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1) Zahlreich sind die Beispiele, die beweisen, daß Eigenkeime durch ein Trauma erst ihre wahre Natur zeigen. Hier möge ein solches Platz finden. Während der ersten Niederschrift dieser Zeilen mußte ich wegen schweren Blutungen, nach bis dahin fieberfreiem Wochenbett, einige Plazentarreste entfernen. Dies geschah von mir selbst in schonendster Weise mit dem Finger, die Hand war mit einem sicher keimfreien Handschuh geschützt, der auch nach dem leichten Eingriffe sich als unverletzt erwiesen hat. Vor und nach dem kurzen Eingriffe wurde der Uterus reichlich mit Jodalkohol durchgespült. Trotzdem rasche Entwicklung einer Staphylokokkämie, Metrolymphangitis, Metroplebitis purulenta, Amputatio uteri supravaginalis, erhebliche Besserung, schließlich Tod an Verschleppungsbakteriämie — alles bedingt durch Infektion mit Eigenkeimen begünstigt durch ein Trauma.

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Herren, ist neben einer ungenügenden Desinfektion eine der Hauptursachen, daß das Kindbettfieber nicht abnehmen will. Hier müssen Sie in gemeinsamer Tätigkeit den Hebel kräftig ansetzen, um dieses Hemmnis des Fortschrittes im Kampfe gegen das Kindbettfieber wegzuräumen! Besondere Schwierigkeiten gibt es dabei nicht zu überwinden. Es heißt nur, keine Eingriffe, keine Operation ohne strengste Anzeigen ausführen, als welche ohne alle Zweifel nur gelten können: vorhandene oder sicher eintretende Gefahr für Mutter und Kind. In allen anderen Fällen wird die Geburt ohne Schaden für Mutter und Kind verlaufen, wenn der Arzt nicht überflüssigerweise sich hineinmischt.

Denken Sie, meine Herren, stets an die möglichen Folgen eines Traumas, an die Begünstigung einer Infektion durch Eigenkeime, an eine Verschlimmerung einer solchen, die durch Fremdkeime im Begriffe steht zu entstehen und Sie werden an der Hand einer zuverlässigen Händereinigung die glänzenden Erfolge der Gebäranstalten übertrumpfen. Lassen Sie sich nicht durch das leicht mißzuverstehende Schlagwort „operative Ära der Geburtshilfe" in der Ausübung der beiden größten Tugenden des Geburtshelfers, die der Geduld und des weitgehendsten Vertrauens in die Naturkräfte erschüttern. Leisten Sie den größten Widerstand der so weit verbreiteten Neigung zu überflüssigen Operationen und Sie werden sich innere Befriedigung holen, Sie werden sich überzeugen, nur genützt und nicht geschadet zu haben, Sie werden die innere Kraft finden, standzuhalten gegenüber absprechendem Urteile dummer Hebammen oder unsauberer Kollegen, die in unlauterem Wettbewerb ihr Heil suchen! Kein Zweifel, daß Sie auf die Dauer auf der ganzen Linie den Sieg erringen werden! Freilich werden noch einige Jahrzehnte vorübergehen, bis eine mißverstandene Ära der operativen Geburtshilfe in ihrer ganzen Gefährlichkeit erkannt worden ist.

Mit Recht wird Boër als leuchtendes Vorbild genannt. Aber, meine Herren, es genügt nicht, von diesem großen Geburtshelfer zu reden, man muß mit der Tat beweisen, daß seine Lehren auch beherzigt werden!

Das physiologische Geburtstrauma ist beim engen Becken am größten. Wird dieses noch durch ein Operationstrauma verschlimmert, so kann an der Hand einer ungenügenden Antisepsis das Schicksal der Wöchnerin leicht ein trauriges werden. Daher auch die bekannte Tatsache der so viel schlechteren Voraussage des engen Beckens in der Hauspraxis. Die Frage der Verminderung des schwereren Geburtstraumas des engen Beckens, sei es durch Vermeidung von Operationen, sei es durch Näherung des Traumas auf das Maß eines nor-

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malen Beckens, meine Herren, sind in der Hauspraxis wichtigste Fragen der Vorbeugung des Kindbettfiebers, die durchaus eingehend erörtert werden müssen.

Von jeher habe ich gelehrt — und diese Lehre habe ich von meinen Lehrern Kaltenbach und Fehling übernommen —, daß bei etwa $^{7}/_{10}$ aller engen Becken keine Kunsthilfe nötig ist. Sie können dieses in meiner Geburtshilflichen Operationslehre, 1893, Seite 145, nachlesen. Eine alte Lehre — daher ist es grundfalsch, wenn immer wieder und wieder betont wird, daß erst die moderne Geburtshilfe das Ziel erreicht hat, der Natur mehr wie bisher zu ihrem Recht verholfen zu haben. Konservative Behandlung der Geburten des engen Beckens, heißt es heute auf der ganzen Linie, aber auch früher lautete die gleiche Losung. Der Konservatismus in der Behandlung des engen Beckens ist durchaus nicht modern, er ist vielmehr recht alt. Verschieden sind nur die Mittel, die angewandt werden, wenn der Konservatismus scheitert oder voraussichtlich nicht zum Ziele führen wird, wenn es sich darum handelt, der werdenden Mutter die Schrecken einer Geburt bei engen Becken zu mildern. Letztere Aufgabe wird allerdings trotz ihrer Humanität heutzutage als unmodern verschrieen und totgeschwiegen. Und doch besteht ein sehr wesentlicher Unterschied, der Sie, meine Herrn der Hauspraxis, ganz besonders interessieren muß. Hier Konservatismus in der Behandlung des engen Beckens mit Hilfe vorbeugender Eingriffe, die für die Mutter kaum gefährlich sind, allerdings etwas mehr Kinder fordern, dort Konservatismus mit Hilfe der großen geburtshülflichen Operationen des Kaiserschnittes und der Beckenspaltungen, die unter allen Umständen für die Mütter gefährlich sind, wenn sie auch einer Anzahl von Kindern mehr das Leben retten, aber sie fallen für die Hauspraxis aus. Die moderne Behandlung brüstet sich damit, daß dabei die größte Zahl von Spontangeburten beobachtet wird, ohne weitergehende Gefährdung der Mütter und der Kinder, ihr stehe die alte Lehre in jeder Beziehung nach. Auch dieser Ausspruch entspricht nicht den Tatsachen, ist daher falsch! Hier der Beweis:

Der Konservatismus, der im Frauenspital Basel-Stadt herrscht, dürfte überall bekannt sein und wird von keiner anderen Anstalt der Jetztzeit übertroffen. Beweis hierfür: unter rund 8000 Geburten meiner Leitung sind in rund 0,5 % der Fälle Erzwungene Entbindungen, so übersetze ich das Accouchement forcé, vorgenommen worden. Darunter befinden sich Erweiterungen mit dem vorzüglichen Instrumente von Bossi: 5, vaginale Hysterotomien: 3, Erweiterung nach Harris und Bonnaire: 3, Anwendung von Tarniers Erweiterer: 12, der Rest betrifft Hystereurysen, diese fast alle bedingt durch Placenta

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praevia. Der Konservatismus wird weiterhin damit bewiesen, daß bei über 4000 Geburten mit beinahe 400 engen Becken nur siebenmal die Hebosteotomie ausgeführt worden ist und dabei ist kein einziges lebendes Kind perforiert worden. Und wie verhält es sich damit anderswo? Ich las vor kurzem, daß in einer großen Anstalt auf noch nicht 1000 Geburten sechsmal dieser Eingriff in einem Jahre vorgenommen wurde! Und wie oft hört man bei viel weniger Geburten, selbst aus der Tätigkeit von Hausärzten heraus, über 30—40 vaginalen Kaiserschnitten reden? Konservativ ist daher auch die Behandlung des engen Beckens, allerdings mit Hilfe vorbeugender Eingriffe, insbesondere der künstlichen Frühgeburt. Dieser Eingriff ist nicht nur im Interesse der Kinder, sondern ganz besonders auch zu Nutz und Frommen der Mütter vorzunehmen, um ihnen ein größeres Geburtstrauma zu ersparen — so schrieb ich in meiner vorher erwähnten Geburtshilflichen Operationslehre, Seite 93. *Das Mildern eines gesteigerten Geburtstraumas auf das physiologische Maß ist der Hauptzweck der künstlichen Frühgeburt,* nicht etwa die Vermeidung der Tötung eines lebenden Kindes. Wer das nicht einsehen will, der schließt absichtlich seine Augen vor einer unbestreitbaren Tatsache, er will nicht das Wesen dieser so segensreichen Operation erkennen. Dieser Zweck ist von der größten Bedeutung. Er ist leicht zu erreichen, wenn zur Einleitung der Geburt der Blasenstich, der an sich gar kein Trauma wie etwa die Hystereuryse mit nachfolgender Wendung setzt, vorgenommen wird. Und wie gestalten sich die Ergebnisse?

Unter 12420 Geburten (1896—1906) fanden sich in Basel 1150 enge Becken = 9,3 %. Von diesen 1150 Geburten endigten 917 ohne Kunsthilfe = 79,8 %. Unter den Operationen sind erwähnenswert 14 Kaiserschnitte mit zwei Verlusten, drei Schamfugenschnitte mit einem Tode und vier Schambeinschnitte, d. h. 21 große geburtshilfliche Eingriffe neben allerdings einer großen Anzahl von künstlichen Frühgeburten, 120, neben einigen wenigen vorbeugenden Wendungen, da ich im allgemeinen ein Gegner dieses letzteren Eingriffes bin. In Basel betrug die Summe der großen Eingriffe 1,9 % gegenüber 6,1 % bei Döderlein in Tübingen. Geboren wurden 1159 Kinder, von denen 116 tot geboren oder doch innerhalb der nächsten Stunden oder Tage starben = 10 % Sterblichkeit. Darunter befanden sich aber 19 vor der Geburt abgestorbene luetische Früchte. Werden diese, wie billig abgezogen, so beträgt die Sterblichkeit der Kinder 8,5 %.

Von den 1150 Frauen sind 12 gestorben — alle bis auf eine bei operativem Eingriff. Die mütterliche Sterblichkeit beträgt somit rund 10,5 % ohne jedwelchen Abzug. Die Todesursachen waren fünfmal

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Uterusruptur, dreimal Verblutung an Atonia uteri (einmal zugleich Cervixruptur), viermal an Kindbettfieber, darunter einmal nach Schambeinschnitt, zweimal nach Kaiserschnitt, einmal nach Zange, worunter zwei dieser Frauen bereits infiziert eingeliefert worden waren. Der Behandlung des Beckens erlagen somit zwei Frauen = 0,17 % und diese Opfer entsprechen dem modernen Konservatismus in der Behandlung des engen Beckens und hätten vielleicht durch Opferung der Kinder vermieden werden können.[1]) Werden alle Temperatursteigerungen, die 37,9° in der Achsel übersteigen, in Rechnung gesetzt, so fieberten 16 %.

Behandlung	Spontangeburten	Operative Geburten	Hohe Zangen	Vorbeugende Wendungen	Künstliche Frühgeburten	Summe der vorbeugenden Eingriffe	Perforation des lebenden Kindes	Beckenspaltungen	Kaiserschnitte	Summe der großen Eingriffe	Kindersterblichkeit ohne jeden Abzug	Kindersterblichkeit ohne Lues	Sterblichkeit der Mütter ohne jeden Abzug	Sterblichkeit d. Mütter infolge
Leipzig (Zweifel)	78,4	21,6	2,1	2,5	0,5	5,1	2,4	4,8	3,7	8,5	9,9	?	—	0
Tübingen (Döderlein)	80,0	20,6	1,0	0,6	0,5	2,2	0,6	2,1	4,0	6,1	6,5	?	—	0
Basel (Bumm, v. Herff) 1896-1906	79,8	20,2	2,9	2,2	10,4	15,5	0,08	0,6	1,2	1.9	10,2	8,5	10,5	0 (0
Übersicht d. Operationen in Basel[1]) . . .	917	232	72[2]) (6,0 %)	63[3]) (5,3 %)	120[4])	180	25[5]) (2,1 %)	7	14	21	119	100[6])	12[7])	

1) In diese Rubrik sind alle operativen Eingriffe jeder Art, mit Ausnahme der wenigen Extraktionen aus Beckenendlagen, 29, verzeichnet. 2) Darunter 34 hohe Zangen. 3) Darunter 26 vorbeugende Wendungen, die in den letzten 6 Jahren nicht mehr ausgeführt wurden. 4) Durch die künstliche Frühgeburt wurden 89,4 % Kinder lebend geboren und 79,7 % lebend entlassen. 5) Eine Perforation des lebenden Kindes (1900). 6) 19 Kinder waren vor der Geburt infolge Lues gestorben. 7) Die Todesfälle, ohne jeden Abzug, bestanden aus 5 Frauen, die mit Uterusrupturen

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1) Während der Niederschrift dieser Zeilen bildete sich bei einer Kreißenden mit leicht verengtem Becken, Conj. vera = 9 cm, aber mit einem sehr großen Kinde mit sehr hartem Kopf rasch eine Physometra aus. Das Kind lebte, so versuchte ich die Entwicklung mit meiner Achsenzugzange — vergeblich! Da der fötale Herzschlag noch gut war, folgte Hebosteotomie nach Bumm. Blasenverletzung, Episeotomie. Kind frisch lebend. Dieses wurde durch das Fruchtwasser infiziert und erlag in der dritten Woche einer Lungenentzündung. Die Mutter mußte ein schweres Krankenlager durchmachen, das jetzt nach drei Monaten noch nicht zu Ende ist und wahrscheinlich mit einer dauernden Schädigung der Blasentätigkeit endigen wird. Wäre es für die Mutter nicht zweckmäßiger gewesen, die Frucht angebohrt zu haben, das Endergebnis wäre fürwahr ein besseres gewesen!

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zur Vollendung der Geburt der Anstalt überwiesen worden sind (3 mal Kranioklasie, 1 mal Zange, 1 mal Extraktion aus Fußlage). 2 Frauen wurden infiziert eingeliefert (1 mal Zange, 1 mal Kaiserschnitt). 3 Frauen verbluteten sich an Atonia uteri (1 mal spontane Geburt, 1 mal Zange, 1 mal künstliche Frühgeburt mit Wendung und Perforation). 2 Frauen wurden in der Anstalt infiziert (1 mal Kaiserschnitt [1896], 1 mal Schamfugenschnitt [1897]). Will man die drei Todesfälle an Verblutung der Art der Behandlung des engen Beckens zuschieben, so wären dieser 5 Mütter = 0,4 % erlegen.

Meine Herren, das sind genau die gleichen Ergebnisse, wie sie die modernste Behandlung erzielt. So steht Tübingen, wenn man die Wahrscheinlichkeitsrechnung berücksichtigt, mit seinen 80 % Spontangeburten, 6,6 % Kindersterblichkeit und 0,1 % mütterlichen Verlusten Basel gegenüber völlig gleich und dabei hat Basel ein Drittel weniger große Eingriffe zum großen Vorteil der Mütter zu verzeichnen![1]) Es kann ja nicht ausbleiben, daß auf die Dauer Kaiserschnitte und Beckenspaltungen die Todesfälle der Mütter erheblich vermehren müssen. Ferner ist zu bedenken, daß nach künstlichen Frühgeburten das Wochenbett wie ein jedes andere verläuft und keinerlei Dauerschädigungen folgen, daß nach Kaiserschnitten und Beckenspaltungen ein mehr oder weniger, zum mindesten unbequemes, häufiger schmerzhaftes, selbst langes Krankenlager folgen kann, daß nach den großen Eingriffen dauernde Schädigungen der Gesundheit in Gestalt von Hernien, Senkungen, Vorfällen, Störungen der Harnentleerung und Behinderung der Gehfähigkeit durchaus nicht selten sind. Wo in aller Welt, meine Herren, kann man ein solches langes und trauriges Sündenregister der künstlichen Frühgeburt, deren Hauptzweck die Herabsetzung des pathologisch gesteigerten Geburtstraumas des engen Beckens auf das physiologische Maß ist, aufstellen — es sei denn, daß man der Wahrheit ins Gesicht schlagen will? Aber totgeschwiegen wird diese Tatsache in einem fort!

Der Satz ist somit bewiesen: *Die abwartende Geburtsleitung gestützt auf vorbeugende Eingriffe ist in ihren Endergebnissen nicht nur völlig gleich, sondern muß auf die Dauer besser sein als die abwartende Geburtsleitung gestützt auf Kaiserschnitt und Beckenspaltungen.* Wer der Richtschnur folgt: vor allem die Mutter und dann das Kind, wer die Mutter sicher und in jedem Falle vor dauerndem Schaden

1) Bei einer Wahrscheinlichkeit von 0,9953 berechnet sich der wahrscheinliche Fehler in Leipzig wie in Tübingen auf etwa ± 4% bei 800 Fällen, in Basel auf ± 3,5% bei 1000 Fällen und 20% operative Geburten. Daraus folgt, daß die Ergebnisse dieser zwei Anstalten mit größter Wahrscheinlichkeit gleichwertig sind, selbst für den Fall, daß die Extraktionen aus Beckenendlagen als „operative Eingriffe" einbezogen werden und die Zahl der operativen Geburten in Basel mit 22,7% in Rechnung gesetzt werden.

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bewahren will, wer in der allgemeinen Hauspraxis steht, der weiß nunmehr, welchem Konservatismus, ob dem älteren oder dem modernen Pinards er zu folgen hat. Lassen Sie sich darin, meine Herrn, nicht beirren, forschen sie selber nach, denn gar zu leicht werden heutigen Tages unbequeme Tatsachen übergangen.

Eine Rückkehr zur alten Lehre, zunächst den Verlauf der Geburt genau zu verfolgen, um Störungen zu verhüten, dann einzugreifen, wenn wirklich Gefahr droht, vorzubeugen, wenn solche zu erwarten ist, muß unbedingt verlangt werden, dafür müssen wir Lehrer der Geburtshilfe mit allem unsern Einfluß einstehen! Dann, aber nur dann wird die Zahl der Kindbettfieber in Stadt und Land rasch abnehmen, das bessere Zeitalter, nach dem sich Semmelweis vergeblich gesehnt hat, zur Wirklichkeit werden. Fort also mit einer mißverstandenen operativen Ära der Geburtshilfe! Helfen sie diese zu bekämpfen, meine Herren, und sie werden sich den Dank der Menschheit, wenn auch erst in der Zukunft erwerben!

Daß das Trauma, d. h. die Setzung von zerrissenen, gequetschten Wunden, Gewebsquetschungen, die bis zur Zermalmung der Gewebe gehen können, durch Operation eine verhängnisvolle Rolle in den Ursachen des Kindbettfiebers spielt, ist eine Tatsache, die sich nicht wegleugnen läßt. Hierfür habe ich in meiner Abhandlung über Kindbettfieber manche Beweise beigebracht. Neuerdings hat Dohrn[1]) nachgewiesen, daß unter den Entstehungsursachen der Kindbettfieberfälle am häufigsten (ca. 25%) ärztliche Eingriffe — manuelle Plazentarlösungen, Wendungen und Zangen, besonders die „Luxuszange“ — aufgeführt werden müssen. In 82% lag die Schuld nicht an der Hebamme allein, in 18% an der Hebamme — das sind doch Zahlen die sehr zu denken geben. Mögen doch die Regierungen derartige höchst wertvolle Untersuchungen anordnen und nachhaltig unterstützen.

Auch Hegar[2]) kommt in seiner jüngsten Abhandlung: „Die operative Ära der Geburtshilfe“ auf diese Frage in dankenswertester Weise zurück. Ich entnehme daraus folgende kleine Tabelle, die die Verhältnisse in Baden beleuchtet.

Jahrgänge	Zahl der Geburten	Todesfälle	Mortalität in % der Geburten	Zahl der Operationen	Zahl der Operationen zur Zahl der Geburten	Todesfälle nach Operationen	Mortalität in % der Operationen	Mortalität nach Operationen in % der Geburten	Mortalität nach Operationen in % aller Todesf.
1870—72	171832	—	—	6909	1 : 24,8	364	5,2	0,212	—
1873—77	302070	2444	0,809	13178	1 : 22,9	763	5,8	0,253	31,3
1878—82	285721	2046	0,716	13204	1 : 21,6	682	5,2	0,258	33,3
1883—87	271283	2035	0,750	17533	1 : 15,4	555	3,2	0,204	27,2

1) Sonderabdruck aus dem offiziellen Bericht der 22. Hauptversammlung des preußischen Medizinalbeamtenvereines.

2) Beiträge zur Geburtshilfe und Gynäkologie 1907, 12. Bd., 2. Heft, auch Volkmanns klin. Vorträge Nr. 351.

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Hegar führt aus, daß die Sterblichkeit nach Operationen $^1/_4$—$^1/_5$ der Gesamtsterblichkeit beträgt. Die Sterblichkeit nach Operationen, in Prozenten der Operationen ausgedrückt, ist in der Zeit mit gutausgebildeter Asepsia entschieden geringer als in den Perioden ohne alle oder mit unvollkommener Asepsia.

Die Sterblichkeit nach Operationen, in Prozenten sämtlicher Geburten ausgedrückt, ist dagegen in der Zeit mit vollkommener und ohne alle Asepsia fast vollständig gleich. Das berechtigt zum Schluß, daß die größere Frequenz der Operationen in der aseptischen Zeit 1:15,4, im Gegensatz zu der geringeren Frequenz 1:25 in der Zeit ohne alle Asepsia, das wieder verdarb, was die Asepsia und wohl auch bessere Technik Gutes erzielte. Fürwahr, Altmeister Hegar hat leider auch für die neueste Zeit nur allzusehr recht!

Welch Unheil eine mißverstandene chirurgische Ära der Geburtshilfe anzurichten vermag, zeigt eine Empfehlung, die ich soeben lese, nämlich die normale Geburtsdauer abzukürzen, und die sich in der Nummer 1 der Berliner klinischen Wochenschrift dieses Jahres vorfindet. Unglaublich, aber doch wahr, hier wird die „prophylaktische Zange" bei weitem übertroffen! Theodor Landau schlägt vor, die Eröffnungszeit, wohlgemerkt bei normalen Geburten, durch digitale Erweiterung des Muttermundes entsprechend dem Verfahren von Bonnaire und Harris abzukürzen. Die Entschuldigung Landaus, daß ja bei Gebrauch von Handschuh und Desinfektion der Kreißenden (!) keine Infektion zu erwarten steht, ist so recht bezeichnend für unsere Zeit, der man das Motto geben könnte: Nur nicht auf den Grund gehen, hübsch auf der Oberfläche bleiben! Mag sein — ich kann dies nicht beurteilen —, daß in einer mehrfachen Millionenstadt für einen vielbeschäftigten Spezialisten ein dringendes Bedürfnis vorliegt eine normale Eröffnungsperiode abzukürzen — aber mit solchen Vorschlägen verschone man die zahlreichen Geburtshelfer, die über die vornehmste Tugend einer solchen „Geduld" noch verfügen und die durchaus nicht der Ansicht sind, es müsse nunmehr alles mit Elektrizitätsgeschwindigkeit gehen. Nein, eine solche üble Vielgeschäftigkeit verdient als wissenschaftlicher Unfug auf das schärfste öffentlich getadelt zu werden. Solches zu tun liegt im dringenden Interesse der werdenden Mütter, in meinem eigenen Interesse als Lehrer angehender Ärzte um zu verhindern, daß solche Ratschläge einer mißverstandenen operativen Ära der Geburtshilfe befolgt werden.

Der selbstverständlichen Forderung, nur auf strengste Anzeige hin zu operieren, wird mir von Kalt, einem älteren erfahrenen Geburtshelfer, vorgehalten[1]), daß solches in der Hauspraxis nicht möglich sei. Es heißt dort: „Solange es viele Frauen gibt, denen die nötige Geduld zur Abwartung des natürlichen Geburtsverlaufes fehlt, sondern die mit beharrlichem Ungestüm die künstliche Befreiung von ihren Leiden verlangen, worin sie vom ängstlich besorgten Ehemann und andern Anverwandten mit kategorischen Imperativen unterstützt werden und das Können und Erwerben des Arztes noch in Frage gestellt wird, solange dieser Arzt noch vielen Berufspflichten anderer Klientelschaft im Momente nachkommen soll, welche Pflichten ihm nicht stundenlange Versäumnisse bei Geburten gestatten, so lange ist eben oft die Macht der Verhältnisse stärker bestimmend, als die exakte wissenschaftliche Indikationsstellung und — der Arzt unterliegt eben der stärkeren menschlichen Beeinflussung![2])

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1) Die ausschließliche Benutzung von Entbindungsanstalten zur Abwicklung des Geburtsvorganges und der Wochenbettspflege. Dissert. Zürich 1907.

2) Gewiß ist es sehr erfreulich, wenn ein erfahrener praktischer Geburtshelfer sich entschließt, öffentlich zu diesen Fragen Stellung zu nehmen. Auch ich habe in einer fünfjährigen Tätigkeit als Hausarzt diese Umstände gründlichst kennen

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Die Verhältnisse sind so, wie sie Kalt schildert. Ich gestehe aber offen, daß ich mich gescheut haben würde, so ohne weiteres zu sagen, daß die Geburtshelfer sich von der Kreißenden und ihrer Umgebung, also von Laien, die Operationsanzeige gegen besseres Wissen stellen lassen.[1]) Die tieferen Gründe sind meines Erachtens andere und die soll man auch mitteilen. Diese sind in der Furcht vor unlauterem Wettbewerb inkollegialer Ärzte zu suchen — tust du meinen Willen nicht, so wird es ein anderer gerne tun, heißt es gelegentlich — und leider finden sich solche Ärzte, die der Kreißenden und ihrer Umgebung zuliebe ohne weiteres deren Wünsche erfüllen, um womöglich den standhaften und gewissenhaften Arzt bloßzustellen und sich als Retter aus der angeblichen Not preisen zu lassen. Ferner die Abhängigkeit so manchen Arztes von einer einflußreichen, allmächtigen Hebamme, die sich ärgert, wenn ein Arzt sich erkühnt, ihre Anzeigen nicht zu befolgen usw. Gewiß sind es sehr traurige Verhältnisse, auch lassen sie es begreifen und selbst bis zu einem gewissen Grade entschuldigen, wenn Ärzte ihrer Macht unterliegen. Wo solches stattfinden kann, sind aber die Ärzte schuld, sie haben die Pflicht in gemeinsamer Tätigkeit sich von diesen Verhältnissen zu befreien. Solches läßt sich, meiner Erfahrung nach, in weitgehender Weise erreichen — man muß nur den richtigen Willen haben, unlauteren Wettbewerb zu ersticken und gegen eingebildete und übermütige Hebammen gemeinsam Front zu machen.

Der weitere Einwand Kalts, daß die Asepsis und Antisepsis in der Hauspraxis nicht durchführbar sei, ist nicht stichhaltig. Jede gut geleitete geburtshilfliche Poliklinik, die doch nichts weiter ist als Hausgeburtshilfe bei den Ärmsten, beweist dieses ohne weiteres. So verfügt die geburtshilfliche Poliklinik meiner Anstalt, die von Herrn Dr. Labhardt geführt wird, über nahe an 1000 Hilfeleistungen unter der Ahlfeldschen Heißwasser-Alkoholdesinfektion und dank strenger Operationsanzeige ohne einen Verlust an Kindbettfieber. Und wie viele ältere erfahrene Geburtshelfer gibt es — allerdings keine Anhänger einer mißverstandenen chirurgischen Ära der Geburtshilfe, sondern Schüler Boërs — die in vieljähriger Tätigkeit keinerlei Verlust an Kindbettfieber erlebt haben! Der Einwurf Kalts ist nicht nur an sich nicht richtig, sondern er ist sehr bedenklich, weil er gar zu leicht ein schwaches Gewissen ganz einzuschläfern vermag, und dem laisser faire das Wort nur zu sehr redet. Man muß nur recht wollen, so geht auch die Desinfektion im Hause, in einer Hütte!

Besonders günstige Bedingungen für die Einpflanzung von Genitalkeimen bietet das Einlegen von Fremdkörpern aller Art in die Gebärmutter, vor allen die so beliebten Ausstopfungen mit Gaze, selbst wenn sie mit Antiseptika — Xeroform, Vioform, Jodoform usw. — durchtränkt sind, aber auch das Einführen von Gummiballons (Hystereuryse). Aus diesem Grunde empfehle ich daher die Einleitung der künst-

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gelernt, mich aber nicht gebeugt. Wie dies geschehen kann, das lehrt ein jeder von uns seinen Schülern im Operationskurs!

1) Selbst „Spezialisten“ — traurig genug — lassen sich ihre Anzeigen durch die Umgebung der Kreißenden stellen! So lese ich: Das andere Mal sah ich mich in einem Dorf einer älteren Erstgebärenden, ihren Angehörigen und dem Kollegen gegenüber (sic!) genötigt, 3 Tage nach dem Blasensprung und am 3. Tage der Wehen die Geburt zu beenden. Manuelle Dilatation des rigiden Muttermundes, äußerst langwierige Zangenextraktion des im Beckeneingange kaum feststehenden Kopfes usw. Fürwahr, ein treffendes Beispiel für die Irrwege der chirurgischen Ära der Geburtshilfe!

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Jahrgang	Gesamtzahl der Berufungen	Geleitete Wochenbetten nach Aborten	Geleitete Wochenbetten nach Geburten	Zahl der Operationen	Gesamtmorbidität	Durchsch.	Genitalmorbidität	Durchsch.	Schwere Erkrankungen und Todesfälle
1903	106	28	69	88	6,6 %		6,0 %		J. Nr. 97 Müller. Bakteriämie; diesseits nicht untersucht; nur Credé. Spital. J. Nr. 70 Jäggi. Querlage, Plac. praevia. Mit Bakteriämie angetreten. Ins Spital. Vaginale Totalexstirpation.
1904	195	53	121	127	11,3 %		7,0 %		† J. Nr. 89. Placenta praevia — Embolie 3 Stunden post part.
1905	202	47	130	134	22,2 %	14,4 %	18,0 %	11,3 %	† J. Nr. 136. Am 29. Tage post partum an Embolie.
1906	233	65	138	141	17,7 %		15,4 %		J. Nr. 103 Mild. Placenta praevia. Wendung. Verschleppungsbakteriämie. Unerlaubte Extraktion durch den Praktikanten, ohne Desinfekt. Spital. † J. Nr. 186. Luftembolie. — Placenta praevia. Wendung. † J. Nr. 99. Moribund eingetreten. (Bakteriämie.)
1907	224	67	144	153	14,2 %		10,4 %		
Total	960	260	602	643					

Von 1. Januar 1905 an, Desinfektion nach Ahlfeld.
Von 1. April 1904 an, genauere Temperaturkontrolle.
Bei der Gesamt- und Genitalmorbidität sind stets die Fälle mit eingerechnet, die febril eintraten, und die besonders bei Aborten einen hohen Prozentsatz ausmachen.

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lichen Frühgeburt mit dem Blasenstich, welche Methode zweifellos in der Hauspraxis erheblich bessere Ergebnisse ergeben wird, zumal die vielfach, so jüngsthin noch von Hannes, behaupteten schweren Nachteile des frühzeitigen Blasensprunges nur in gewissen Lehrbüchern, nicht aber am Kreißbette zu finden sind. Mögen diese Gegenstände noch so sicher keimfrei gemacht werden, sie können jederzeit Keime mitverschleppen, wie denn alle sogenannte antiseptische Gaze in kürzester Zeit von zahlreichen Spaltpilzen durchwachsen wird.

Mit der Einführung einer zuverlässigen Desinfektion und der Verminderung des Geburtstrauma auf ein Minimum sind die Vorbeugungsmaßregeln noch nicht alle erschöpft. Auch diese mögen heute kurz angeführt werden, wiewohl sie an Wichtigkeit zurückstehen. Die gefährlichsten Eingriffe sind jene, die sich an der Plazentarstelle, wie überhaupt außerhalb des Chorionssackes abspielen — ein Satz, der Ihre größte Beachtung fordert. Wie gefährlich die Lösung der Nachgeburt ist, von welch großer Wichtigkeit eine sachgemäße Leitung der Nachgeburtsperiode, die diesem Eingriff vorbeugt und auf wenige Fälle beschränkt, sein muß, geht, meine Herren, ohne weiteres aus folgenden Zahlen hervor. Nach Seeligmann[1]) wurde in Hamburg in den Jahren 1896—1905 1123 mal die Lösung der Nachgeburt gemacht, davon starben 232 Frauen, d. h. 20,8 %! Kein Zweifel, daß viele dieser Infektionen durch Ärzte, wenn auch nur mittelbar durch Verschleppung und Einpflanzung von Scheide- und Schamkeimen in die Plazentarstelle selbst, veranlaßt worden sind.

Ich schließe daraus, meine Herren:

Je sorgfältiger die Nachgeburtsperiode geleitet wird, d. h. je geduldiger die Ausstoßung der Nachgeburt den Naturkräften überlassen wird, desto weniger leicht werden Nachblutungen eintreten, desto seltener wird es zur Verhaltung der Plazenta oder einiger ihrer Abschnitte kommen, desto eher entfällt die Notwendigkeit, die für eine Infektion so gefährliche manuelle Plazentarlösung vornehmen zu müssen.

Eine Verminderung der Zahl dieser gefährlichen Eingriffe ist sicher zu erwarten, wenn bei Wehenschwäche nicht wegen dieser, sondern nur trotz dieser eingegriffen wird, d. h. wenn die Wehenschwäche an sich keine Anzeige zur Beendigung einer Geburt gibt, sondern nur deren Folgezustände, sofern diese Gefahr für Mutter und Kind bedingen. Muß einmal trotz der bestehenden Wehenschwäche eine Operation vorgenommen, etwa die Zange angelegt werden, so müssen Sie in einer solchen Zwangslage sich gegen eine etwaige Nachblutung durch rechtzeitige Darreichung von Ergotin vor Beendigung der

1) Zentralbl. f. Gyn. 1908, S. 107.

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Geburt so gut, wie es geht, sichern[1]), einen Rat, den ich schon lange (Geburtsh. Operationslehre Seite 180) lehre. Daß unter allen Umständen verhaltene Plazentarteile, wenn deren Fehlen erkannt wird, alsbald zu entfernen sind, brauche ich Ihnen gegenüber nicht näher auszuführen. Dagegen ist die Entfernung verhaltener Eihäute, selbst des ganzen Eihautsackes völlig unnötig und gefährlich, selbst bei keimfreier Hand, weil ein Mitverschleppen von Genitalkeimen in die Uterushöhle niemals sicher vermieden werden kann. Wie mir eine vielhundertfältige Beobachtung innerhalb 20 Jahren lehrt, genügt es in allen solchen Fällen, während der ersten Woche des Kindbettes Ergotin darzureichen und zweimal täglich die Scheide auszuspülen.

Daß häufiger einfache leichte Temperatursteigerungen nach Verhaltung von Eihäuten, in Basel 15% gegen 8—9%, eintreten, ist richtig. Diese haben aber nichts zu bedeuten, wenn man sich nicht zu unnötigen und um diese Zeit des Wochenbettes sehr gefährlichen Eingriffen, wie etwa Ausschabungen, verleiten läßt.

Die Vorbeugung des Kindbettfiebers im Wochenbett befolgt die gleichen Grundsätze wie während der Geburt. Außer Abspülen der Scham ist gegebenenfalls durch Ergotin und heiße Scheidenspülungen Sorge zu tragen für eine rasche Rückbildung der Gebärmutter, nötigenfalls für eine raschere Ausstoßung verhaltener Eihäute oder Blutgerinnsel, bei übelriechendem Wochenflusse selbst ohne Fieber, gegen Blutungen infolge mangelhafter Rückbildung oder Thrombenlösungen. Solcher Spülungen wären zwei bis dreimal täglich mit 3% Bazillol, 3% Therapogen, 2% Seifenkresol, auch wohl mit Chlorwasser 1:3 oder Jodwasser 1:3000 anzuordnen.

Von der allergrößten Wichtigkeit aber ist der Grundsatz: Während des Wochenbettes ist, wenn irgend möglich, keinerlei operativer Eingriff, insbesondere keiner innerhalb der Gebärmutter vorzunehmen. Auch die sekundäre Naht ist zu unterlassen, ich habe nach solcher mehrfach Wöchnerinnen an Verschleppungsbakteriämie sterben sehen. Und wie oft sind nicht nach unnötigen Ausschabungen wegen verhaltener Eihaut oder aus sonst einem sehr notwendigen Grunde, z. B. nach einer Entfernung von Plazentarpolypen, schwere und tödliche Erkrankungen ausgelöst worden? Kein Handschuh, keine Ausspülungen, keine Einpinselungen schützen sicher vor Einimpfung der Spaltpilze des Wochenflusses, unter welchen sich auch bei völlig gesunden Frauen in sehr vielen Fällen die gefürchteten Streptokokken

1) Unter den vielen gangbaren Ergotinpräparaten verdient im Bereiche der Geburtshilfe das Sekakornin die erste Stelle, während in der gynäkologischen Praxis das Extractum secalis cornuti fluidum vorzuziehen ist.

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befinden, in die frischen Wunden, die bei diesen Eingriffen unbedingt entstehen müssen.

Auf eine besonders strenge Ruhelage einer sonst gesunden Wöchnerin zu dringen, ist gewiß übertrieben und unnötig! Etwa 2—3 Tage Rückenlage, dann Seitenlage, am 5. Tage Aufsitzen und am Anfang der zweiten Woche Aufstehen sollte vollkommen genügen. Eine längere Bettruhe ist nur nach besonders schweren Geburten, nach Verletzungen — Dammrissen — nach Blutungen, bei Verhaltung von Eihäuten wie überhaupt bei minderwertiger Körperentwicklung und selbstverständlich bei Erkrankungen aller Art, besonders streng bei Wundinfektionen, nötig.

Meine Herren! Ich bin am Schlusse meiner kurzen Übersicht der wichtigsten Vorbeugungsmaßregeln des Kindbettfiebers angekommen — jene während der Schwangerschaft fallen in das Gebiet der Hygiene dieser Zeit. Wenn ich noch einmal das Wesentliche zusammenfassen darf, so kann ich nur das wiederholen, was ich früher gesagt habe:

Je ausgedehnter, je tiefer eine Verletzung unter der Geburt gesetzt wird, desto größer ist die Wahrscheinlichkeit des Ausbruches eines Kindbettfiebers, das besonders dann gefährlich werden wird, wenn die Schädigung unter dem Deckmantel einer Scheinantisepsis gesetzt wird. Eigenkeime, die sonst kein Unheil anrichten, Fremdkeime, die etwa von der Hand einer Hebamme herrühren und zunächst verhältnismäßig harmlos sind, werden durch ein Trauma — Zange, Plazentarlösung — gar zu leicht in die Tiefe der Gewebe — in Wunden, in die Plazentarstelle — eingepreßt. Sie können nunmehr unbehelligt von den wirksamsten Schutzwehren des Körpers, die noch nicht zur Stelle sein können, ihre unheilvolle Tätigkeit widerstandslos entfalten. So stellt die Vieltuerei mancher Ärzte zu guter Letzt und häufig die Hauptursache des Stillstandes, ja der Zunahme des Wochenbettfiebers dar!

Ergreifen Sie, meine Herrn, die Initiative, bekämpfen Sie nachdrücklichst eine mißverstandene chirurgische Ära der Geburtshilfe, es wird Ihnen sicher ein köstlicher Sieg über das Kindbettfieber erblühen. Die Regierungen mögen aber auch ihrerseits sich ihrer Pflicht auf diesem Gebiete erinnern und mit ihren Machtmitteln die Ausmerzung bedenklicher Auswüchse auf dem Gebiete der Vorbeugung des Kindbettfiebers, insbesonders bei den Desinfektionsvorschriften für Hebammen erleichtern!

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(Gynäkologie Nr. 178.)

Über abdominale und vaginale Köliotomien.

Eine vergleichende Studie über den Wert und die Leistungsfähigkeit beider Operationen nebst Bemerkungen zur Indikationsstellung und Technik.[1])

Von

J. Pfannenstiel,

Kiel.

Dank der Fortschritte auf dem Gebiete der pathologischen Anatomie einerseits und der Vervollkommnung der allgemeinen chirurgischen Technik andrerseits haben die Köliotomien im Laufe der Jahre eine derartige Entwicklung erreicht, daß es dem Operateur ermöglicht ist, mit großer Zuversicht an den Eingriff heranzugehen.

Nicht zum wenigsten hat zu dieser Vervollkommnung beigetragen die durch die Einführung der vaginalen Köliotomie in die gynäkologische Operationstechnik hervorgerufene Konkurrenz zwischen den beiden Operationsverfahren der abdominalen und der vaginalen Köliotomie. Beide Operationsarten sind heute in einer Weise ausgearbeitet, daß es uns, rein technisch betrachtet, in der Tat möglich ist, beinahe alle gynäkologischen Eingriffe, die wir an interperitoneal gelegenen Organen zu erledigen haben, auf beide Arten auszuführen, durch den abdominalen und durch den vaginalen Weg.

Es ist daher vielleicht an der Zeit, einmal Umschau zu halten über das gesamte Arbeitsgebiet und die Leistungsfähigkeit festzustellen, welche den beiden genannten Operationsverfahren innewohnt. Daß sie beide

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1) Vortrag, bestimmt für den am 26.—28. Mai 1908 zu Philadelphia tagenden Kongreß der American Gynecological Society.

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ihre Vorzüge haben, ist klar; sonst würden sie nicht nebeneinander bestehen können. Aber wo Licht ist, ist auch Schatten. Es gilt daher vor allem, neben den Vorzügen auch der Nachteile zu gedenken, welche die beiden Methoden in sich tragen und danach zu konstatieren, auf welchem Wege wir die uns anvertrauten Patienten am schnellsten und sichersten zu dem Ziele einer guten und dauerhaften Heilung zu führen vermögen.

Bei der Wahl eines Operationsverfahrens spielen naturgemäß zum nicht geringen Teil rein äußerliche Momente eine gewisse Rolle, vor allem die persönliche Übung und Gewohnheit des Operateurs, sowie gewisse Liebhabereien. Allein diese Gesichtspunkte sollten niemals maßgebend sein bei der Wahl eines Operationsverfahrens, sondern einzig und allein das leibliche Wohl des Kranken. Auch ästhetische Rücksichten, wie die Schönheit oder Sichtbarkeit einer Narbe sollte nicht schwer in die Wagschale fallen, es ist vielmehr derjenige Weg vorzuziehen, welcher sowohl primär wie sekundär die besten Chancen darbietet für Leben und Gesundheit des Menschen.

Vergleichen wir von diesem Gesichtspunkte die beiden Operationsverfahren ganz im allgemeinen miteinander, so ist es klar, daß die abdominale Köliotomie ganz bedeutende Vorzüge besitzt: Die Möglichkeit, die Öffnung in der Bauchwandung beliebig groß anzulegen, erschließt den Vorzug der größtmöglichen Übersicht über das Operationsfeld im engeren und weiteren Sinne. Die zu operierenden Organe können ohne allzu große Dislokation in Angriff genommen werden. Die Operation kann mit einer Sorgfalt durchgeführt werden, wie sie besser nicht gedacht werden kann. Wir können mit der gleichen Sicherheit dem Prinzipe des Konservativismus und des Radikalismus Sorge tragen, je nach den Erfordernissen des Falles. Ganz abgesehen von der Möglichkeit, die dem Verlauf der großen Gefäßstämme folgenden Lymphdrüsen wo es nötig ist aufzusuchen, gibt es keine größere Übersicht über die so schwer zugänglichen Befestigungsstellen der inneren Genitalien. Die Laparotomie gestattet auch am sichersten eine Schonung der benachbarten Organe. Die Operation kann ohne Schwierigkeiten des Zuganges auf Nachbarorgane ausgedehnt werden, wie Blase, Ureteren, Rektum, Flexur und die Appendix des Blinddarmes, wie auf andere, höher gelegene Organe des Leibes, Därme, Netz, Mesenterium, Gallenblase, Magen usw. Die Laparotomie gibt uns auch am raschesten Gewißheit über die Grenzen der Operabilität. Die Laparotomie gestattet ferner in ganz besonders sorgfältiger Weise die intraperitonealen Wundflächen mit Peritoneum zu versorgen und trägt damit wesentlich bei zur Vermeidung stören-

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der Verwachsungen in der Bauchhöhle und zur Verhütung des postoperativen Ileus. Endlich gewährt die Laparotomie die Möglichkeit, bei solchen Neubildungen, welche eine gewisse Neigung haben, durch Implantation von Geschwulstteilchen in der Bauchhöhle oder in der Schnittnarbe Metastasen zu bilden, solche Implantationen zu verhüten. Es kommen hier besonders die epithelialen Ovarialtumoren in Betracht, welche auch in Gestalt der ganz gutartigen Kystome zu dieser Form von Metastasenbildung neigen. Bei hinreichend großer Öffnung in der Bauchwandung und sorgfältiger Technik in der Herausbeförderung der Geschwulst, sei es mit, sei es ohne Verkleinerung der Kystome, gelingt es, die Entstehung von Narbenimplantationen zu verhindern. In dieser Beziehung ist die abdominale Operation der vaginalen ganz entschieden überlegen, insofern das ganz besonders schädliche Morcellieren der Tumoren in Wegfall kommt, welches bei dem vaginalen Operieren oft nicht zu vermeiden ist. Inwieweit das bei der Indikationsstellung bezüglich der Ausführung der Ovariotomie von Wichtigkeit ist, soll noch gezeigt werden.

Die Nachteile und Gefahren der Laparotomie liegen in der komplizierten Zusammensetzung der Bauchdecken, die wir durchschneiden müssen, und in der hinter Därmen versteckten Lage der Genitalien. Ersterer Umstand hat die Narbenhernie im Gefolge gehabt, letzterer hat viel schlimmere Übelstände gezeitigt: die Gefahr einer Schädigung des Bauchfells in größerem Umfang mit ihren Folgen für die Rekonvaleszenz und für das Leben der Kranken durch Eintritt peritonitischer Entzündung.

Allein diese Nachteile haben an Bedeutung verloren durch Verbesserung der Asepsis und Technik, und wir können heute sagen, daß es mit großer Sicherheit gelingt, ebensowohl Narbenhernien vollkommen zu vermeiden, wie den Eintritt einer Peritonitis zu verhüten, vorausgesetzt, daß wir an einem aseptischen Operationsobjekt zu arbeiten haben.

Es ist deshalb die Laparotomie das gegebene Verfahren für alle im Sinne der Asepsis reinen Fälle von Neubildungen, Blutergüssen und Mißbildungen, aber auch von chronischen Entzündungen der Genitalien, sie ist also das gegebene Verfahren für die gutartigen Neubildungen des Uterus (Myome), für die Tumoren der Ovarien, Tuben und Ligamente, für die Extrauteringravidität, für komplizierte Gynatresien, für die chronische Pelveoperitonitis adhaesiva usw.

Nicht absolut vermeidbar sind die geschilderten Gefahren der abdominalen Köliotomie, wenn es sich handelt um ein nicht aseptisches Operationsobjekt. Hier kann der vaginale Weg der bessere sein. Es ist klar, daß man einen intraperitoneal gelegenen abgekapselten

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Eiterherd, einen Beckenabszeß oder eine verjauchte Hämatozele oder einen im Douglas verlöteten Tuben- oder Ovarialabszeß nicht auf dem Wege an gesunden Därmen vorbei, also abdominal, zur Entleerung bringen wird. Ebenso kann es wichtiger sein, infizierte Organe, sofern sie überhaupt der Exstirpation bedürfen, vaginal zu entfernen, also ein infiziertes Myom oder total vereiterte, mit multiplen Abszessen durchsetzte chronisch kranke innere Genitalien. Aber es muß auch die Gewähr gegeben sein, daß das infektiöse Material durch solchen Eingriff sicher und vollkommen entfernt wird und daß die Prinzipien des Konservativismus und des Radikalismus je nach der Individualität des Falles gewahrt bleiben.

Der Scheidenbauchschnitt hat im allgemeinen den Vorzug, daß die Operation besser vertragen wird und daß selten eine sichtbare oder störende Narbe zurückbleibt. Daß die Operation besser vertragen wird, drückt sich aus in einer besseren Mortalitätsstatistik und in einer glatteren Rekonvaleszenz. Das trifft bei der vaginalen Köliotomie gewiß zu, allein bei näherer Betrachtung stellt sich heraus, daß dieser Satz nur für eine bestimmte Kategorie von Fällen gilt. Es unterliegt keinem Zweifel, daß eine vaginale Totalexstirpation des karzinomatösen Uterus bessere Resultate gibt als eine abdominale, selbst wenn dieselbe nicht radikal ausgeführt wird. Es ist ebenso sicher, daß bei nicht ganz vollkommener subjektiver Asepsis die vaginalen Resultate besser sind als die abdominalen. Darüber hat uns die frühere Zeit zur Genüge belehrt. Aber in heutiger Zeit hat die vaginale Fixation des Uterus keine bessere Mortalitätsstatistik als die ventrale Fixation, und auch die vaginale Ovariotomie steht in den Fällen, in denen sie überhaupt gut ausführbar ist, nicht besser als die gleiche Operation vom Abdomen ausgeführt. Hat man also ein aseptisches Operationsobjekt vor sich, so stehen sich beide Operationswege von diesem Gesichtspunkte aus gleich und nur bei der Operation an einem nicht aseptischen Organ, wie z. B. an einem karzinomatösen Uterus oder auch nur bei unvollkommener Asepsis des Operateurs macht sich der Unterschied geltend. Die Ursache ist nicht schwer zu finden. Sie liegt in der bereits vorhin erwähnten Lagerung der Genitalien unterhalb der Därme. Sind weder am Operationsobjekt noch an den Händen des Operateurs krankmachende Keime, so schadet die Berührung der Därme nicht. Sind aber solche Keime vorhanden, so ist der Weg an den Därmen vorbei gefährlich, während das subintestinale Operieren besser vertragen wird.

Das gleiche, was von der Mortalitätsstatistik gesagt wurde, gilt natürlich für die Rekonvaleszenz nach der Operation. Bei der

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heutigen Asepsis und Technik hat die ventrale Fixation oder die Ovariotomie keine schlechtere Rekonvaleszenz als die Vaginofixation oder die vaginale Ovariotomie, besonders seitdem die Kranken nach dem Leibschnitt nicht mehr so unnötig lange Bettruhe halten.

Daß eine Kolpoköliotomie für gewöhnlich keine sichtbare Narbe hinterläßt, ist klar. Aber wenn man überhaupt auf das kosmetische Resultat Wert legt, was ich für recht unwesentlich halte, so läßt sich durch eine gut ausgeführte Technik und eine sorgfältige Naht die Bauchnarbe sehr dünn und fein gestalten, ja, sie kann sogar fast unsichtbar gemacht werden, wie ich noch zu erläutern haben werde.

Zuweilen ist die vaginale Narbe zwar „unsichtbar", aber doch recht lästig und dauernd schmerzhaft, sowohl die im Scheidengewölbe liegende als ganz besonders die Scheidendammnarbe, welche nach schwierigen, eine große Inzision erfordernden Operationen entsteht. Ich habe sowohl in der eigenen Praxis als in der von andern Operateuren sehr häßliche Narben gesehen, Narben, welche die Patienten besonders beim Sitzen und Gehen hinderten, Narbenbeschwerden, wie man sie heutzutage bei der Laparotomie gar nicht mehr zu sehen bekommt.

Und damit komme ich auf die Nachteile der Kolpoköliotomie zu sprechen, welche gipfeln in der zu kleinen Öffnung mit allen ihren Folgen. In der Zeit, in denen die vaginalen Operationen ausgearbeitet wurden, hat diese kleine Öffnung großen Schaden gestiftet und sogar manches Opfer durch ungenügende Blutstillung, durch Verletzung von Blase, Uretèren und Mastdarm und durch Fehler in der Asepsis gefordert. Heutzutage, wo die Indikation für diese Operation enger begrenzt worden ist, ist es besser geworden, aber der Übelstand der kleinen Öffnung macht sich immer wieder von neuem geltend, besonders seitdem wir die innigen Beziehungen zwischen dem Blinddarm und den inneren Genitalien kennen gelernt und die Notwendigkeit eingesehen haben, die Radikaloperation beim Karzinom zu erweitern. Alle Operationen, bei denen ein vollständiger Überblick über das ganze Operationsgebiet nebst seiner näheren und ferneren Umgebung notwendig ist, passen nicht für den vaginalen Schnitt.

Es eignet sich deswegen die Scheidenoperation im allgemeinen nicht für die Karzinome der inneren Genitalien. Daß die bösartigen Ovarialneubildungen auf vaginalem Wege radikal und sorgfältig nicht zu entfernen sind, darüber ist man sich wohl allerseits im klaren, und ebenso wird das Uteruskarzinom von den meisten Führern der gynäkologischen Wissenschaft abdominal erledigt. Nur Schauta operiert vaginal mit seinem eigens

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dazu ausgearbeiteten erweiterten Verfahren. Die Zukunft muß entscheiden, ob dieses Verfahren in summa die gleiche Leistung aufzuweisen hat, wie die abdominale Operation. Daß der Vaginalist auf die Drüsenentfernung verzichtet, halte ich nicht für so bedenklich, aber ob die Ausräumung der Parakolpien und Parametrien bezüglich Radikalismus einerseits und Schonung der Nachbarorgane andrerseits ebenso vollkommen ausfällt wie bei abdominalem Operieren, das erscheint mir fraglich. Mein Standpunkt bezüglich des Uteruskarzinoms ist in der Berl. klin. Woche 1905, Nr. 27 gekennzeichnet. Im allgemeinen soll der Uteruskrebs abdominal erledigt werden. Aber wir sollten die guten Erfahrungen, die wir in früherer Zeit mit der vaginalen Exstirpation gewisser Karzinomarten gemacht haben, ausnützen und solche Fälle auswählen lernen und sie für die zweifellos mit geringerer primärer Mortalität belastete vaginale Exstirpation reservieren. Es sind das gewisse Formen von Portio- und von Korpuskarzinom, nämlich solche, die sich noch im Beginn der Ausbreitung befinden, die erst in höherem Lebensalter auftreten und die hart, bindegewebsreich und zellarm sind. Speziell an der Portio kommen in Betracht die am eigentlichen Muttermund beginnenden Karzinome. Alle diese Formen, welche langsam wachsen und wenig zur Drüseninfektion neigen, können getrost vaginal erledigt werden. Alle andern, besonders die weiter vorgeschrittenen Fälle, die weicheren, zellreichen Formen, die jugendlichen Fälle, besonders aber alle Cervixkarzinome sollten ebenso wie die primären vaginalen Karzinome vom Abdomen aus so radikal wie möglich in Angriff genommen werden.

Von den gutartigen Neubildungen der inneren Genitalien werden die Myome, wenn sie eine gewisse Größe überschreiten, am besten abdominal erledigt. Ich bin von der weitgehenden Anwendung der vaginalen Operation wieder etwas zurückgekommen, nachdem ich gesehen habe, daß die Resultate in denjenigen Fällen, die durch ihre geringfügige Größe sich auch für das vaginale Verfahren eignen, auch bei abdominalem Operieren nicht schlechter sind. Überkindskopfgroße Myomen werden schonender und sorgfältiger abdominal erledigt, aber auch bei kleineren Tumoren ziehe ich die Laparotomie vor, wenn der Introitus und die Scheide eng, virginell und straff sind, weil mir der Schnitt durch die Bauchdecken schonender erscheint als der paravaginale Hilfsschnitt. Nur eine Indikation anerkenne ich für die vaginale Myotomie, das ist die Operation bei bestehender Infektion eines myomatösen Uterus. Es sind das meist ziemlich harmlose Infektionen mit saprämischen Mikroben, bei denen aber doch die abdominale Operation schlechter vertragen wird

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als die vaginale. In solchen Fällen soll man sich bemühen, vorausgesetzt, daß der vaginale Zugang nicht zu eng ist, womöglich auch noch die überkindskopfgroßen Tumoren unter geschickter Zerstückelung vaginal zu entfernen. Dazu gehört besondere Übung, ein besonderes Instrumentarium und eine gute Assistenz.

Was die Ovarientumoren anlangt, so sind von vornherein von der vaginalen Operation auszuschließen alle soliden und alle malignen Tumoren, sowie alle durch Verwachsung, intraligamentären Sitz, Stieltorsion und andre Störungen komplizierte Fälle. Aber auch die unkomplizierten, zystischen Tumoren werden besser abdominal entfernt, weil auf diese Weise am sichersten der Implantation von Geschwulstteilchen in der Bauchhöhle vorgebeugt wird. Die kleine Öffnung im Scheidengewölbe bei der Kolpoköliotomie bringt es mit sich, daß die Geschwulst oftmals nicht ohne Morcellement zerkleinert werden kann, was bei allen epithelialen Neubildungen entschieden zu verwerfen ist. Es sollte deshalb die vaginale Ovariotomie allgemein fallen gelassen werden. In den einfachen Fällen ist sie überflüssig und der Laparotomie an Erfolg nicht nachstehend. In den schwierigen Fällen, besonders bei den parvilokulären Kystomen und ganz besonders bei den bösartigen Tumoren ist sie geradezu gefährlich. Alle diese Zustände sind nun aber ebenso wie die zuvor genannten Komplikationen nicht mit Sicherheit vor der Operation zu diagnostizieren. Ich lehne deshalb die vaginale Ovariotomie trotz ihrer oft bequemen Ausführbarkeit im Prinzip ab. Ausnahmen von dieser Indikationsstellung werden ebenso wie bei den Myomen auch bei den Ovarialtumoren gemacht werden müssen. Bei der Einklemmung eines zystischen Tumors sub partu kann gelegentlich die vaginale Operation vorzuziehen sein usw.

Eine besondere Stellung nehmen ein die Ovarialabszesse bzw. die vereiterten, unilokulären, im Becken liegenden Tumoren. Hier empfehle ich folgendes Verhalten: Es wird zunächst durch vorsichtige Probepunktion mit dünner Nadel die Art der in dem Abszeß enthaltenen Bakterien festgestellt. Findet sich ein septischer Inhalt, so soll vaginal inzidiert und die Ausheilung der Abszeßhöhle abgewartet werden, um später den Tumor abdominal zu entfernen. Bei harmlosen Bakterien, ebenso bei allen größeren und multilokulären Tumoren ist ohne Zaudern abdominal zu operieren, mit großem Bauchschnitt und unter dem Bestreben, den Tumor möglichst unzerkleinert zu entfernen.

Was die entzündlichen Erkrankungen anlangt, so wurde der abszedierenden Formen schon gedacht. Sofern das infektiöse Material vollständig entfernt werden kann, soll der vaginale Weg bevorzugt werden. Bei den chronischen Entzündungen jedoch ist schon mit

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Rücksicht auf die Erfordernisse des konservativen Handelns der übersichtliche Weg der abdominalen Operation der zweifellos bessere. Soviel wie möglich das Ovarium schonen, muß die Losung sein, bei allen chronisch entzündlichen Erkrankungen einschließlich der Tuberkulose der innern Genitalien. Die Tube und der Uteruskörper sind bei allen entzündlichen Erkrankungen viel stärker gefährdet als der Eierstock, sie bedürfen deshalb — vorausgesetzt, daß überhaupt eine Operation indiziert ist — in der Regel viel mehr der radikalen Entfernung. Vor allem allem aber ist mit Rücksicht auf die so häufige Mitbeteiligung des Processus vermiformis an der chronischen Entzündung die Laparotomie angezeigt.

Zu der chronischen Entzündung der Genitalien gehört auch die chronische Pelveoperitonitis. Sofern dieselbe andauernde Beschwerden macht, ist die abdominale Operation auszuführen. Auch die mit chronischer Pelveoperitonitis verbundene Retroflexio uteri (sog. „Retroflexio uteri fixati") gehört zu dem Bereich der Laparotomie, nicht der vaginalen Operation.

Auch die Extrauterinschwangerschaft wird am besten per laparotomiam erledigt. Gewiß ist es möglich, die Tube auch durch die Kolpoköliotomie zu entfernen, aber angesichts des weichen und morschen Zustandes der Gewebe ist ein schonendes und exakt blutstillendes Operieren allein durch die Laparotomie zu gewährleisten. Nur die verjauchte Hämatozele ist wie ein Beckenabszeß zu behandeln uud vaginal zu inzidieren.

Die komplizierten Gynatresien gehören gleichfalls der Laparotomie, während der einfache Hämatokolpos natürlich von unten zu entleeren ist. Bei allen höher gelegenen Atresien kommt man am besten abdominal zum Ziele und braucht dabei den konservativen Gedanken nicht aus dem Auge zu verlieren. Man kann sogar, wie ich gezeigt habe[1]), unter Umständen die fehlende Verbindung zwischen dem durch retiniertes Blut ausgedehnten Uteruskavum und dem Scheidenrest herstellen und zwar mit dem Effekt der vollen Funktion.

Was die Lageveränderungen der Genitalien anlangt, so stehe ich auf dem Standpunkt, daß hier im allgemeinen viel zu viel operiert wird. Meistens ist überhaupt keine Lagekorrektion angezeigt. Wo dies der Fall ist, handelt es sich in der Regel um kompliziertere Fälle. Der Deszensus und Prolapsus der Ovarien kann gelegentlich Beschwerden machen. Es ist klar, daß hier nur die Laparotomie zu helfen vermag, durch Ovariopexie.

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1) Pfannenstiel in Festschrift für Fritsch, Leipzig, Breitkopf u. Härtel 1902, S. 344 ff.

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Die Retroflexio uteri bedarf in unkomplizierten Fällen niemals der Lagekorrektion. Zu den Komplikationen gehört die Pelveoperitonitis, von welcher bereits die Rede war. Hier ist die ventrale Operation angezeigt, bestehend in Lösung der Verwachsungen einschließlich der erforderlichen Operationen an den Adnexen mit nachfolgender Ventrifixur, um den Uterus aus dem Verwachsungsgebiet herauszuheben. Die andere überaus häufige Komplikation der Retroflexio ist die Totalerschlaffung des Organs und die Senkung der Scheide. Schlaffheit des Isthmus uteri (unteres Uterinsegment) und Atonie der Beckenbodenmuskulatur gehen fast immer Hand in Hand. Zu diesem Krankheitsbild gehört dann neben andern splanchnoptotischen Erscheinungen (Wanderniere, Senkung des Magens und Querdarms usw.) auch die Zystozele. Alle diese Erschlaffungszustände des Urogenitalapparates lassen sich nicht auf dem abdominalen Wege heilen. Hier ist die einzige Methode, welche auf einen Dauererfolg bezüglich der Beckenorgane rechnen kann, eine richtig ausgeführte vaginale Fixation des Uterus, verbunden mit Kolporrhaphie und Dammplastik. Weder die Ventrifixur noch die Alexander-Adamsche Operation gibt gute dauernde Resultate: der schlaffe Uterus, welcher durch ebenso schlaffe Bandapparate nicht genügend gehalten ist, bleibt trotz ventraler oder inguinaler Fixation nicht in der durch die Operation hergestellten „Normallage", wenn man überhaupt von einer solchen sprechen darf, er senkt sich wieder, ev. unter Ausbildung von langgedehnten Fixationsbändern, und das Rezidiv ist da. Vor allem aber besteht die Gefahr der Wiederausbildung einer Zystozele mit ihren Folgen für Uterus und Scheide. Diese läßt sich durch eine ihren Hebel allein am Corpus uteri ansetzende Operation nicht beheben, im Gegenteil: durch eine ventrale oder inguinale Befestigung des Uterus wird die Blase noch stärker in der Richtung nach abwärts gedrängt, so daß die besten Kolporrhaphiemethoden nicht standhalten. Und mit dem Zystozelenrezidiv tritt das Prolaps- und Retroflexionsrezidiv ein. Hier kann nur die Interposition des Uterus zwischen Scheide und Blase helfen, also die vaginale Operation, bestehend in einer Kombination von gut ausgeführter Vaginofixation mit entsprechender Colphorrhapia anterior, posterior und Dammplastik.

Mit diesen Ausführungen habe ich natürlich nur die hauptsächlichsten Indikationen besprechen können; es würde mir die Zeit fehlen, um noch andere, vielleicht auch nicht ganz unwichtige Gesichtspunkte heranzuziehen. Ich kann resümieren, daß nach meiner Auffassung und nach meiner Erfahrung für die intraperitonealen gynäkologischen Operationen die abdominale Köliotomie bei weitem den Vorzug verdient, daß aber auch

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für die Kolpokõliotomie eine Anzahl ziemlich scharf umgrenzter Indikationen übrigbleibt. Hat man noch vor nicht langer Zeit Grund gehabt, das Prinzip aufzustellen: was irgend der vaginalen Operation zugänglich ist, soll vaginal erledigt werden, so haben die Fortschritte in der Asepsis und in der Technik der letzten 10 Jahre diesen Grundsatz geändert. Die Asepsis hat bedeutend gewonnen durch die Einführung des Alkohols in die Händedesinfektion, sowie die Verwendung der Gummihandschuhe und andrer Maßnahmen zur Verbesserung des Wundschutzes. Die Technik, welche eine Zeitlang etwas vernachlässigt war in dem irrigen Glauben, daß die Antisepsis die Hauptsache sei, ist wieder mehr zu ihrem Recht gekommen. Wir sind heute mehr denn je überzeugt, daß die Schonung der biologischen Kräfte des Körpers von großer Wichtigkeit ist, sowohl bezüglich der allgemeinen Vitalität wie ganz besonders der lokalen Gewebsfunktion, daß wir geschickt, schnell und sorgfältig operieren müssen, daß aber die Sorgfalt wichtiger ist als die Schnelligkeit. Die Organe und Gewebe sind vor unnützen Berührungen und besonders vor stärkerem Druck zu bewahren. Die lang anhaltende Kompression der Bauchdecken durch Spekula, namentlich durch die selbsthaltenden, ist zu meiden. Das Bauchfell ist vor Luftkeimen und sonstigen Schädlichkeiten zu schützen, die Därme sollen womöglich nicht eventriert werden. Desinfizientien sind von Wunden und Bauchfell fernzuhalten. Die Beckenhochlagerung soll nicht zu steil sein und nicht zu lange andauern, nicht allein wegen der stärkeren Belastung des Zwerchfells durch Intestina mit ihren Folgen für die Atmung, sondern auch weil dadurch eine zu intensive Hyperämie des Peritoneums entsteht. Die Blutstillung soll peinlich genau sein. Die interperitonealen Wunden sind sorgfältigst mit Peritoneum zu übernähen. Drainage und Tamponade sind nach Möglichkeit zu vermeiden. Als Nahtmaterial ist für die versenkten Nähte Catgut vorzuziehen und Silkwormgut für die Haut. Die Vor- und Nachbehandlung des Kranken soll auf die Schonung seiner Kräfte Bedacht nehmen und so viel wie möglich den physiologischen und individuellen Verhältnissen angepaßt sein.

Als beste Methode für die abdominalen Köliotomien empfehle ich meinen suprasymphysären Faszienquerschnitt, welcher bei richtiger Anwendung für etwa $^9/_{10}$ aller gynäkologischen Laparotomien gut anwendbar ist. Der Schnitt durch die Haut kann erforderlichenfalls von einer Spina des Darmbeins zur andern geführt werden, der Schnitt durch die Aponeurosen kann bequem über die lateralen Ränder der Musculi recti nach außen verlängert werden, wobei die Musculi obliqui entsprechend ihrer Faserrichtung zu

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spalten sind. Die Ablösung der Aponeurosen vom Musculus rectus soll nach oben bis zum Nabel, nach unten bis zur Symphyse gehen, wobei die Musculi pyramidales an der Faszienplatte bleiben. Wenn alsdann die Musculi recti und das Bauchfell in der Linea alba getrennt sind, dann haben wir eine sehr große Öffnung der Bauchwand, welche für die größten und eingreifendsten Operationen geeignet ist und besonders das Operieren in der Tiefe und in den Seitenteilen des Beckens erleichtert. Ich habe bei nahezu 1000 Operationen nicht einmal nötig gehabt, zu dem Querschnitt einen Längsschnitt durch die Faszie in der Richtung nach oben über den Nabel hinaus hinzuzufügen. Die Naht der Bauchdecken wird in vier Etagen ausgeführt: 1. Bauchfell, 2. Rektusmuskeln, 3. Aponeurose, 4. Haut. Die Peritoneal- und Muskelnaht wird mit einem fortlaufenden Catgutfaden ausgeführt, die Fasziennaht ebenso mit einem Catgutfaden, die Hautnaht mit Silkwormknopfnähten. Auf sorgfältige Blutstillung (zur Vermeidung von Hämatomen) und genaue Vereinigung und Adaptierung der Wundränder lege ich den größten Wert.

Diese Art des Leibschnittes hat den großen Vorteil, daß er den Därmen schon bei mäßiger Beckenhochlagerung großen Schutz vor der Außenwelt gewährt und damit einer Reihe von Schädigungen vorbeugt. Es macht sich dies sehr deutlich bemerkbar in der Mortalitätsstatistik. Ich hatte in der Zeit von 1902—1904 bei 456 Fällen von Faszienquerschnitt eine Gesamtsterblichkeit von 5,24% und in der gleichen Zeit bei 104 Längsschnitten in der Linea alba eine solche von 9,25%, wobei ich bemerken muß, daß in der Summe der Querschnittfälle auch die unsere moderne Statistik stark belastenden Uteruskarzinomoperationen, sowie andere an einem nicht aseptischen Objekt auszuführenden Operationen enthalten sind.

Die Rekonvaleszenz vollzieht sich leichter und die Heilung geht rascher vonstatten bei den mit Querschnitt Operierten, namentlich seitdem ich nach dem Vorbilde amerikanischer Operateure und in Deutschland nach dem Vorgange Krönigs die Kranken zeitig aufstehen und sich bewegen lasse.

Die Heilung der Bauchdecken war eine absolut gute (prima intentio) in 95,4% bei Querschnitt gegenüber 94,4% bei Längsschnitt. Erscheint auch dieser Unterschied minimal, so spricht er doch insofern zugunsten des Faszienquerschnittes, als ich gerade die eitrigen und infektiösen Erkrankungen durch den Querschnitt zu erledigen pflege, vor allem auch das Uteruskarzinom, die Peritoneal- und Genitaltuberkulose und die entzündlichen Adnexerkrankungen.

In innigem Zusammenhang mit der Bauchdeckenheilung steht die Beschaffenheit der zurückbleibenden Narbe und die Frage der Bauch-

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hernie. Die Narbe ist äußerst fein, niemals breit oder entstellend, sie verschwindet entsprechend der Spaltrichtung der Haut in der Unterbauchgegend allmählich mehr und mehr, zuweilen bis zur völligen Unsichtbarkeit, meist wird sie verdeckt durch die Querfalte der Haut, welche dieser Gegend eigentümlich ist. Narbenhernien wurden in dem Gesamtmaterial beobachtet in 0,94%, wobei ich bemerke, daß dieselben ausschließlich in den Fällen von Bauchdeckeneiterung eintraten. In den Fällen, in denen prima intentio zu verzeichnen war, ist 0mal eine Hernie beobachtet worden (bei mehr als 300 nach Jahr und Tag untersuchten Fällen).

In den verhältnismäßig seltenen Fällen, in denen ich einen Längsschnitt in der Linea alba ausführe (bei sehr großen, soliden Tumoren, bei Komplikationen derart, daß eine Operation am Nabel oder in höher gelegenen Regionen der Bauchhöhle erforderlich ist), halte ich nach den bisherigen Erfahrungen die Schnittführung nach Lennander und die von Ch. Noble geübte Fasziennaht für die besten Methoden.

Bezüglich der Kolpoköliotomietechnik kann ich mich kurz fassen: Ich halte den Weg durch das vordere Scheidengewölbe, also zwischen Blase und Cervix, mit Döderlein für entschieden gekünstelt, wenn ich auch zugeben muß, daß die Operation eine bessere Übersicht zu geben pflegt als die Kolpotomia posterior. Da ich beide Operationen nur noch selten und ausnahmsweise ausführe, so unterlasse ich es, an dieser Stelle auf Einzelheiten der Technik dieser Operationsmethode einzugehen.

Bei Beckenabszessen ist der Weg durch das hintere Scheidengewölbe selbstverständlich. Bei vaginalen Totalexstirpationen individualisiere ich, indem ich zuweilen vorn, zuweilen hinten den Schnitt beginne. Die mediane Durchschneidung des Uterus oder sonstige Zerstücklung kann ratsam sein. Ist die Radikaloperation wegen Vereiterung der Genitalien erforderlich, so kann es wünschenswert sein, Dauerklemmen für 2 Tage anzulegen und Jodoformgazedrainage anzuwenden. Sonst ist, wenn irgend möglich, das Peritoneum und die Scheidenwunde sorgfältig in Etagen zu schließen.

Für die Prolapsoperation ist das vordere Scheidengewölbe der gegebene Weg. Ich lege hier Wert darauf, die Blase nicht allein in der Mitte, sondern auch an der Seite vollkommen von Cervix und Scheide abzutrennen, um sie möglichst vollständig nach oben schieben und den Uterus zwischen Blase und Scheide einschalten zu können. Es ist dies die einzige Möglichkeit, um dem Wiedereintritt einer Zystozele vorzubeugen. Die Annähung des Uterus an die Scheide soll ferner nicht am Fundus, sondern im oberen Drittel der vorderen

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Korpusfläche geschehen. Der vorn auszuschneidende Scheidenlappen (Colporrhaphia anterior) soll nicht zu breit sein, statt dessen ist die Scheide seitlich etwas zu unterminieren, um dem Uterus Platz zu schaffen für die Interposition. Die Naht der Scheide soll ausgeführt werden, ohne daß die Portio vor die Vulva gezogen wird, weil es sonst nicht gelingt, die Portio hinten und mehr oben zu erhalten. Eine entsprechende Kolpoperineorrhaphie vervollständigt die Operation, indem sie den gesunkenen Genitalien einen neuen, festen Halt und Untergrund verschafft.

Hiermit schließe ich meine Ausführungen, indem ich mir vollkommen bewußt bin, daß ich nicht alle bei der operativen Behandlung der interperitonealen Genitalerkrankungen in Betracht kommenden Gesichtspunkte erörtert habe. Es genügte mir, die allgemeinen Grundsätze zu besprechen, welche nach meiner Auffassung bei der Wahl der Operationsmethoden gelten sollten, und zu zeigen, wie es möglich ist, diesen Grundsätzen Rechnung zu tragen. Im übrigen muß natürlich das Bestreben walten, so viel wie möglich zu individualisieren, zum Heile des Patienten, und weiterhin die Fortschritte zu verwerten, welche wir von der chirurgischen Kunst noch zu erwarten haben.

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489/90/91.

(Chirurgie Nr. 142/43/44.)

Über den heutigen Stand der Erkennung und Behandlung der Appendizitis. I.

Erörtert an 550 von Geheimrat Garrè behandelten Fällen.

Von

Dr. Ad. Ebner,

Königsberg-Pr.

Nur selten haben sich innerhalb kurzer Zeit auf dem therapeutischen Gebiet einer Erkrankung so tiefgreifende Änderungen der Anschauungen sowohl der internen Ärzte, wie der Chirurgen vollzogen, wie auf dem der Erkrankung des Wurmfortsatzes und ihrer Folgezustände. Zu danken ist dieser Fortschritt im wesentlichen den immer weiter gestellten Indikationen der Chirurgen, welche schließlich zum operativen Eingriff in jedem Stadium der Erkrankung geführt haben. So gewährte die Autopsie in vivo einen immer größeren Einblick in die z. T. so außerordentlich verderblichen und heimtückischen Veränderungen dieses Organs und warf ein immer helleres Licht auf das grelle Mißverhältnis, das namentlich auch in einem frühen Stadium schon zwischen den geringen klinischen Erscheinungen und der Schwere der anatomischen Veränderungen an dem erkrankten Organ selbst bestehen kann.

Durch dieses Mißverhältnis drängte sich dann immer noch mehr die Notwendigkeit einer möglichst weitgehenden Indikationsstellung zum operativen Eingriff auf, so daß man heute allgemein die Appendizitis nicht nur bei Ärzten, sondern auch schon großenteils im Publikum als eine Krankheit anerkennt, bei der dem Chirurgen nicht nur das letzte, sondern am richtigsten bereits das erste Wort mitzureden zusteht, welche im wahren Sinne des Wortes als eine chirurgische Erkrankung zu bezeichnen ist.

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Dementsprechend ist innerhalb einer verhältnismäßig kurzen Zeit das Anschwellen der Appendizitisliteratur — Sprengel führt in seinem Standartwerk über Appendizitis allein 117 Seiten Literatur an — ein so gewaltiges geworden, daß es für den Spezialisten schwer und für den praktischen Arzt kaum noch möglich ist, dieselbe in ihrem vollen Umfange zu übersehen und sich in dem zum Teil noch recht auseinandergehenden Streit der Meinungen zurechtzufinden, die verschiedenartigen Benennungen und Einteilungen dieser Erkrankung auseinanderzuhalten und, was vor allem für den Patienten das wichtigste ist, in den verschiedenen Stadien der Erkrankung den richtigen Zeitpunkt zur Operation herauszufinden, in welchem er seinen Patienten dem Chirurgen zu überliefern hat, ohne ihn erst der Gefahr einer Allgemeinperitonitis auszusetzen und damit die Chancen seiner Heilung wesentlich zu verschlechtern oder ihn zum mindesten einem langen und quälenden Krankenlager zu überliefern.

Das idealste im Interesse des Patienten wäre ja fraglos die schon von manchen Chirurgen und theoretisch mit Recht gestellte Forderung, zu jeder als Appendizitis auch nur verdächtigen Erkrankung einen Chirurgen frühzeitig hinzuzuziehen. Demgegenüber darf man aber nicht verkennen, daß einerseits noch lange Zeit vergehen dürfte, bis die Berechtigung dieser Forderung allgemeine Anerkennung bei den praktischen Ärzten finden wird, die ja vielfach noch gewöhnt sind, in der Appendizitis, bzw. den einzelnen Anfällen, die sie ja meist nur zu sehen bekommen, eine relativ harmlose Erkrankung zu erblicken. Andererseits sind aber auch bei dem besten Willen die Ärzte auf dem Lande oder in kleineren Städten gar nicht in der Lage, gleich einen Chirurgen an der Hand zu haben, auf dessen Erfahrung sie bei Überwachung ihres Falles sich stützen könnten.

Es wird also nach wie vor das Schicksal der meisten Appendizitiskranken lediglich in der Hand der praktischen Ärzte liegen, und es werden nach wie vor diese auch die Verantwortung für den Erfolg oder Mißerfolg eines operativen Eingriffs zu tragen haben. Denn darüber darf heute kein Zweifel mehr herrschen, daß fast niemals die Operation als solche — unter Voraussetzung der notwendigen Technik und Erfahrung des Operateurs — die Verantwortung bei einem Mißerfolg zu tragen hat, sondern ausschließlich der mehr oder minder günstige Zeitpunkt, in welchem der Kranke in die rettende Hand des Chirurgen gelangt, mit anderen Worten der, welcher diesen Zeitpunkt zu bestimmen hat, und das wird fast immer der praktische Arzt sein.

Für diesen möchte ich daher in erster Linie die im Laufe von 12 Jahren an der Rostocker, Königsberger und Breslauer chirurgischen Klinik gemachten Beobachtungen verwerten, die sich auf 550

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von Herrn Geheimrat Garrè behandelte bzw. operierte Fälle erstrecken, für deren gütige Überlassung ich an dieser Stelle nicht verfehlen möchte, meinem ehemaligen, sehr verehrten Chef, Herrn Geheimrat Garrè, meinen ergebensten Dank zum Ausdruck zu bringen.

Dabei gleichzeitig ein möglichst kurzes und übersichtliches Bild der bisher in der Literatur niedergelegten Anschauungen, soweit sie für die Betätigung des praktischen Arztes am Krankenbett in Frage kommen, in dieser anspruchslosen Studie zu liefern, soll in erster Linie mein Bestreben sein.

Es liegt daher im Interesse der Kürze und Übersichtlichkeit unserer Abhandlung, wenn wir unsere Aufmerksamkeit vor allem der Benennung, Einteilung, Entstehung, Erkennung und Behandlung der Appendizitis zuwenden und uns mit anderen Worten auf die rein klinische Seite der Appendizitis beschränken. Wer sich auch über die pathologisch-anatomischen Veränderungen näher zu informieren wünscht, dem seien die eingehenden Monographien von v. Brunn, Fowler, Körte, Sonnenburg, Rotter und ganz besonders das Standardwerk von Sprengel zur eingehenden Durchsicht empfohlen.

Wenden wir uns zunächst der Benennung der Erkrankung des Wurmfortsatzes zu, so hat sich als die in Deutschland am meisten gebrauchte von den verschiedenen, vorgeschlagenen Bezeichnungen die Perityphlitis herausgeschält. Der Ausdruck stammt noch aus jener Zeit her, in der man den Ausgangspunkt der Erkrankung vielfach in dem Typhlon, dem eigentlichen Blinddarm, erblickte, woher ja auch die volkstümliche Bezeichnung der Blinddarmentzündung ihren Ausgang genommen hat. Diese Anschauung fand ihren Ausdruck in der zuerst von Sahli widerlegten Lehre von der Typhlitis stercoralis, deren Ursache man in Kotstauungen im Typhlon erblicken zu müssen glaubte. Im Gegensatz zu der intraperitonealen Entzündung um das Typhlon herum, der Perityphlitis, hat man die extraperitoneal bzw. retrocöcal gelegene Entzündung als Paratyphlitis bezeichnet. Im Einklang dazu hat dann Küster als dritten Ausdruck die Epityphlitis, die Entzündung des Epityphlon, des auf dem Typhlon gelegenen Anhangs, hinzugefügt. Diese letztere Bezeichnung insbesondere kann man nicht als sehr glücklich betrachten, denn in logische Analogie mit den ersten Bezeichnungen gebracht heißt sie weiter nichts, als eine auf dem Typhlon gelegene Entzündung, gleichwie die Perityphlitis einer um das Typhlon gelegenen Entzündung entspricht. Es ist kein logischer Grund vorhanden, warum hier mit dem Begriff einer Entzündung und ihrer Lokalisation plötzlich der Begriff eines neuen Organs — im Gegensatz zum Typhlon — verbunden werden soll, das die relativ wenig bekannte Bezeichnung Epityphlon trägt. Zum mindesten

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setzt diese Benennung voraus, daß jeder Leser wissen muß, was Küster darunter verstanden wissen will, denn aus der reinen Analogie mit der Peri- und Paratyphlitis allein geht eine richtige Deutung der Epityphlitis im Sinne der Auffassung von Küster doch nicht hervor.

Auch Küster hat sich bei seinem Vorschlage wohl von der Erwägung leiten lassen, daß es wenig logisch erscheint, eine Erkrankung unter Übergehen des eigentlichen Ausgangspunktes derselben nach einem anderen in der Nähe gelegenen Organ zu benennen, das in keiner näheren Beziehung zu derselben steht, als der, welche man aus früheren und mittlerweile unrichtig befundenen Anschauungen hinsichtlich der Ätiologie der Erkrankung herleiten kann. Dann müßte er aber konsequenterweise von der Epityphlitis eine Periepityphlitis und Paraepityphlitis ableiten, was kaum zu einer wesentlichen Vereinfachung und leichteren Unterscheidung der Begriffe für den Praktiker führen dürfte.

Viel näher liegend, klarer und den tatsächlichen Verhältnissen entsprechend ist wohl für die Erkrankung des Appendix allein die einfache Bezeichnung Appendizitis, wie sie zuerst in Amerika von Fitz 1886 vorgeschlagen und zunächst von den amerikanischen Ärzten, namentlich von Senn vertreten und eingeführt worden ist. Diese Bezeichnung hat denn auch mittlerweile derartigen Anklang gefunden, daß sie heute die antiquierte Perityphlitis überall, namentlich bei den englisch sprechenden Nationen, mehr und mehr in den Hintergrund drängt.

Auch Sprengel hält den Ausdruck Perityphlitis nicht mehr für zeitgemäß und findet, daß die Bezeichnung Appendizitis entschiedene Vorzüge hat, wenn man das erste sprachliche Grauen überwunden hat. „Sie ist kurz, eignet sich für weitere, wenn auch formell nicht einwandfreie Ableitungen, wie Endoappendizitis, Peri- und Paraappendizitis, ist sachlich unseren heutigen Anschauungen absolut entsprechend und in fast allen Kulturländern, außer vielleicht in Deutschland eingebürgert."

Küster hält der Benennung entgegen, daß sie den Fehler der Zusammenstellung eines lateinischen Namens mit einer griechischen Endung habe. Da wir heute aber in der Medizin eine ganze Reihe ähnlicher Zusammenstellungen bereits haben, an denen niemand Anstoß nimmt, und da der Gesichtspunkt der reinen Zweckmäßigkeit doch wichtiger sein dürfte, als der einer sprachreinen Ableitung und Zusammenstellung unserer Nomenklatur, so wird man trotz der philologischen Bedenken den Ausdruck auch ferner beibehalten dürfen. Daraus die Möglichkeit einer Verwechslung mit den Appendices epiploicae herzuleiten, wie sie Hoffmann in einer Arbeit bei Küster für wahrscheinlich hält, erklingt doch etwas gezwungen, da einmal

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reine Entzündungen der Appendices epiploicae äußerst selten sind, wenn sie idiopathisch überhaupt vorkommen, und da andererseits die Appendizitis bereits viel zu sehr im Sprachgebrauch auch in Deutschland eingebürgert ist, als daß jemand einen anderen Begriff damit verbinden würde, als den einer Erkrankung des Appendix. In gleicher Weise entspricht auch der Gebrauch des männlichen Artikels für Appendix durchaus dem Sprachgebrauch, indem das an Stelle des deutschen Wurmfortsatzes oder des lateinischen Processus vermiformis getretene, zunächst weniger gebrauchte Fremdwort den männlichen Artikel (desselben) übernommen hat. Spricht doch auch der Luftschiffer in wissenschaftlichen Werken stets von dem, und nirgends von der Appendix des Ballons. Man darf eben dem Sprachgebrauch nicht jede Berechtigung absprechen. Er setzt sich schließlich in der Praxis dennoch stets durch, wenn auch vom theoretischen Standpunkt aus die rein wissenschaftlichen Ableitungen, wie die Appendix u. a. durchaus zu Recht bestehen bleiben.

Wie weit man im übrigen mit rein sprachlichen Ableitungen kommen kann, zeigt sehr schön die seinerzeit von Nothnagel vorgeschlagene Bezeichnung Skolekoiditis, die von der griechischen Benennung des Appendix „Skolekoeides apophysis“ abgeleitet ist und den Vorzug einer für manche ziemlich schwierigen Aussprache mit einer sprachreinen Ableitung der Endung verbindet. Zudem klingt die Bezeichnung wegen des nur wenig bekannten griechischen Ableitungswortes für die Mehrzahl so fremd, daß sie außer vom rein sprachlichen Gesichtspunkt wohl kaum eine Berechtigung zur Erwerbung des Bürgerrechtes in unserer Nomenklatur hat. Tatsächlich habe ich sie auch in keiner Einteilung außer bei Rose erwähnt gefunden.

Der von manchen gebrauchte Ausdruck Perityphlitis appendicularis für den gleichen Krankheitszustand drückt ja die Ätiologie richtig im Adjektivum aus, warum man aber die Periappendizitis nicht vorziehen soll, die denselben Begriff ebenso klar, nur kürzer zum Ausdruck bringt, ist nicht einzusehen.

Früher oder später wird die Bezeichnung aller Voraussicht nach wohl die alleinherrschende werden, sowohl der Logik, wie der Zweckmäßigkeit nach. Einstweilen ist der von früher übernommene Ausdruck der Perityphlitis ein so festgewurzelter, daß man noch auf Jahre hinaus mit ihm zu rechnen haben wird. Nur soll man sich dabei gegenwärtig halten, daß er einen Krankheitszustand betrifft, der bereits über den Appendix hinausgegriffen hat. Er entspricht somit einem sekundären Stadium der Erkrankung, in welchem allerdings erst die meisten Ärzte ihre Patienten zu Gesicht bekommen, und für diese Fälle

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besteht wenigstens vom anatomischen Gesichtspunkt aus die Perityphlitis zu Recht.

Gehen wir nun zu der Einteilung der Appendizitis in die einzelnen Stadien der Erkrankung über, so finden wir darin bei den verschiedenen Autoren gewisse prinzipielle Unterschiede, je nachdem die Einteilung auf rein ätiologisch-genetischer, topographischer, pathologischer oder klinischer Basis erfolgt ist. Da nun die meisten Einteilungen weniger auf Grund der rein klinischen Symptome, als auf Grund der Autopsie — sei es in vivo oder in mortuo — und auf Grund eines Vergleiches bzw. einer Resümierung des hierbei gebotenen Befundes mit den vorherigen klinischen Symptomen aufgestellt worden sind, so wird naturgemäß für die meisten der Einteilungen der pathologisch-anatomische Gesichtspunkt der hauptsächlich maßgebende sein, während der klinische erst in zweiter Linie kommt.

Daß dabei eine vollständige Übereinstimmung zwischen beiden Gesichtspunkten nur schwer oder gar nicht zu erzielen ist, ergibt sich aus der Tatsache, daß in vielen Fällen die klinischen Symptome mit den per autopsiam erhobenen Befunden entweder wenig übereinstimmten oder gar in einem schreienden Mißklang dazu standen. Es liegt daher auf der Hand, daß bei Einteilungen, welche mehr oder weniger beiden Punkten gerecht zu werden suchen, auch theoretisch der Einklang zwischen beiden Punkten nur schwer herbeizuführen ist, zum mindesten wird dieser Einklang für die Praxis vielfach nur ein scheinbarer sein.

Für den Praktiker wird daher in erster Linie eine rein klinische Einteilung in Frage kommen, und daß diese eine möglichst einfache sein wird und muß, ergibt sich einmal aus der Forderung einer möglichst großen Übersichtlichkeit und Klarheit derselben, sowie aus der Erwägung heraus, daß gerade die einfachste und kürzeste klinische Einteilung am ehesten allen pathologisch-anatomischen Gesichtspunkten gerecht zu werden vermag, ohne durch ein zu subtiles Eingehen auf die Einzelheiten den Mißklang zwischen klinischen und pathologischen Befunden unnötig zum Ausdruck zu bringen. So sehr auch die Einteilungen auf rein pathologisch-anatomischer Basis vom theoretisch wissenschaftlichen Standpunkt aus zu schätzen sind, so sind sie dennoch praktisch undurchführbar, da sie eben klinisch nicht zu machen sind.

Daß man namentlich in letzter Zeit seitens unserer klinischen Autoren immer mehr Wert darauf legt, dieser richtigen Erkenntnis Ausdruck zu verleihen, ergibt sehr schön eine vergleichende Durchsicht der früheren Einteilungen im Gegensatz zu den heutigen. Es tritt dabei eine immer größere Einfachheit und ein immer größeres

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Vorwiegen des rein klinischen Gesichtspunktes in die Erscheinung. Kommt es doch für den praktischen Arzt sowohl, wie für das Wohl des Kranken vielmehr darauf an, an der Hand einer kurzen und übersichtlichen Einteilung auf rein klinischer Basis schnell und präzise den richtigen Zeitpunkt zum rettenden operativen Eingriff bestimmen zu können, als in dem Bestreben, einer möglichst subtilen Einteilung die Diagnose seines Falles anzupassen, Gefahr zu laufen, daß dieser wichtige Zeitpunkt versäumt wird.

Die schönste und subtilste Diagnose nützt eben in solchen Fällen dem Patienten nichts, wenn sie nicht schnell gestellt wird und wenn dabei in jedem nicht ganz leichten Fall nicht der Gesichtspunkt der einzig maßgebende ist: Wann ist es Zeit zum operativen Eingriff, wann kann oder wann muß der Arzt pflichtgemäß den Patienten der Hand des Chirurgen überliefern?

Zum Schluß möchte ich noch einige Sätze Sprengels anführen, in denen er in geradzu klassischer Weise für die Vorzüge einer rein klinischen Einteilung eintritt: „Der von einigen (Sonnenburg) aufgestellte Grundsatz, daß nur diejenige Einteilung wissenschaftlich berechtigt sei, welche auf pathologisch-anatomischer Grundlage beruhe, ist theoretisch anfechtbar, praktisch undurchführbar und tatsächlich unmöglich.“ Man darf eben nicht vergessen, daß die angewandte Wissenschaft in erster Linie für den Praktiker und Patienten da ist.

Ferner: „Jede Klassifizierung nach pathologisch-anatomischen Grundsätzen muß in der ideellen Absicht unternommen werden, in der Praxis ihren Prüfstein zu finden. Solange wir sie nur mit dem exstirpierten Wurmfortsatz vor uns probat erfinden, bleibt sie von des Gedankens Blässe angekränkelt.“

Wenn ich nun auch nicht im einzelnen auf die verschiedenen Einteilungen vorwiegend deutscher Autoren des näheren eingehe bzw. dieselben besonders anführe, da dieselben lediglich vom theoretischen Gesichtspunkt aus Interesse darbieten, so möchte ich doch in einem kurzen Hinweis auf dieselben versuchen, dem Praktiker ein Bild von dem Reichtum an solchen, sowie den Gesichtspunkten zu geben, welche für die Autoren dabei maßgebend waren. Diejenigen, welche sich eingehender dafür interessieren, verweise ich auf die außerordentlich umfassende Zusammenstellung der Einteilungen, welche Sprengel allerdings nicht nach vergleichenden Gesichtspunkten, sondern nach chronologischer Reihenfolge geordnet in seinem Standardwerk bringt.

Zunächst den rein ätiologisch-genetischen Standpunkt vertritt eine Einteilung, welche Haim 1905 angegeben hat. Er geht dabei von dem Grundsatz aus, daß die Appendizitis als eine rein bakterielle

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Erkrankung zu bezeichnen sei, welche endemischen Charakter besitzt, und macht dementsprechend seine Unterscheidung nach der Art der verschiedenen Infektionserreger, die dabei in Betracht kommen können.

Einen mehr ätiologisch-pathologischen Gesichtspunkt vertreten die Einteilungen von Czerny, Sonnenburg und Federmann. Dabei legt Sonnenburg besonderen Wert auf die Betonung des ätiologischen Momentes und hebt gleich Czerny den Perforationsvorgang durch eine besondere Abteilung für denselben besonders hervor. Federmann vertritt naturgemäß die gleiche Einteilung wie Sonnenburg mit dem Unterschied, daß er zu der Appendicitis simplex auch die serös-fibrinöse Exsudation im freien Peritoneum hinzunimmt, nicht ganz in Übereinstimmung mit der Auffassung seines Chefs Sonnenburg, da eine solche Exsudation nicht stattfinden kann, wenn das Peritoneum absolut gesund ist, wie es Sonnenburg für seine Appendicitis simplex ausdrücklich hervorhebt.

Im übrigen hebt auch Federmann die Identität der perforativen Peritonitis mit der zirkumskript-eitrigen Peritonitis und ebenso der gangränösen Appendizitis mit der freien, progredienten Peritonitis in der Art seiner Einteilung hervor. Wenn es nun für die Mehrzahl der Fälle auch zutreffen mag, daß eine Perforation in der Regel von einer zirkumskripten Peritonitis und eine Gangrän des Processus von einer progredient-diffusen Peritonitis gefolgt sein wird, so wird es doch eine große Anzahl von Fällen geben, für welche diese Identität und somit auch die Einteilung Sonnenburgs nicht zutreffend ist. Zudem kann erwiesenermaßen der Perforationsvorgang am Appendix derartig larviert in seinen klinischen Äußerungen verlaufen, daß er als solcher klinisch überhaupt nicht kenntlich ist, so daß er als Unterscheidungsmerkmal für eine Einteilung vom klinischen Standpunkt aus wenig geeignet erscheint.

Es liegt in der Natur der Sache begründet, daß wir bei den pathologisch-anatomischen Einteilungen auch die der ältesten Autoren vertreten finden, da diesen ja zunächst die pathologischen Obduktionsbefunde die einzigen Unterscheidungsmerkmale für eine Einteilung liefern konnten. So unterscheidet Bamberger 1853 in einer der ältesten Einteilungen zwischen intraperitonealen Entzündungen, meist vom Processus oder vom Cöcum ausgehend, die schlechthin als Typhlitis bezeichnet werden, und extra- oder retroperitonealen Entzündungen in dem der hinteren Cöcalwand angelagerten Bindegewebe, die als Perityphlitis bezeichnet werden. Es fällt hierbei auf, daß man damals dem Begriff der Perityphlitis dieselbe Bedeutung unterschob, welche wir heute durch die Bezeichnung Paratyphlitis zum Ausdruck bringen, während man die Typhlitis in erweiterter

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Bedeutung entsprechend unserer heutigen Perityphlitis für die intraperitoneal lokalisierten Entzündungsvorgänge in Anwendung brachte. Oppolzer hebt bereits 1858 diesen Unterschied sehr richtig hervor, indem er unter Perityphlitis die Entzündung des serösen Überzuges der Darmgebilde und unter Paratyphlitis die Entzündungen im retrocöcalen Bindegewebe versteht, eine Auffassung, die bis heute ihre Gültigkeit nicht verloren hat. Von neueren Einteilungen könnte man hierzu rechnen diejenige von Carl Beck 1898, Bloß 1894 und von Büngner 1904. Dieselben vertreten vorwiegend den pathologischen Gesichtspunkt und geben im einzelnen Unterschiede, die man wohl mit dem Appendix in der Hand nach Eröffnung der Bauchhöhle — wie Sprengel sagt — zu machen in der Lage ist, die aber rein klinisch zu machen für den Praktiker unmöglich ist.

Etwas mehr dem pathologisch-klinischen Standpunkt nähern sich Roux 1892, Rotter 1896, Dörffler 1898, Jonescu 1903. Jedoch finden wir bei Roux wieder dem Perforationsvorgang eine besondere Abteilung eingeräumt, was aus den oben besprochenen Gründen nicht sehr glücklich erscheint. Auch Sahli äußert sich mit Recht gegen eine derartige Gruppierung nach perforativen und nicht perforativen Formen, weil sie nicht immer wesentlich verschiedene Krankheitsbilder hervorzurufen brauchen.

Rotter unterscheidet einfach zwischen der Perityphlitis circumscripta und diffusa, indem er dabei den Begriff der primären Appendizitis unter den sekundären der Periappendizitis unterordnet. Letzteres erscheint mir etwas unmotiviert, da beide Begriffe klinisch gut voneinander zu trennen sind.

Den Mißklang des pathologischen und klinischen Prinzips der Einteilungen und die daraus resultierende Unbrauchbarkeit derselben für den Praktiker hat besonders Hans Dörffler empfunden, der selbst als praktischer Arzt tätig ist. Trotzdem hat er bei seinem Versuch einer derartigen Einteilung vom rein praktischen Gesichtspunkt aus sich nicht ganz vom pathologischen Prinzip freimachen können, wie seine Unterscheidung zwischen Periappendicitis serosa und suppurativa beweist, die man aus dem Vorhandensein oder Fehlen des Fiebers vor eingetretener Abkapselung doch nicht immer mit Sicherheit machen kann. Ebenso erscheint mir seine Gleichstellung der perforativen mit der eitrigen Periappendizitis, von denen die letztere der weitere Begriff ist, nicht zweckmäßig, da in vielen Fällen die Perforation als klinisches Unterscheidungsmerkmal versagt und andererseits nicht jede eitrige auch eine Perforationsperitonitis zu sein braucht.

Dagegen ist die Einteilung von Jonescu ziemlich frei von pathologischen Anklängen bis auf das Hervorheben der septischen oder

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eitrigen Peritonitis und die gesonderte Abteilung der Appendicitis hypertoxica, die wohl richtiger als Periappendizitis zu bezeichnen wäre. Einen Unterschied zwischen allgemeiner Peritonitis appendicularis und appendikulärer Septhämie zu machen ist ebenfalls nicht empfehlenswert, da beide Formen dieselben Veränderungen und Symptome aufweisen können und sich nur durch größere Virulenz der Erreger und entsprechend vermehrte Schnelligkeit des deletären Verlaufes der Erkrankung voneinander unterscheiden, ein Punkt, der auch bei Sprengel entsprechende Berücksichtigung gefunden hat.

Als rein klinisch darf man wohl die Einteilungen von Rose, Broca, Scholz, Riedel und Sprengel bezeichnen, von denen die Einteilung von Rose mehr durch ihre Eigenart, als durch ihre Brauchbarkeit Beachtung verdient. Am kürzesten und relativ zweckmäßigsten erscheinen mir die Einteilungen der letzteren Autoren, von denen Scholz einfach zwischen leichten, mittelschweren und schweren Fällen unterscheidet. Auch Riedel hält eine Trennung vom anatomisch-pathologischen Standpunkt nicht für statthaft und schlägt vor, nur zwischen Appendicitis non purulenta und purulenta zu unterscheiden. In beiden Einteilungen haben jedoch meines Erachtens die klinisch erkennbaren und trennbaren Erscheinungen des Peritoneums zu wenig Beachtung gefunden, so daß sie nach dieser Richtung etwas über das Ziel hinausschießen dürften.

Diesen Nachteil vermeidet die Einteilung von Broca, ohne im übrigen dadurch an Übersichtlichkeit und Kürze einzubüßen. Es wird darin nämlich unterschieden:

1. akute einfache Appendizitis (ohne Peritonitis),
2. zirkumskripte Peritonitis (adhäsive oder eitrige),
3. diffuse Peritonitis (septische oder eitrige),
4. chronische Appendizitis (mit oder ohne akute Anfälle).

Die drei letzten Abteilungen faßt Sprengel in eine zusammen, indem er gleichwie Riedel und Scholz nur zwei Gruppen unterscheidet, die er als Appendicitis simplex und Appendicitis destructiva bezeichnet. Eine Trennung in A. ulcerosa, perforativa und gangraenosa hält Sprengel für hinfällig, da sie in der Peritonealhöhle die gleichen Veränderungen verursachen, worauf bereits oben gelegentlich der Appendicitis hypertoxica von Jonescu hingewiesen ist.

Wenn man nun trotz der zahlreichen und zum Teil recht brauchbaren Einteilungen es wagen darf, eine weitere Einteilung vorzuschlagen, so werden für dieselbe vom Standpunkt des Praktikers allein das klinische und das therapeutische Prinzip in Frage kommen. Ersteres wird vornehmlich beherrscht von den Reiz- bzw. Entzündungserscheinungen des Peritoneums. Durch sie allein wird der Stand der Er-

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krankung klinisch erkennbar nach außen projiziert, ganz gleich ob dieselben von einer Gangrän, Perforation oder Ulzeration ihren Ausgang genommen haben. Diese allein können also für eine klinisch verwertbare Einteilung in Betracht zu ziehen sein.

Folgen wir von diesem Gesichtspunkt aus dem durchschnittlichen Entwicklungsgang der Erkrankung, so werden sich gewisse klinisch in der Regel trennbare Stadien ergeben, bei denen jedoch zu bedenken ist, daß die Erkrankung günstigenfalls in einem frühen Stadium abbrechen kann, so daß die späteren Stadien fortfallen, daß sie aber ungünstigenfalls — bei großer Virulenz oder massenhaftem Einbruch der Erreger in das Peritoneum und verminderter Widerstandsfähigkeit desselben — die früheren Stadien so schnell durchmachen bzw. überspringen kann, daß diese so gleichfalls in Fortfall kommen können. Eine Möglichkeit, die ja für die Wandlung der Indikationsstellung zum operativen Eingriff in den letzten Jahren ausschlaggebend geworden ist.

Nach dieser Reihenfolge der Vorgänge würde sich dann folgende Einteilung ergeben, deren klinische Unterscheidungsmerkmale und Vorgänge wir gelegentlich der Besprechung der Symptomatologie der Appendizitis näher erörtern werden:

1. Appendicitis simplex. Peritoneum völlig frei. Die Erkrankung hat noch nicht auf die Serosa übergegriffen und begreift in sich den Katarrh des Appendix mit vermehrter Schleimabsonderung und die weiter fortgeschrittene Form der Erkrankung, welche sich durch Granulationsgewebe zwischen den tubulösen Drüsen und Neigung zu Blutungen des Bindegewebes auszeichnet und von Riedel als Appendicitis granulosa haemorrhagica bezeichnet wird. Diese letztere darf man wohl, abgesehen von akzidentellen Ursachen, auf die wir später zu sprechen kommen werden, für die Mehrzahl der Fälle als Ausgangspunkt der Erkrankung betrachten.

2. Periappendicitis incipiens. Die Erkrankung hat auf die Serosa des Appendix und seiner näheren Umgebung übergegriffen, aber sich noch nicht fest gegen die Umgebung abgegrenzt. Ich habe dieses Stadium bisher in keiner Einteilung erwähnt gefunden, halte aber dessen besondere Aufstellung und Umgrenzung auch vom klinischen Standpunkt aus für berechtigt und notwendig, da vom therapeutischen Standpunkt aus dieses Stadium tatsächlich bereits eingeführt und benannt worden ist. Es ist dieses das sogenannte intermediäre Stadium der Erkrankung, das heute besonders aktuell ist durch den noch nicht einwandsfrei entschiedenen Streit der Meinungen über die Indikationsstellung zum operativen Eingriff während desselben. Einer-

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seits spricht die therapeutische Aufstellung dieses besonderen intermediären Stadiums dafür, daß man es auch klinisch bis zu einem gewissen Grade von den anderen Stadien unterscheiden kann, worauf wir später näher zu sprechen kommen werden, und andererseits tritt für die weitere Umgrenzung desselben die Erfahrungstatsache ein, daß der Zeitraum vom Übertritt der Entzündung auf das Peritoneum bis zur festen Umgrenzung derselben in der Regel sich bis zum achten Krankheitstage abzuspielen pflegt. Diese beiden Gründe lassen es meines Erachtens gerechtfertigt erscheinen, wenn man dieses tatsächlich vorhandene und therapeutisch bereits im Gebrauch befindliche Stadium der Appendizitis auch für die klinische Einteilung derselben gesondert anführt. Wenn die ursprünglich von der zeitlichen Umgrenzung abgeleitete Bezeichnung des intermediären Stadiums in unserer Einteilung durch Periappendicitis incipiens ausgedrückt wird, so geschieht das, um das diesem Zeitraum in der Regel entsprechende Verhalten des Peritoneums zum Ausdruck zu bringen und damit gleichzeitig eine Übereinstimmung mit den anderen Bezeichnungen unserer Einteilung herbeizuführen.

3. Periappendicitis (und Paraappendicitis) circumscripta. Die über die Serosa des Appendix hinausgegangene Entzündung des Peritoneums ist zur Abgrenzung und zum festen Abschluß gekommen. Dieselbe kann serofibrinöser Natur oder der Regel nach mehr purulenter Natur mit entsprechender Exsudatbildung sein, die meist in einem deutlich lokalisierten Palpationsbefunde nachweisbar ist. Zeitlich würde dieses Stadium dem sogenannten Spätstadium entsprechen.

4. Periappendicitis diffusa, entsprechend der Peritonitis appendicularis diffusa purulenta, welche ohne Begrenzung entweder schubweise fortschreitend (Peritonitis fibrinopurulenta appendicularis von Mikulicz), oder gleichmäßig sich in schnellem Tempo verbreitend (Peritonitis appendicularis libera nach Sprengel oder acuta progrediens), oder gewissermaßen mit einem gewaltigem Sprunge auf einmal innerhalb kürzester Zeit (appendikuläre Septhämie oder Appendicitis hypertoxica bei Jonescu, foudroyante allgemeine septische Peritonitis bei Dörffler) das gesamte Peritoneum ergreift. Die Schnelligkeit ihres Fortschreitens ist abhängig von der Zahl und Virulenz der angreifenden Erreger einerseits und der natürlichen Widerstandskraft des Peritoneums andererseits.

5. Die abgelaufene oder chronisch rezidivierende Periappendizitis bzw. das chronische Stadium mit dem völligen Rückgang sämtlicher akuten und subakuten Entzündungserscheinungen des Peritoneums und den aus den zurückgebliebenen Entzündungsresten resultierenden, meist nur wenig hervortretenden Beschwerden, welche

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klinisch neben der Anamnese der Diagnose die einzige Handhabe darbieten.

Vom rein therapeutischen Standpunkt hat sich zur Festlegung der operativen Indikationsstellung und Prognose in den einzelnen Stadien der Erkrankung eine etwas andere Einteilung herausgebildet, welche nach zeitlichen Umgrenzungen des Krankheitsverlaufes aufgestellt ist, sich aber im großen und ganzen mit unserer klinischen Einteilung decken muß, da beide nach denselben Gesichtspunkten, die klinische nach den tatsächlich vorhandenen und die therapeutische nach den erfahrungsgemäß vorauszusetzenden Veränderungen des erkrankten Peritoneums, gemacht worden sind.

Eine entsprechende Zusammenstellung beider Einteilungen würde sich daher folgendermaßen gestalten:

1. Das Frühstadium der Erkrankung (absolute Frühoperation), welches umgreift den ersten und zweiten Tag vom Beginn der Erkrankung gerechnet. Die gewonnenen Operationsbefunde in demselben entsprechen in der Regel der Appendicitis simplex, sowie häufig der Periappendicitis incipiens.

2. Das Zwischenstadium (Intermediärstadium) vom dritten bis achten Tage der Erkrankung gerechnet, entspricht dem von uns unter Periappendicitis incipiens verstandenen Erkrankungszustand des Peritoneums. (Zwischenoperation = Intermediäroperation.)

3. Das Spätstadium vom neunten Tage der Erkrankung ab gerechnet, entspricht der Periappendicitis circumscripta (Spätoperation).

4. Das Notstadium (Notoperation) oder richtiger Zuspätstadium, welches naturgemäß zeitlich nicht umgrenzt ist, der therapeutischen Vollständigkeit halber jedoch ebenfalls angeführt werden muß und der Periappendicitis diffusa entspricht. Therapeutisch sind bei ihm die ersten beiden Tage vom Beginn der diffusen Erkrankung ab von Wichtigkeit wegen der relativ günstigen Prognose für den operativen Eingriff während derselben (relatives Frühstadium; relative Frühoperation).

5. Das chronische Stadium, in welchem die Operation die größte Sicherheit bietet: Sicherheitsstadium; Sicherheitsoperation.

Rein schematisch würde sich also folgende Parallele ergeben:

1. Absol. Frühstadium = Appendicitis simplex oder Periappendicitis incipiens: absolute Frühoperation.

2. Zwischenstadium = Periappendicitis incipiens: Zwischenoperation.

3. Spätstadium = Periappendicitis circumscripta: Spätoperation.

4. Notstadium = Periappendicitis diffusa: Notoperation in 48 Std.; relatives Frühstadium: relative Frühoperation.

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5. Sicherheitsstadium = Periappendicitis chronica: Sicherheitsoperation.

Ich glaube, daß eine derartige Einteilung, welche in gleicher Weise den klinischen und therapeutischen Gesichtspunkten gleichzeitig gerecht zu werden vermag ohne an Übersicht und Verständlichkeit zu verlieren, am ehesten dem Bedürfnis des Praktikers genügen dürfte zur Orientierung am Krankenbett sowohl, wie in der an einer verschiedenartigen Benennung gleicher oder korrespondierender Begriffe noch laborierenden Literatur. Im übrigen hat sie den Vorteil, daß sie den häufigen Verwechselungen zwischen Intermediär = Zwischenstadium und Intervall = Sicherheitsstadium definitiv ein Ende macht.

Das Bestreben für korrespondierende Begriffe die entsprechenden Bezeichnungen beizubehalten ist auch der Grund gewesen, weswegen ich in meiner Einteilung die sonst übliche Bezeichnung der Peritonitis nicht gebraucht habe, gleichwie Rotter bereits 1896 in seiner Einteilung der Perityphlitis circumscripta die P. diffusa als den korrespondierenden Ausdruck gegenübergestellt hat. Ich glaube, daß sich dieses aus Gründen der Logik und der Einfachheit empfiehlt. Jede über den Appendix hinausgeschrittene Entzündung ist eben eine Peritonitis, ganz gleich ob sie umgrenzt oder nicht umgrenzt ist. Für die umgrenzte Entzündung hat man der Ätiologie Rechnung getragen, indem man dafür ausdrücklich den korrespondierenden Ausdruck der Periappendicitis circumscripta geprägt hat. Für die nicht umgrenzte Entzündung geht man wenig konsequent von dem eben geprägten Ausdruck wieder ab, und bringt die Ätiologie bzw. die primäre Erkrankung im Adjektivum zum Ausdruck, um eine vorherrschende Krankheitserscheinung sekundärer Natur durch den Gebrauch des Substantivs für dieselbe plötzlich ganz besonders zum Ausdruck zu bringen.

Das wäre berechtigt, wenn der erstere Ausdruck weniger klar wäre. Das ist aber nicht der Fall. Man kann unter einer diffusen oder progredienten Periappendizitis unmöglich etwas anderes verstehen, als eine Peritonitis appendicularis diffusa oder progrediens, nur muß man sich im letzteren Fall zur Bezeichnung desselben Begriffes eines weiteren Wortes bedienen. Sowohl der Logik, wie der Einfachheit wegen verdient aber meines Erachtens in solchen Fällen der kürzere und korrespondierende Ausdruck zur Bezeichnung artgleicher Begriffe stets den Vorzug.

Sprengel hält dem entgegen, er vermöge nicht einzusehen, warum man einer Peritonitis, die vom Appendix ausgeht, nicht einen selbständigen Namen geben soll, wenn man andererseits der Peritonitis nach Magen- und Darmperforationen doch auch ihren richtigen Namen

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gibt. Das wäre berechtigt, wenn man die Peritonitis als selbständige Erkrankungsform hinstellen will. Letzteres geht aber wohl nicht an, da sie tatsächlich in einem, wie im anderen Fall den mehr oder weniger ausgeprägten Folgezustand einer Erkrankung des Appendix, des Magens oder sonst eines Organs der Abdominalhöhle darstellt. Jedenfalls ist bisher eine idiopathische Peritonitis von keiner Seite nachgewiesen worden. In beiden Fällen scheint mir daher eine besondere Berechtigung nicht vorzuliegen, die primäre Erkrankung plötzlich durch einen Folgezustand derselben zum Ausdruck zu bringen.

Die Art dieser Bezeichnung stammt wohl noch aus der Zeit, als man zunächst nur mit der Peritonitis, als dem äußerlich manifesten Symptom der Erkrankung zu rechnen pflegte und mangels genügender ätiologischer Klarstellung in vielen Fällen keine andere Wahl übrigblieb. Nachdem sich dieses geändert hat, und man in jedem Fall die Peritonitis als Folgeerscheinung der Erkrankung eines anderen Organs betrachten muß, scheint mir die Beibehaltung dieses Gebrauchs für die Erkrankung anderer Organe der Bauchhöhle ein gleich konservatives Verhalten gegenüber der im Vordergrund des Interesses stehenden Appendizitis nicht mehr zu rechtfertigen. Einen Beweis dafür darf man wohl in der ausdrücklich geprägten und gebrauchten Periappendicitis circumscripta erblicken, welche logischerweise auch eine Periappendicitis diffusa zur Voraussetzung haben muß.

Gehen wir nun zu der Ätiologie und Genese der Appendizitis über, so finden wir, daß einzelne früher mehr weniger willkürlich angenommene Faktoren allmählich völlig ausgeschieden sind. So darf man auf Grund der Ausführungen von Sahli und anderer Autoren die Theorie von der Typhlitis und Perityphlitis stercoralis heute mit Recht als der Vergangenheit angehörig betrachten.

Ebensowenig haben sich für den Zusammenhang des häufigeren Konstatierens der Appendizitis mit dem vermehrten Gebrauch emaillierter Kochgeschirre, von denen sich abgesprungene Teile im Appendix festsetzen sollten, irgendwelche Anhaltspunkte ergeben. Vielmehr dürfte sich das anscheinend vermehrte Auftreten der Appendizitis erheblich zwangloser erklären lassen durch die wesentlichen Fortschritte auf diagnostischem Gebiet einerseits und die erhöhte Aufmerksamkeit, welche seitens der praktischen Ärzte dieser Erkrankung zugewendet wird, andererseits. Jeder erfahrenere Arzt wird bei Erkrankungen des Intestinaltraktus stets die Appendizitis in den Bereich seiner Erwägungen mit einbeziehen und wird so dieselbe vielfach bereits in ihren ersten Anfängen zu konstatieren in der Lage sein, Fälle, die früher als Darm- und Magenkatarrhe, Darmkoliken

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oder generell als Bauchfellentzündungen angesprochen wurden und so der Statistik völlig entgangen sind.

Ferner hat man früher dem Vorhandensein von Fremdkörpern eine allzu große Wichtigkeit hinsichtlich der Ätiologie der Erkrankung beigemessen. Man hat wohl in einer Anzahl von Fällen Fremdkörper verschiedenster Art im Appendix gefunden, die allerdings die Veranlassung zu den weiteren pathologischen Erscheinungen bilden konnten und teilweise auch gebildet haben. Im Verhältnis zu der Gesamtzahl der Erkrankungen sind jedoch diese Fälle so selten, daß sie für die Ätiologie der Appendizitis nur eine ganz untergeordnete Bedeutung in Anspruch nehmen können.

Ebenso hat auch die Annahme einer zerstörenden Wirkung von Darmparasiten auf den Appendix nach Metschnikoff sich nicht als haltbar erwiesen. Sie werden vielmehr in der Regel auf den Processus die gleiche Wirkung wie jeder andere Fremdkörper ausüben durch gelegentliche Okklusion seines Lumens mit nachfolgender Retention und den entsprechenden Folgeerscheinungen. Eine spezielle Wirkung derselben kann nur unter der Voraussetzung in Frage kommen, daß sie imstande sind, kleine Kontinuitätstrennungen des Epithelbelages der Schleimhaut zu setzen, welche zur Eingangspforte für eine bakterielle Invasion werden könnten. Diese Voraussetzung wird für vereinzelte Arten von Darmparasiten zwar zugegeben, aber auch bei diesen nur in äußerst seltenen Fällen erfüllt sein.

Gänzlich haltlos, wenn auch nicht ohne Interesse ist die von Rubin aufgestellte Möglichkeit eines Zusammenhanges der Zurückhaltung von Winden mit pathologischen Veränderungen am Appendix.

Kommen wir nun zu den tatsächlich für Ätiologie der Appendizitis in Betracht kommenden Ursachen, welche heute allgemeine Anerkennung gefunden haben, so werden wir dieselben im Interesse einer besseren Übersicht am besten folgendermaßen einteilen:

1. Somatische Ursachen a) kongenitaler, b) erworbener Natur.

2. Ursachen, die durch das Lebensalter oder das Geschlecht der Patienten prädisponierend in Frage kommen.

3. Ursachen äußerer Natur bedingt durch a) Lebensweise und Ernährung, b) Klima, c) Traumen, d) bakterielle Invasion.

Bezüglich der somatischen Ursachen ist von Wichtigkeit die anatomische Beschaffenheit des Appendix und seines Mesenteriolums einerseits, sowie das Verhalten der umliegenden Organe andererseits. Namentlich bei dem ersten Punkte tritt die Möglichkeit einer mehr weniger hereditären Disposition zur Erkrankung in den Vordergrund. Es kann durch eine besondere Länge des Appendix, welche nach einwandfreien Beobachtungen auch erblich vorkommt, eine vermehrte

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Gelegenheit zu Knickungen des Lumens und Retention des Inhalts gegeben werden, namentlich wenn damit noch ein kurzes Mesenteriolum verbunden ist, das an sich schon eine gewisse Knickung des Appendix bedingen kann.

Neben einer abnorm geringen Widerstandsfähigkeit der Wandung überhaupt kann nach Hansemann auch eine herabgesetzte Widerstandsfähigkeit der Gerlachschen Klappe von besonderer Bedeutung für die Zirkulation bzw. Stauung von Darminhalt im Processus mit ihren Folgezuständen sein. Diese verminderte Widerstandsfähigkeit kann in beiden Fällen sowohl kongenitaler, wie erworbener Natur sein. Kongenital kann es ferner durch intrauterine Entzündungsvorgänge zu einer Verwachsung des Testikels mit dem Appendix kommen, wobei der Testikel ihn bei seinem Deszensus mit hinunternimmt, oder es kann nachträglich der Appendix mit dem Cöcum in den offen gebliebenen Processus vaginalis hinabgleiten und hier einer Abklemmung oder Einschnürung unterliegen, die eine Appendizitis im Bruchsack zur Folge hat. Daß diese Fälle nicht ganz selten sind, beweist die Tatsache, daß in der Albertschen Klinik unter 250 Bruchoperationen der Appendix 9mal (= 3,6%) im Bruchsack gefunden wurde. Dagegen fand Maydl unter 443 Hernien nur 11mal das Cöcum mit oder ohne Appendix im Bruchsack = 2,5% der Fälle.

Desgleichen kann als Folge intrauteriner Entzündungen eine besondere Kürze und Abknickung des Mesenteriolums zurückbleiben (Sprengel), auf deren Folgen oben bereits hingewiesen ist.

Bezüglich der Einwirkung anderer Organe auf den Appendix hat Karl Beck 1898 auf das Verhalten der Wanderniere hingewiesen. Er beobachtete nämlich 3 Fälle, in denen eine rechtsseitige Wanderniere den nach hinten gerichteten Appendix gegen das Darmbein drückte und so nach seiner Meinung zu einer Verlegung des Lumens und Zirkulationsstörung die Veranlassung abgab. Auch Edebohls weist auf die Bedeutung der rechtsseitigen Wanderniere für die Entstehungsmöglichkeit einer Appendizitis hin, und zwar soll dieselbe durch Druck auf die Ven. mes. sup. und sekundäre Stauung im Appendix zustande kommen. Sprengel, gleichwie Fueth und Hadra, kann sich diesen letzteren Gründen von Edebohls nicht anschließen, namentlich da Hyperämie heute noch weniger, als früher zu den entscheidenden Bedingungen der Entzündung gehört. Letzteres dürfte jedoch nur für die warme Hyperämie zutreffen. Sobald es auf Grund der obigen Vorgänge zu einer kalten Hyperämie wirklich kommen sollte, dürften die Vorbedingungen für Ernährungsstörungen der Wand und sekundäres Vordringen der Entzündungserreger durchaus gegeben sein. Die Richtigkeit der Annahme von Edebohls dürfte also von

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dem Grade der Stauung abhängig zu machen sein, welche eine rechtsseitige Wanderniere auf den Appendix ausüben könnte. Daß dieser so hoch sein sollte, ist allerdings kaum anzunehmen und zum mindesten bisher nicht bewiesen.

Von erworbenen anatomischen Ursachen werden vor allem Kotsteine im Lumen, sowie Narbenstrikturen, Knickungen und Verwachsungen als Residuen früherer Entzündungen, sei es am Appendix selbst, sei es in seiner Umgebung, den Anlaß zu den folgenden und meist schwerer verlaufenden Anfällen bilden können. Ausnahmsweise kann auch eine Torsion des Appendix um seine Längsachse, sowie der Druck von Tumoren anliegender Organe die Veranlassung zu einer Störung der Blut- oder Kotzirkulation im Appendix abgeben.

Auch dem Lebensalter kann eine gewisse Wichtigkeit für die Prädisposition zur Appendizitis nicht abgesprochen werden. So soll einerseits nach den Untersuchungen von Monti und Sonnenburg bei Kindern in den ersten Lebensjahren der Appendix eine besondere Neigung zu Knickungen haben, die dann zu den entsprechenden Folgeerscheinungen führen können. Andererseits soll nach Mynter auch durch ein überwiegendes Vorhandensein von Lymphgewebe im Appendix während des jugendlichen Alters eine größere Neigung zur Erkrankung gegeben sein. Trotzdem erkranken jedoch nach Hansemann Kinder vor dem dritten Lebensjahr relativ selten an Appendizitis, da die Gerlachsche Klappe in diesem Alter noch nicht genügend ausgebildet ist. Der Zusammenhang des Appendix mit dem Cöcum besteht dann in einer breiten, mehr trichterförmigen Kommunikationsöffnung, so daß es seltner zu Stauungen des Inhalts im Appendix kommen kann.

Ebenso ist auch im höheren Lebensalter die Disposition zur Erkrankung eine erheblich geringere, da dann ein starker Schwund der lymphatischen Gewebsbestandteile der Wandung des Appendix einzutreten pflegt. Gleichzeitig wird erfahrungsgemäß die Disposition zur Bildung von Kotsteinen im höheren Lebensalter eine wesentlich geringere. So hat Ribbert bei Kindern unter 5 Jahren keinen Kotstein und im Alter von 5—20 Jahren doppelt so häufig Kotsteine gefunden, als nach dieser Zeit. Tatsächlich ist auch nach den übereinstimmenden Berichten sämtlicher Autoren das häufigste Auftreten der Erkrankung in das Alter von 10—30 Jahren zu verlegen.

Daß auch das Geschlecht von Wichtigkeit für Ätiologie der Appendizitis ist, beweist die empirisch erwiesene Tatsache, daß die Erkrankung bei dem männlichen Geschlecht erheblich häufiger vorkommt, als bei dem weiblichen. Diese Beobachtung findet ihre Erklärung nach der Mehrzahl der Autoren in der besseren Blutversorgung des

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Appendix durch das Lig. appendico-ovaricum bei dem weiblichen Geschlecht, wobei allerdings zu bedenken ist, daß z. Z. nach Sprengel die Existenz des Lig. appendico-ovaricum noch keineswegs unbestritten dasteht. Zum mindesten sind über die Häufigkeit seines Vorhandenseins sichere Angaben bisher nicht gemacht worden. Zwar erscheint das weibliche Geschlecht während der Zeit der Menses besonders für eine Erkrankung des Appendix disponiert wegen der stärkeren Blutstauung und Schwellung der Genitalorgane in dieser Zeit, an welcher der Appendix naturgemäß einen gewissen Anteil hat durch seine Gefäßverbindungen im Lig. appendico-ovaricum. Davon kann jedoch für die Appendizitis nur eine stärkere Schwellung und dadurch bedingte Verlegung des Appendixlumens in Betracht kommen, da nach den neueren Anschauungen in der vermehrten Blutzufuhr zum Appendix innerhalb dieser Zeit, soweit sie zu einer warmen Stauung führt, eher ein entzündungsverringernder, als befördernder Faktor zu erblicken wäre. Ob und wie häufig aber die Schwellung des Appendix auf derartiger Grundlage eine so hochgradige werden kann, wird von entsprechenden Untersuchungen abhängig zu machen sein, die z. Z. noch ausstehen.

Gegenüber dieser ganz kurzen Zeit einer vielleicht etwas vermehrten Disposition beim weiblichen Geschlecht ist das männliche Geschlecht durch sein ganzes äußeres Berufsleben in einem viel höheren Grade Traumen und Infektionsmöglichkeiten ausgesetzt und hat daher unter viel zahlreicheren Gelegenheitsursachen zu leiden, als das weibliche Geschlecht. Dazu kommt noch, daß Männer durch kaltes Trinken, übermäßiges Rauchen u. dgl. viel eher zu vorübergehenden oder chronischen Darmkatarrhen die Veranlassung geben, durch welche dann eine verminderte Widerstandsfähigkeit und erhöhte Infektionsmöglichkeit der Darmwandung bedingt wird, als deren Locus minoris resistentiae der Appendix ja ohnedies zu betrachten ist.

Von äußeren Einwirkungen auf den Appendix werden vor allem Lebensweise, Ernährung, Klima, Traumen und Infektionen des Körpers allgemeiner Natur in Frage kommen.

So ist als ein wichtiges Moment für die Ätiologie allgemein anerkannt die übertriebene bzw. sehr reichliche Fleischnahrung, wie sie namentlich in Amerika, England und Norddeutschland genossen wird, im Gegensatz zu den Japanern und verschiedenen Eingeborenenstämmen Afrikas, denen die Fleischnahrung durch Religionskult oder örtliche Verhältnisse mehr weniger entzogen ist. Die Seltenheit bzw. das gänzliche Fehlen der Appendizitis bei diesen Volksstämmen steht im strikten Gegensatz zu der Häufigkeit der Erkrankung bei den ersteren (Kümmel, Lange, Beck u. a.). Auf Grund dieser Beobach-

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tung ist man wohl früher zu der Annahme einer Prädisposition gewisser Nationen für die Appendizitis gelangt.

Bezüglich des Klimas ist es gleichfalls erwiesen, daß der Aufenthalt in den Tropen entsprechend der größeren Häufigkeit von Darmerkrankungen bei Europäern überhaupt auch eine entsprechende Vermehrung der Krankheitsfälle an Appendizitis zur Folge hat. Ebenso können die Jahreszeiten insofern von gewissem Einfluß sein, als in den Sommermonaten eine vermehrte Gelegenheit zu Magen- und Darmstörungen gegeben ist.

Auch Traumen verschiedenster Art können, sei es als auslösende Ursache einer latent bestehenden Reizung, sei es als primäre Ursache überhaupt den Anlaß zu einer Appendizitis abgeben. So weisen unter anderen Nimier und Sachs auf Fälle von körperlichen Überanstrengungen hin, die eine Appendizitis zur Folge hatten. Ebenso vertritt auch Neumann in einer Arbeit an der Bachmannschen Klinik die Möglichkeit, daß am normalen Appendix eine Erkrankung auf traumatischer Grundlage entstehen könne, und stellt selbst 10 Fälle dieser Art zusammen. Nun dürfte es ja einerseits schwer sein mit Sicherheit zu beweisen, daß in solchen Fällen tatsächlich ein normaler Appendix vor dem Trauma vorgelegen habe. Spricht doch das Fehlen subjektiver Beschwerden bekanntlich nicht sicher gegen das Vorhandensein einer reinen Appendizitis, die noch nicht durch eine stärkere Stauung im Appendix oder durch Übergreifen des Entzündungsprozesses auf die Serosa zu Reizerscheinungen des Peritoneums geführt hat. Andererseits kann aber ebensowenig ein zwingender Beweis gegen die Möglichkeit angeführt werden, daß es unter besonderen Umständen, bei besonders dünnen und weiten Bauchdecken, wie z. B. bei Frauen mit Rektusdiastase u. a., gelegentlich durch ein Trauma zu Quetschungen der Wandung des Appendix kommen kann, welche zur Schädigung der Gewebsvitalität bzw. Blutungen und Kontinuitätstrennungen der Schleimhaut führen und so eine mehr weniger große Eingangspforte für eine bakterielle Invasion schaffen können.

Allerdings kann man mit Neumann und Hoffmann annehmen, daß in der Regel dazu ein Kotstein erforderlich sein wird. Wenn es dabei nach Neumann auch durch die Mitwirkung der Bauchpresse allein zu Zerrungen an der Stelle des Kotsteines kommen soll, die eine nachfolgende Läsion und Infektion der Schleimhaut bedingen können, so kann man dieser Möglichkeit wohl beistimmen, aus Gründen, die ich inzwischen in einem Gutachten näher entwickelt habe, welches ich über einen derartigen Fall aus der Prof. Lexerschen Privatklinik abgegeben und in der Berliner klinischen Wochenschrift publiziert habe. Sprengel hält es überhaupt für ausgeschlossen, daß die

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mechanische Quetschung eines Kotsteines gegen die Wandung zu Appendizitis führen könne oder daß ein mit infektiösem Material gefüllter Appendix durch einen Stoß gegen die Bauchwand oder die Bauchpresse allein zum Bersten gebracht werden könne. Diese Annahme Sprenglers scheint doch etwas zu weit zu gehen. Bei normalen Bauchdecken dürfte allerdings für einen völlig gesunden Appendix ohne Kotstein ein Trauma ziemlich bedeutungslos sein, was auch der Anschauung von Sonnenburg entspricht. Bei dem Zusammenfallen einer bereits primär am Appendix vorhandenen pathologischen Veränderung oder eines Kotsteines im normalen Appendix mit einem Trauma kann letzteres doch als auslösende Ursache eines nachfolgenden Anfalles sehr wohl in Betracht kommen. Daß ein Trauma gewissermaßen indirekt schließlich auch auf einen normalen Wurmfortsatz ohne Kotstein einwirken kann, scheint mir ein von Fink veröffentlichter Fall zu beweisen, in dem sich 2 Stunden nach einem Fall auf den Bauch ein typischer Appendizitisanfall ohne jegliche vorausgegangene Krankheitserscheinungen entwickelte. Nach einem zweiten Appendizitisanfall wurde die Operation vorgenommen und ergab eine Stildrehung des langgestielten Wurmfortsatzes mit nachfolgender Behinderung des Sekretabflusses und den entsprechenden Folgeerscheinungen. Man dürfte nicht fehlgehen, wenn man diese Stieldrehung in Anbetracht des Fehlens vorausgegangener Erscheinungen und des zeitlichen Zusammenhanges der Erkrankung mit dem Unfall in ursächlichen Zusammenhang mit dem vorausgegangenen Unfall bringt.

Das wichtigste Moment für die Ätiologie der Appendizitis, das heute als grundlegend für die Genese dieser Erkrankung in ihren verschiedenen Erscheinungsformen allgemeine Anerkennung gefunden hat, ist das der bakteriellen Infektion. Tatsächlich hat man den Appendix sowohl seiner Gestalt, wie seinem makroskopischen und histologischen Bau nach als einen wahren Prädilektionsort für die Ansiedlung einer jeden Infektion im Digestionstraktus anzusehen. Und zwar wird in der Regel die Infektion von dem Darmlumen ihren Ausgang nehmen. Wenn auch ausnahmsweise wirklich einmal eine Infektion auf dem Blutwege zustande kommen könnte, wie es nach den Versuchen von Josuè an Kaninchen den Anschein hat, so ist diesem Infektionsmodus für den Menschen zum mindesten eine sehr geringe Bedeutung beizumessen. Ob die Erreger im übrigen auf endogenem Wege auch die unverletzte Schleimhaut des Wurmfortsatzes zu passieren vermögen, wird an mancher Stelle angezweifelt und ist zum mindesten bis heute noch nicht einwandfrei bewiesen. Andererseits steht aber auch nichts der Annahme im Wege, daß bei Herabsetzung

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der Widerstandskraft des Körpers und der allgemeinen oder örtlichen, natürlichen Gewebsvitalität durch die Aufnahme von Toxinen in das Blut in gleicher Weise eine Einwanderung von Bakterien durch das unverletzte Epithel stattfinden kann, wie andererseits die nachgewiesene Auswanderung der Leukozyten ebenfalls durch das unverletzte Epithel tatsächlich stattfindet.

Für die besondere Prädisposition des Appendix zur Infektion vom anatomischen Standpunkt aus genügt es auf den Reichtum an Lymphknötchen bzw. lymphadenoidem Gewebe der Wandung hinzuweisen, das nach Mynter besonders im jugendlichen Appendix hervortritt und diesen somit noch stärker zur infektiösen Erkrankung disponiert. Dieser Reichtum des Organs an lymphadenoidem Gewebe ist ja auch für Sahli der Grund gewesen, es in Parallele mit den Gaumentonsillen zu stellen, indem es für die häufigen Erkrankungen des Digestionstraktus die gleiche Rolle spielt, wie die Rachen- bzw. Gaumentonsillen für diejenige des Pharynx. In Analogie dazu hat Sahli die Bezeichnung der Angina des Wurmfortsatzes, der Angina appendicularis, vorgeschlagen, welche recht geeignet ist, die Ähnlichkeit der Vorgänge zum Ausdruck zu bringen. Daß damit auch ein ganz besonderer ätiologischer Zusammenhang zwischen Angina tonsillaris und Appendizitis ausgedrückt werden soll, muß ausdrücklich verneint werden. Auch Sprengel kann nach seinen Erfahrungen dem ätiologischen Zusammenhang zwischen Angina tonsillaris und Appendizitis eine besondere Bedeutung nicht beimessen, da er diese beiden Erkrankungen nicht häufiger nebeneinander gefunden hat, als andere Erkrankungsformen auch. Sie hat eben den gleichen Einfluß auf den Appendix, wie ihn andere Infektionskrankheiten schließlich auch haben können. —

Verfolgen wir nun den Vorgang der Genese der bakteriellen Appendizitis weiter, so kommt es nach Aschoff im mittleren Alter durch Verminderung des Drüsenreichtums an zirkumskripten Stellen zum Vorhandensein von Lakunen, so daß auf diese Weise die Epithelschicht durch eine Lücke in der Muscularis mucosae direkt mit der Submukosa in Verbindung treten kann. In diesen Lakunen kann dann um so leichter eine Retention von Sekret und Infektionserregern stattfinden, die weiter zu einer Druckarrosion des Epithels mit nachfolgender Infektion der Wandung des Processus führen kann, gänzlich analog den Vorgängen bei der Angina lacunaris.

Was nun die Erreger selbst anbelangt, so wird es sich ja bei dieser Infektion in der Regel um Bacterium coli commune, meist in Verbindung mit Staphylo- und Streptokokken handeln. Außer diesen ist es aber Tavel und Lanz gelungen, noch eine große Reihe anderer

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Bakterien im Appendix nachzuweisen, als da sind: B. capsulatus, B. fusiformis, B. foetidus liquefaciens, B. pyocyaneus, Rotz-, Tetanus und tetanusähnliche Bazillen, Staphylococcus aureus, Coccus conglomeratus, Diplococcus intestinalis maj. und min., Spirillen, ferner Typhus und Aktinomyceserreger.

Auffallenderweise haben dieselben Autoren gerade in normalen Appendizes mehrere Bakterienspezies zugleich (bis zu 6) gefunden, während unter den pathologisch veränderten Appendizes 10% steril gefunden wurden. Je weiter die pathologischen Veränderungen der Appendizes vorgeschritten waren, desto geringer war die Zahl der verschiedenen Bakterienarten, welche sich darin feststellen ließen. Bei den kalten appendizitischen Abszessen fand man sogar in 75% der Fälle völlige Sterilität, eine Tatsache, welche die Verfasser durch eine vermehrte Leukozytenansammlung und daraus resultierende verstärkte Phagozytose während der Entzündung zu erklären versuchen. Es erscheint nicht unberechtigt, diese Vorgänge in eine gewisse Analogie mit der Theorie über die Heilungsvorgänge bei der Bierschen Stauung zu setzen.

Als weitere Faktoren für die erhöhte Infektionsmöglichkeit der Wandung des Processus vermiformis kommen einerseits die ungeheure Menge von Bakterien im Verhältnis zu dem geringen Lumen, dem geringen Querschnitt der Wand und der geringen Flächenausdehnung der Schleimhaut des Processus und andererseits die vielfach durch Stauungen des Inhaltes gesteigerte Virulenz der Erreger in Betracht. Diese Verhältnisse in Verbindung mit der lymphadenoiden Beschaffenheit des histologischen Baues der Wandung des Processus sind wohlgeeignet, denselben zu einem Locus minoris resistentiae des gesamten Digestionstraktus zu machen, der besonders leicht auf eine Infektion der Verdauungswege reagieren muß.

Diesen unverhältnismäßig reichlichen und virulenten Offensivfaktoren steht ferner noch gegenüber die relativ leicht herabsetzbare Defensivkraft der Wandung infolge der einseitigen Blutversorgung des Appendix, die ja dem Endbezirk der im Mesenteriolum verlaufenden Arterie entspricht. Es kann dadurch sehr leicht zu Ernährungsstörungen der Wand kommen, sobald ein genügender Zufluß oder Abfluß des Blutes durch pathologische Gefäßveränderungen primärer oder sekundärer Natur in Frage gestellt wird, insbesondere ist auch an die schlechte Gefäßversorgung der Spitze des Appendix bei primärer Kürze des Mesenteriolums zu denken, auf die Karewski hingewiesen hat.

Ob diese Gefäßveränderungen nun primärer oder sekundärer Natur sind, kommt für die verderblichen Folgen derselben wenig in Betracht

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und wird auch nicht immer leicht zu entscheiden sein. So hält v. Brunn die sekundäre Entstehung der Gefäßerkrankungen für wahrscheinlicher. Er gelangt zu dieser Anschauung durch einen Vergleich der Art, des Sitzes und der Ausdehnung der Gefäßerkrankungen mit den pathologischen Veränderungen an der Wandung des Processus. Allerdings kann für einzelne Fälle die Möglichkeit zugegeben werden, daß die Gefäßveränderungen als die primäre Affektion die Veranlaßung für weitere und stärkere Entzündungsvorgänge abgegeben hätten. Jedoch ist die direkte Notwendigkeit eines solchen Zusammenhanges nirgends ersichtlich.

Ebenso hält O. Nordmann die Thrombose der Mesenterialvenen stets für eine sekundäre Erkrankung, der primär eine Verengerung bzw. Stauung im Processus durch Veränderungen seines Lumens vorausgegangen ist.

Als sicher sekundärer Natur spricht v. Brunn die Veränderungen an den Lymphgefäßen an, an denen er eine Erweiterung, Anfüllung mit mono- und polynukleären Leukozyten, Lymphangitis und Thrombose in akuten Fällen, sowie Intimaverdickungen und Obliteration in abgelaufenen Fällen beobachten konnte.

Auch Karewski hält die primären Zirkulationsstörungen und embolischen Vorgänge für selten.

Kehren wir nach dieser kurzen Abschweifung zu der bakteriellen Invasion des Appendix zurück, so muß in der Regel für dieselbe noch ein rein mechanisches Moment dazukommen. Dieses wird fast stets durch Verengerung bzw. Verschluß des Lumen infolge Knickung, Narbenstriktur oder Kotsteine geboten, welche eine Retention und Stauung des Inhaltes mit Steigerung der Virulenz und vermehrter Resorption desselben durch die Lymphgefäße zur Folge haben kann. Insbesondere können nach Roux und Trachenberg die Kotsteine durch eine Art kugelventilähnlicher Wirkung die schnelle Entstehung einer Stauung begünstigen.

Auf diese Weise können dann infolge starker Dehnung der Wand einerseits kleine Dehnungsgeschwüre im Sinne von Kocher und Prutz znstande kommen. Andererseits ist es zum mindesten nicht ausgeschlossen, daß es infolge dauernden Druckes eines Kotsteines oder Fremdkörpers auf eine Stelle der Wand zu einer Zirkulationsstörung der Wand und oberflächlichen Erosion der geschwollenen Schleimhaut im Sinne der Dekubitalgeschwüre von Nothnagel kommen kann. Letzteres kann jedoch nur eintreten, wenn eine Einklemmung des Kotsteins bzw. ein Erlöschen der Peristaltik des Processus eine Verlagerung des Kotsteins nicht mehr zustande kommen läßt. Die Mehrzahl der Autoren neigt zu der Anschauung, daß die größere

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Gefährlichkeit der Kotsteine weniger in ihren mechanischen Eigenschaften, denn in ihrer Einwirkung als konzentrierte Bakterienanhäufungen zu erblicken ist. Diese können naturgemäß schon auf dem Lymphwege allein zu einer Infektion des Peritoneums und den entsprechenden Folgeerscheinungen führen. Gehen wir bei dieser Gelegenheit etwas näher auf die Bedeutung ein, welche den Kotsteinen für die Ätiologie und Genese der Appendizitis beizumessen ist, so finden wir in der letzten Zeit mehr und mehr die Tendenz, den ursächlichen Zusammenhang zwischen Kotsteinen und Appendizitis erheblich einzuschränken, wenn nicht gänzlich zu negieren, namentlich soweit das rein mechanische Moment in Betracht kommt.

Die hauptsächlichsten Vertreter dieser Anschauung haben wir vornehmlich in Aschoff und Sprengel zu erblicken. Beide sind geneigt dem Vorhandensein eines Kotsteines einen größeren Einfluß auf den Verlauf, als auf die Entstehung der Erkrankung zuzuschreiben. Beide gelangen auf Grund ihres Untersuchungsergebnisses, das Aschoff an 130 exstirpierten Wurmfortsätzen des Rotterschen Materials und Sprengel an seinem eigenen großen klinischen Material erhoben hat, zu der Ansicht, daß eine rein mechanische Einwirkung eines Kotsteines im Sinne einer Appendizitis ohne gleichzeitige bzw. vorausgegangene Mitwirkung einer Infektion von der Hand zu weisen sei. Wieweit aber bei der gegebenen Möglichkeit einer Infektion im Darmtraktus der Stein eine solche erleichtern bzw. begünstigen kann, scheint mir eine Frage zu sein, die durch die Darlegungen Aschoffs und Sprengels nicht genügend klargelegt wird, zum mindesten nicht in negativem Sinne entschieden werden kann. Wenn vielmehr beide die Bedeutung des Kotsteines ohne gleichzeitige Infektionsmöglichkeit gänzlich ableugnen, so darf man dem wohl ohne weiteres zustimmen. Wenn sie hingegen bei gleichzeitiger Infektionsmöglichkeit die Infektion als den primären Vorgang unabhängig vom Kotstein und die Bedeutung des Kotsteines erst in sekundären Krankheitsvorgängen annehmen zu müssen glauben, so darf man hierin vielleicht doch eine etwas zu weit gehende Schlußfolgerung aus ihren Untersuchungsergebnissen erblicken, wie ich versuchen will im folgenden des näheren auszuführen.

Wenn Aschoff z. B. sagt, daß eine nicht bakterielle, durch einfache Einklemmung des Kotsteines hervorgerufene Reizung des Wurmfortsatzes äußerst selten, wenn überhaupt beweisbar ist, so ist dem durchaus beizupflichten, was die Schwierigkeit der Beweisführung für einen derartigen Vorgang anbelangt. Andererseits ist aber nicht recht einzusehen, warum es nicht ähnlich dem Hydrops der von Aschoff vergleichsweise herangezogenen Gallenblase auf Grund von Steinver-

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schluß gelegentlich auch zu einer Sekretverhaltung im Processus auf der gleichen Grundlage kommen kann, da doch fragelos ein vorhandener Kotstein durch dauernde Apposition zu einer Größe anwachsen kann, welche in einem absoluten Mißverhältnis zu der Größe und Dehnungsfähigkeit des Lumens des Processus steht, so daß eine Verengerung bzw. Verlegung des Lumens daraus resultieren kann. Wenn dagegen die dauernde Peristaltik des Wurmfortsatzes angeführt wird, so ist dazu zu bemerken, daß diese einmal individuell äußerst verschieden sein kann je nach Länge, Form, Lage und Haltung des Wurmfortsatzes und dem sonstigen Allgemeinzustand des Individuums und ferner, daß gerade diese infolge eines erhöhten Reizes durch den als Fremdkörper wirkenden Kotstein schließlich zu vermehrten und verstärkten Kontraktionen an der Stelle des Kotsteines gelangen und so schließlich selbst zu Einklemmung des Steines beitragen kann. Es liegt ferner auf der Hand, daß trotz einer gewissen Steigerungsfähigkeit die Leistungsfähigkeit der Wurmfortsatzperistaltik doch immerhin eine begrenzte ist und daß sie bei entsprechender Vergrößerung des Kotsteines an einer Grenze anlangen kann, wo sie eine Verschiebung und Verlagerung des Kotsteines nicht mehr zu bewirken vermag. Daß daraus dann ebenfalls Einklemmungserscheinungen resultieren können, ergibt sich von selbst.

Ohne eine derartige Annahme wäre es zum mindesten schwierig, die tatsächlich vorhandenen und palpatorisch auch durch entsprechendes Schmerzgefühl nachweisbaren Erscheinungen der Appendikularkoliken zu erklären, welche ja vielfach ohne Fieber und sonstige klinische Erscheinungen verlaufen und wieder abklingen können, und welche somit vielfach das einzige objektive Merkmal vorübergehender Retentionserscheinungen im Appendix bilden.

Allerdings werden von einzelnen Autoren derartige Koliken auch auf rein entzündliche Vorgänge allein zurückgeführt. Bei der kurzen Dauer derselben von bisweilen nur 1—2 Stunden scheint mir aber eine derartige Erklärung für diese Fälle nicht auszureichen. Da naturgemäß die Patienten in einem derartigen Stadium schnell vorübergehender und fieberfreier Appendikularkoliken selten oder fast nie zur Operation gelangen, läßt sich zum mindesten ebensowenig gegen als für die Annahme einer derartigen Möglichkeit vorbringen, durch die aber andererseits allein die klinischen Erscheinungen eine genügende Erklärung finden dürften.

Daß damit in der Regel entzündliche Erscheinungen auf Grund eines früher oder später einsetzenden, leichteren oder schwereren Infektionsvorganges der Wandung Hand in Hand gehen, wird auch von den Vertretern einer größeren Bedeutung des Kotsteines für die

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Genese der Appendizitis ohne weiteres zugegeben, nur wird von denselben dem Vorhandensein bzw. der mechanischen und bakteriellen Einwirkung des Kotsteines auf den Processus durch primäre oder gleichzeitige Retention des Inhaltes und Dehnung der Wandung des Appendix eine gewisse Erleichterung des an sich vorauszusetzenden Infektionsvorganges zugeschrieben bzw. die Möglichkeit einer primären Beteiligung des Kotsteines bei dem Infektionsvorgange damit angenommen. Aschoff selbst gibt das in gewissem Sinne zu, indem er sagt, daß bei der Infektion eines einen Stein enthaltenden Wurmfortsatzes der Stein die Lokalisation der Entzündung an einer bestimmten Stelle begünstigt und damit dem schwereren Verlauf der Erkrankung bzw. einer verzögerten Heilung Vorschub leistet. Mithin erblickt doch Aschoff selbst ein prädisponierendes Moment in dem Vorhandensein eines Kotsteines im Falle einer gegebenen Infektionsmöglichkeit, das nach seiner Anschauung allerdings erst sekundär in Tätigkeit treten soll.

Auch Sprengel gelangt auf Grund seiner zahlreichen Operationsbefunde zu folgenden Schlußsätzen: 1. Ein Kotstein kann einen Anfall nicht herbeiführen, da er beliebig lange im Wurmfortsatz liegen kann, ohne daß ein Anfall erfolgt. 2. Ein Kotstein kann nicht durch einen einfachen Dekubitus zur Perforation führen, da Sprengel ihn vielfach frei schwimmend im gestauten Sekret gefunden hat. 3. Ein Kotstein kann eine Abflußbehinderung des Sekretes nicht bewirken, da Sprengel ihn nicht nur im proximalen Teil der Erweiterung des Lumens, sondern an beliebiger Stelle gefunden hat. Diesen Thesen ist, ohne eine gleichzeitig gegebene Infektionsmöglichkeit bzw. ohne eine bereits vorhandene Infektion des Appendix ohne weiteres zuzustimmen. Wie liegen die Verhältnisse aber im letzteren Falle, mit dem man es doch in der Regel zu tun hat? Da gibt Sprengel gleich Aschoff zu, daß der an sich vom Kotstein unabhängig entstehende Infektionsprozeß in der Nähe des Kotsteines intensiver verläuft infolge eines verstärkten Reizes durch den letzteren, und daß es auf diese Weise eher zur Verschwellung des Lumens an dieser Stelle mit entsprechenden Folgeerscheinungen kommen kann. Daß im übrigen der Kotstein eine positive Bedeutung für die Entwicklung der Appendizitis haben muß, beweist für ihn auch die Tatsache, daß Ribbert bei Appendizitis 5mal soviel Kotsteine gefunden hat, als bei nicht an Appendizitis erkrankten Personen, eine Tatsache, die in gleicher Weise auch für die häufigere und leichtere Entstehung einer Appendizitis auf der Grundlage eines vorhandenen Kotsteines sprechen könnte.

Meines Erachtens läßt sich nun aus den persönlichen Beobachtungen

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und Untersuchungsergebnissen der beiden Autoren etwas Positives weder für noch gegen ihre Anschauung anführen, solange man nicht in der Lage ist im einzelnen Falle nachzuweisen, ob der Kotstein tatsächlich primär vorhanden war oder erst sekundär entstanden ist. Dieses dürfte jedoch im allgemeinen ziemlich schwierig, wenn nicht unmöglich sein.

Wenn Sonnenburg hierzu ganz allgemein den Grundsatz aufstellt, daß jeder Kotstein als ein Zeichen voraufgegangener Entzündung anzusehen und somit als sekundäre Krankheitserscheinung aufzufassen ist, so dürfte er sich damit weder mit der Mehrzahl der Autoren im Einklang befinden, noch einen einwandsfreien Beweis für diese Anschauung zu erbringen in der Lage sein.

Vielmehr wird fast allgemein die Möglichkeit des primären Vorhandenseins eines Kotsteines in dem an sich normalen bzw. unveränderten Wurmfortsatz als Folge verminderter Peristaltik des Darmes im allgemeinen und des Appendix im besonderen bei chronischer Obstipation usw. ohne weiteres zugegeben. Andererseits liegt es auf der Hand, daß bei einem katarrhalisch bzw. entzündlich veränderten Wurmfortsatz die Bedingungen für die Entstehung eines Kotsteines ungleich günstigere sind und mit entsprechender Einschränkung die Sonnenburgsche Anschauung für eine große Anzahl von Fällen zu Recht besteht.

Beweisend wären demnach die Fälle Sprengels für seine Anschauung von der primären Bedeutungslosigkeit des Kotsteines hinsichtlich der Ätiologie und Genese der Appendizitis erst dann, wenn bei denselben die sekundäre Entstehung des Kotsteines nach einem voraufgegangenen Anfall mit Sicherheit auszuschließen wäre, da in letzterem Falle das Verhalten bzw. die Lage des Kotsteines im Appendix nach ganz anderen Gesichtspunkten zu beurteilen wäre.

Des ferneren will mir die Tatsache, daß ein Kotstein beliebig lange im Appendix liegen kann, ohne daß ein Anfall erfolgt, nicht recht als Beweis dagegen einleuchten, daß im Fall einer gegebenen Infektionsmöglichkeit der Infektionsvorgang und somit die Entstehung des Anfalls dadurch mehr oder weniger erleichtert und begünstigt werden kann. Man braucht eben nur anzunehmen, daß in den Fällen, in denen ein Kotstein beliebig lange gelegen hat, ohne daß ein Anfall erfolgte, eine entsprechend erhöhte Infektionsmöglichkeit ausgeblieben ist. Beweiskräftig wären solche Fälle erst dann, wenn bei allen die gleiche Infektionsmöglichkeit seitens des Darmtraktus sei es durch Darmkatarrhe, erhöhte Virulenz der Darmbakterien u. dgl. gegeben wäre, bzw. wenn sämtliche Fälle unter den gleichen Bedingungen gestanden hätten. Solange etwas derartiges aber nicht experi-

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mentell hergestellt und bewiesen wird, sind doch wohl klinisch die Bedingungen der einzelnen Fälle zu verschieden, als daß allein das Vorhandensein eines Kotsteines und das Ausbleiben eines Anfalles trotz desselben in einer Anzahl von Fällen für die Bedeutungslosigkeit des Kotsteines hinsichtlich der Erleichterung eines Infektionsvorganges am Appendix als beweiskräftig zu betrachten wäre.

Ebensowenig will mir die Tatsache, daß Sprengel in einer Anzahl von Fällen den Kotstein freischwimmend in dem retinierten Sekret gefunden hat, beweiskräftig dafür erscheinen, daß nicht in anderen Fällen nach eventuell vorausgegangener Infektion und Schwellung der Wand der eingeklemmte Kotstein in seiner gleichzeitigen Eigenschaft als Fremdkörper und konzentrierte Bakterienanhäufung durch seine intensive und dauernde Berührung mit der Wand, welche ihrerseits durch Kontraktionen der Muskelschicht eine gewisse Reibung der Schleimhaut auf dem Stein erzeugen kann, eine erleichterte bzw. vergrößerte Möglichkeit des Eindringens der Infektionserreger gerade an dieser Stelle der Wand gewähren kann, wodurch dann der Bildung einer Ulzeration mit nachfolgender Perforationsmöglichkeit ein erheblicher Vorschub geleistet wird. Man kann eben ohne weiteres die rein mechanische Entstehung eines Dekubitus mit eventueller Perforation auf der Grundlage eines Kotsteines von der Hand weisen, ohne deswegen doch jegliche mechanische und bakterielle Mitwirkung des Kotsteines bei der Entstehung eines intensiveren lokalen Infektionsvorganges ableugnen zu müssen, der zu einer Perforation mittelbar auf der Grundlage eben dieses Kotsteines wohl führen kann. Die Fälle Sprengels beweisen demnach einwandsfrei, daß bei ihnen selbst und damit sehr häufig ein derartiger Vorgang wohl auszuschließen ist. Daraus aber die Unmöglichkeit eines derartigen Vorganges für sämtliche Fälle überhaupt herleiten zu wollen, scheint mir eine etwas zuweitgehende Schlußfolgerung zu sein.

Was nun schließlich die Verneinung der Möglichkeit einer Behinderung des Sekretabflusses im Appendix durch einen Kotstein anbelangt, so scheint mir die Tatsache, daß Sprengel den Stein nicht nur im proximalen Ende, sondern auch an beliebiger Stelle des erweiterten Lumens gefunden hat, ebenfalls keinen ganz einwandsfreien Beweis dafür zu liefern auf Grund folgender Erwägungen. Wenn man nämlich primär die Einklemmung des Kotsteines und Sekretstauung im distalen Ende des Appendix als Folge dieser Einklemmung zunächst zugibt, so wird im Falle einer fortschreitenden Stauung des Sekrets und entsprechender Erweiterung des Lumens distal vom Kotstein naturgemäß zunächst das Lumen in der Mitte des distalen Endes, dann aber auch nach der Spitze und der Verschlußstelle zu, mit einem

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Wort nach den beiden Enden hin im Durchschnitt erweitert werden. Physikalisch ausgedrückt wird das zunächst einen Zylinder darstellende Lumen des distalen Teiles das Bestreben haben, in die Form eines Ovals mit dauernd abnehmendem Längsdurchmesser und zunehmendem Querdurchmesser überzugehen, das ideal gedacht schließlich in der Kugelform enden würde.

Welches werden nun die Folgen dieses Vorganges auf den eingeklemmten Kotstein sein können? Es liegt auf der Hand, daß daraus auch eine Erweiterung des Lumens an der Stelle des Kotsteines resultieren muß, die zur Abhebung der prall gespannten Wand vom Kotstein und damit zur Aufhebung der Einklemmung führt, dergestalt daß der Kotstein, sekundär frei geworden, nunmehr im freien Raum dem Gesetz der Schwere folgend seinen Platz verläßt und später am tiefsten Punkt der Höhle bzw. „frei schwimmend" im Sekret gefunden wird. Letzteres ist naturgemäß stets spezifisch leichter als der Kotstein, so daß von einem eigentlichen Schwimmen desselben im Sekret der Höhle keine Rede sein kann. Er wird sich vielmehr in derartigen Fällen stets am tiefsten Punkt der mit dem Stauungssekret gefüllten Höhle aufhalten.

Ist es nun proximal neben bzw. vor dem Kotstein durch die intensiveren Entzündungsvorgänge seiner Umgebung inzwischen zu einer selbsttätigen Verlegung des Lumens vermittelst der geschwellten Schleimhaut und entzündlich fibrinöser Verklebung derselben gekommen, so können sich ohne weiteres Befunde ergeben, wie sie Sprengel bei seinen Fällen erhoben hat, die dann leicht zu der Annahme verleiten können, daß dieser erst sekundär auf der Grundlage des eingeklemmten Steins entstandene, selbsttätige Verschluß des Lumens primär und ohne Zutun des nunmehr frei im Lumen vorhandenen Kotsteines zustande gekommen sei.

Ist dagegen der proximal neben dem Kotstein gelegene Verschluß nicht fest oder nicht dicht genug, so kann sich durch die zwar nicht fest schließende, aber immerhin verengte Stelle des Lumens ein Teil des gestauten Sekretes entleeren und man findet dann distal von der vorhandenen Verengerung in dem mittlerweile wieder mehr zusammengefallenen Lumen des Wurmfortsatzendes den Kotstein „an beliebiger Stelle", scheinbar ohne jeden Zusammenhang mit der stenosierten Stelle des Lumens vor.

Wenn nun ein derartiger Vorgang auch nicht allen entsprechenden Fällen Sprengels voraufgegangen zu sein braucht, so darf man doch einerseits die Möglichkeit eines derartigen Vorganges vom physikalischen Standpunkt aus, dessen Anwendung berechtigt erscheint, nicht ganz von der Hand weisen und andererseits müßte Sprengel

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in seinen Fällen einen derartigen Vorgang mit Sicherheit auszuschließen in der Lage sein, ehe man die von ihm angeführten Fälle als einwandsfreien Beweis für seine Anschauung gelten lassen dürfte. Sie beweisen allenfalls die Möglichkeit, daß eine Behinderung des Sekretabflusses durch einen Kotstein im Lumen des Appendix nicht einzutreten braucht, sie beweisen aber meines Erachtens noch lange nicht die Unmöglichkeit, daß eine Behinderung des Sekretabflusses durch einen Kotstein eintreten kann, womit dann auch die mechanische Bedeutung der Kotsteine für die Ätiologie und Genese der Appendizitis doch nicht so kategorisch von der Hand zu weisen sein würde.

Daß ein Kotstein an sich ohne eine entsprechende Gelegenheitsursache in Gestalt einer erhöhten Infektionsmöglichkeit oder eines Traumas keine Appendizitis hervorrufen wird, ist zu selbstverständlich, als daß es einem Zweifel unterliegen könnte. Daß deswegen aber der Kotstein unter Voraussetzung einer derartigen Gelegenheitsursache als prädisponierendes Moment für die Erkrankung, sei es nun mechanischer oder bakterieller Natur oder beides zugleich, seine Bedeutung nicht behalten sollte, scheint mir danach eine Annahme, die sehr weitgehend und zum mindesten noch nicht genügend bewiesen ist.

Die heute noch geteilten Anschauungen über die Rolle des Kotsteines bei der Genese der Appendizitis mögen es gerechtfertigt erscheinen lassen, wenn ich dieser Frage einen größeren Raum eingeräumt habe, als es eigentlich dem Rahmen dieser Arbeit entsprechen dürfte.

Daß gelegentlich auch ein Trauma durch umschriebene Gewalteinwirkung gegen den Leib bei latenten pathologischen Veränderungen des Processus oder einem mit einem Kotstein versehenen normalen Processus einen akuten Infektionsanfall durch Kontinuitätstrennungen der Wand bzw. Ernährungsstörungen der Wand auslösen kann, ist ohne weiteres anzunehmen und wird auch von den meisten Autoren zugegeben. Für den normalen Processus ohne Kotstein ist jedoch eine derartige Einwirkung eines Traumas bisher mit Sicherheit nicht erwiesen, und dürfte sich selbst unter Voraussetzung einer derartigen Möglichkeit ein Beweis für den vorherigen Normalzustand des Processus stets nur aus Wahrscheinlichkeitsgründen bzw. per exclusionem erbringen lassen. Jedenfalls sprechen die theoretischen Erwägungen mehr gegen, als für die Möglichkeit eines derartigen Vorganges.

Eine Begünstigung der pathologischen Vorgänge kann nach Aschoff stattfinden durch ein Hypertrophie der Wurmfortsatztonsille, die nach seiner Meinung nicht einer chronisch fortschreitenden, destruierenden Entzündung entspricht und uns in ihrer Ätiologie noch

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ebenso unbekannt ist, wie die chronische Hypertrophie der Gaumentonsillen. Durch die hyperplastische Entwicklung und das Aneinanderrücken der Noduli kann es dann im ersten Stadium der Entzündung zu der Entstehung einer körnigen, granulösen Beschaffenheit der Schleimhaut kommen, die zu der Bezeichnung Appendicitis granulosa geführt hat. In dieser Appendicitis granulosa hat man wohl mit der Mehrzahl der Autoren das Anfangsstadium der Erkrankung zu erblicken, aus dem sich dann die weiteren Folgezustände und pathologischen Veränderungen des Appendix langsamer oder schneller entwickeln, je nach der Virulenz der Entzündungserreger einerseits und der Widerstandskraft des Gewebes andererseits.

Die vielfach in dem ersten Stadium beobachteten, kleinen, stigmataähnlichen, über die ganze Schleimhaut des Processus in mehr weniger großer Ausdehnung disseminierten Blutungen, über deren Genese die Akten noch nicht geschlossen sind, haben für Riedel die Veranlassung zu der Bezeichnung Appendicitis granulosa haemorrhagica gegeben, indem Riedel diese Blutungen als eine primäre Manifestation der Entzündung auffaßte, gleichwie man dyspeptische oder enteritische Alterationen am Verdauungstraktus beobachtet hat, die sich ebenfalls durch punktförmige, flächenhafte oder zirkuläre Blutungen in die Schleimhaut charakterisierten. Ich möchte dabei besonders auf die an der Magenschleimhaut bei schweren Infektionen des Darmtraktus (Peritonitis) beobachteten, stigmataähnlichen Blutungen hinweisen, welche bekanntlich auf eine destruierende Toxinwirkung der Entzündungserreger auf die Epithelien der Drüsenausmündungen zurückgeführt werden, indem die Toxine zugleich mit dem Sekret der Drüsen in den Magen abgesondert werden.

Ob in ähnlicher Weise entweder die Toxine der im Processus selbst retinierten und gerade hier in unverhältnismäßig reichlicher Menge vorhandenen, lebenden Bakterien oder andererseits die Endotoxine der im Processus infolge erhöhter Schutztätigkeit der Gewebe abgetöteten Erreger auf die Epithelien der Appendixschleimhaut einwirken können, ist eine Frage, die sich vielleicht zur Diskussion stellen ließe. Namentlich bei den Endotoxinen dürfte vielleicht durch das Freiwerden und die Einwirkung proteolytischer Fermente seitens der Leukozyten auf die Epithelzellen eine derartige Möglichkeit sich nicht ganz von der Hand weisen lassen. —

Auch Lotheisen vertritt entschieden die Ansicht, daß diese kapillaren Blutungen als eine Folge bzw. ein Symptom der Appendizitis aufzufassen sind, welches fast immer im geraden Verhältnis zu den Beschwerden steht, die die Patienten vor der Operation gehabt haben.

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Aschoff, Bayer, Th. Landau und Fränkel halten dagegen diese Blutungen für sekundär und führen sie auf traumatische Insulte zurück, denen der Processus intra operationem ausgesetzt sei. Karewski meint, daß auf traumatischem Wege wohl kleine Sugillationen entstehen können, daß man diese aber anstandslos von Blutungen pathologischer Natur zu unterscheiden in der Lage sei. Lauenstein und Revenstorff führen gegen die Entstehung der miliaren Blutungen auf artifiziellem Wege an, daß diese Blutungen immer nur in der Schleimhaut unb niemals in der Serosa oder Subserosa beobachtet werden. Dieses wäre unverständlich, wenn die Unterbindung die Veranlassung dazu bilden würde. Auch die Häufigkeit ihres Auftretens bei an sich verschiedenen Krankheitszuständen des Processus spricht gegen solche Annahme. Beide Autoren sind daher geneigt, in vorausgegangenen Entzündungsvorgängen die Ursache der Blutungen zu erblicken.

Daß im übrigen eine Stauung den Blutungen keinesfalls zugrunde liegen kann, beweist die Tatsache, daß man am Processus zuerst die Vene und dann die Arterie unterbunden hat, ohne daß derartige Blutungen in die Erscheinung getreten wären.

Sprengel hat ebenfalls durchaus den Eindruck gewonnen, daß diese Blutungen nicht als Folge eines Traumas, sondern als besondere hämorrhagische Form der Entzündung aufzufassen sind.

Auch wir haben in einer Reihe von Fällen Gelegenheit gehabt, derartige kleine, oberflächliche, fast über die ganze Schleimhaut des Processus disseminierte Blutungen zu beobachten, deren Entstehung auf traumatischem Wege durch den operativen Eingriff sich mit Sicherheit ausschließen ließ. Vielmehr boten diejenigen Sugillationen, die durch operative Handgriffe, Fassen mit Zangen, Abbinden usw. entstanden waren, in jedem Falle ein ganz wesentlich anderes Bild dar, als diese sich ganz gleichmäßig über die sonst völlig intakte Schleimhaut ausbreitenden, punktförmigen Hämorrhagien.

Neuerdings hält Aschoff in seiner letzten Arbeit sogar dafür, daß derartige miliare Blutungen auf der Appendixschleimhaut überhaupt keine pathologische Bedeutung haben, da er solche Blutungen viel reichlicher und ausgedehnter in normalen Wurmfortsätzen gefunden habe, als in solchen mit beginnender Entzündung. Er glaubt daher energisch gegen eine Auffassung dieser Blutungen als Zeichen eines chronischen Reizzustandes Widerspruch erheben zu müssen. Ob damit die Frage über die Herkunft dieser miliaren Blutungen als abgeschlossen zu gelten hat, wird von der Bestätigung dieser Auffassung und Beobachtung durch andere Autoren abhängig zu machen sein.

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Immerhin bleibt selbst bei Annahme dieser Auffassung die Frage nach der Entstehung dieser Erscheinung in einem normalen Darmabschnitt auch ferner noch eine offene, und diese Tatsache möge es erklären, daß ich den Anschauungen der Autoren über dieses Thema etwas mehr Raum gewidmet habe.

Fassen wir nach dieser kurzen Abschweifung das Hauptmoment bei der Entstehung der Appendizitis noch einmal kurz zusammen, so entspricht dieses einer endogenen, bakteriellen Infektion nicht spezifischer Natur vom Darmlumen aus. Zum Eintritt derselben sind gewisse Voraussetzungen erforderlich, die wir unter dem Ausdruck einer erhöhten Disposition zur Erkrankung zusammenfassen. Diese werden meistens durch Stauungsvorgänge im Appendix hervorgerufen, die zu veränderten Druckverhältnissen im Appendixlumen, Erhöhung der Virulenz und Menge der Bakterien (Kotsteine!), Ernährungsstörungen der Wand und Herabsetzung der Widerstandskraft des Gewebes führen.

Bevor wir nun zu der Erkennung der Appendizitis übergehen, empfiehlt es sich zum besseren Verständnis derselben in den einzelnen Stadien kurz den Verlauf des Entzündungsvorganges am Bauchfell zu erörtern. Dieser allein vermag uns ja ein Bild von dem jeweiligen Stande der Erkrankung zu liefern und nach diesem allein ist ja demgemäß unsere obige Einteilung getroffen worden, der wir auch bei der Erörterung der Erkennungsmerkmale in den einzelnen Stadien nach Möglichkeit entsprechen werden.

Folgen wir zunächst der Anschauung Sprengels über das Wesen der Entzündung am Peritoneum, so haben wir deren erste Äußerung in einer ödematösen Schwellung der Darmserosa zu erblicken. Diese bildet die Vorstufe zu der Ausscheidung eines serösen Exsudates in die freie Bauchhöhle, das später eine fibrinöse Beimengung erhält. Das seröse Exsudat ist in der Regel als steril zu bezeichnen und entspricht einer Schutzvorrichtung des Körpers vermöge seiner bakteriziden bzw. bakteriolytischen und antitoxischen Eigenschaften. Die Menge dieses serösen Exsudates wird in der Regel abhängig sein von der Schwere und Ausbreitung der Infektion einerseits, wie der entsprechenden Abwehrbetätigung des Peritoneums andererseits. Ob es danach berechtigt ist, wenn Moskowicz alle Fälle mit einem freien serösen Exsudat in der Bauchhöhle als prognostisch besonders schwer bezeichnet, erscheint mir zweifelhaft, insofern dieses Exsudat wohl für eine Infektion aber auch gleichzeitig für die Fähigkeit des Körpers spricht, seinen Angreifern erfolgreich entgegenzutreten. Die fibrinöse Beimengung des serösen Exsudat entspricht einer weiteren Schutzvorrichtung des Körpers durch ihre Bestimmung, Verklebungen der

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Darmschlingen und so eine Umgrenzung des Entzündungsvorganges herbeizuführen.

Als letzte Stufe der Entzündung folgt schließlich die Ausscheidung von Leukozyten in das serofibrinöse Exsudat, deren Menge von der Stärke des Entzündungsreizes d. h. der Virulenz und Menge der eingedrungenen Erreger abhängig zu machen ist.

Sprengel setzt sich mit seiner Anschauung in einen bewußten Gegensatz zu Hägler und Sonnenburg, indem er ohne den Vorausgang eines serösen Exsudats die Ausscheidung von Fibrin für unmöglich hält. Er erblickt eben in der letzteren einen Fortschritt der Entzündungsäußerung und ist zu dieser Anschauung auf Grund der Erfahrungstatsache gelangt, daß er an seinem gewaltigen Material von Frühoperationen nicht einen einzigen Fall ohne dieses Exsudat beobachten konnte. Sprengel leugnet daher auch naturgemäß das Vorhandensein einer selbständig fortschreitenden Form der abhäsiven Peritonitis im Sinne Nothnagels vollständig.

Demgegenüber halten Hägler und Sonnenburg den Eintritt einer alleinigen fibrinösen Verklebung des Appendix mit seiner Umgebung ohne ein seröses Exsudat für möglich. Sie erblicken also in der Fibrinausscheidung eine verschiedene Form der Entzündungsäußerung, die nach Hägler auf eine geringere Virulenz der Erreger zurückzuführen ist. Sonnenburg erblickt in dieser Form den Ausdruck einer chemisch-toxischen Peritonitis d. h. einer durch die Wirkung ausgeschiedener Toxine oder Endotoxine oder beider zusammen hervorgerufenen Entzündung, bei welcher Bakterien selbst noch nicht in den Peritonealraum eingedrungen sind.

Wägen wir nun beide Anschauungen gegeneinander ab, so dürfte zunächst der Beweis für das tatsächliche Vorhandensein einer chemisch-toxischen Peritonitis im Sinne Sonnenburgs sich schwer erbringen lassen. Es müßte dazu nachgewiesen werden, daß die Peritonealhöhle völlig bakterienfrei, und daß vor oder auch zugleich mit der rein fibrinösen Ausscheidung nicht doch ein mit bakteriziden Eigenschaften ausgestattetes Exsudat — sei es auch in noch so geringer Menge — ausgeschieden wäre, das die etwa in den Peritonealraum eingedrungenen Bakterien zum Absterben gebracht haben könnte. Im anderen Fall wäre es dann keine chemisch-toxische, sondern eine infektiöse Entzündung, mit der wir es zu tun hätten.

Gibt man selbst diese Möglichkeit ohne weiteres zu, so ist immer noch für den Begriff der chemisch-toxischen Peritonitis zu erwägen, daß ja im Grunde die infektiöse Peritonitis ebenfalls auf einer chemisch-toxischen Wirkung der eingedrungenen lebenden und abgestorbenen Bakterien bzw. ihrer entsprechenden Stoffwechselprodukte

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beruht. Danach ist also die Art des Reizes bei der chemisch-toxischen Peritonitis die gleiche, wie bei der infektiösen Peritonitis. Der grundlegende Unterschied zwischen beiden besteht eben nur in dem Fehlen oder Vorhandensein der Erreger im Peritonealraum. Es dürfte daher auch bezüglich des Reizzustandes der Unterschied zwischen beiden nicht in einer verschiedenen Art, sondern in einem verschiedenen Grade desselben zu erblicken sein.

Danach erscheint es aber nicht recht verständlich, daß ein an sich gleichartiger Reiz auf das gleiche Gewebe in verschiedenem Grade ausgeübt eine gänzlich andere Form der Entzündung bzw. der Abwehrbetätigung des Gewebes zur Folge haben sollte. Daß auch Hägler selbst den Reiz als den gleichen, nur in verschiedener Stärke annimmt, erhellt schon daraus, daß er für die chemisch-toxische Peritonitis bzw. die primäre Fibrinausscheidung nur eine geringere Virulenz der Bakterien zur Voraussetzung macht.

Der Begriff der chemisch-toxischen Peritonitis hat daher in diesem Zusammenhang etwas theoretisch Gekünsteltes an sich, ohne darum einer Erklärung der biologischen Vorgänge im Sinne von Hägler und Sonnenburg zwanglos gerecht werden zu können.

Demgegenüber will es mir als ein Vorzug der Sprengelschen Anschauung erscheinen, daß er von vornherein auf eine so feine Unterscheidung der Begriffe verzichtet, vielmehr nur mit einem stärkeren oder schwächeren Reiz auf das Peritoneum und dementsprechend sich stufenweise steigernder Abwehrbetätigung des Peritoneums rechnet. Als Ausdruck der letzteren findet die nachfolgende fibrinöse Beimengung des vorausgehenden serösen Exsudates vom rein biologischen Standpunkt aus eine ungezwungenere und logischere Erklärung, als im ersten Fall. Wenn nämlich ohne weiteres zuzugeben ist, daß gegenüber einem rein serösen, leicht resorbierbaren Exsudat in einer fibrinösen, viel langsamer zurückgehenden Ausscheidung und Verklebung der größere Effekt zu erblicken ist, so erscheint es durch einen einfachen Rückschluß wenig einleuchtend, daß gerade dieser größere Effekt nach Hägler und Sonnenburg theoretisch auf Grund eines geringeren Reizes zustande kommen sollte.

Den gleichen Vorteil wird uns die Anschauung Sprengels für die Erklärung des Verbreitungs- und Umgrenzungsvorganges der Entzündung am Peritoneum gewähren. Werfen wir nämlich die naheliegende Frage auf, warum das serofibrinöse Sekret im Sinne Sprengels in der Nähe des Entzündungsherdes zu Verklebungen führt, während das im Überschuß gebildete Exsudat resorbiert wird, so finden wir von Sprengel selbst eine Erklärung für diese Frage nicht angegeben. Die geringere Beweglichkeit des Appendix bzw. die An-

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nahme einer verringerten örtlichen, auf toxischen Wirkungen beruhenden Darmbewegung dürfte als eine rein mechanische Erklärung der Vorgänge kaum genügen.

Treten wir nun an der Hand der Sprengelschen Anschauung den Vorgängen näher, so können wir zunächst die Art des Reizes als etwas Gegebenes betrachten, daß nur in seiner Stärke wechseln wird. Es liegt nun auf der Hand, daß die Stärke dieses Reizes nach der Peripherie des Entzündungsherdes eine geringere, der Reiz an der Peripherie ein schwächerer sein muß, als nach der Mitte des Entzündungsherdes zu. Mathematisch gesprochen muß die Stärke des Reizes sich proportional der Entfernung vom Ausgangspunkt der Entzündung verringern. Dementsprechend wird auch die Abwehrbetätigung des Peritoneums nach der Peripherie hin eine geringere sein müssen, als nach der Mitte des Entzündungsherdes.

Es wird also im Sinne der Auffassung von Sprengel logischerweise nach dem Appendix hin das seröse Exsudat über einen größeren Reichtum an Fibrin verfügen, als nach der Peripherie zu. Die Folge dieser äußerst zweckmäßig gesteigerten Abwehrbetätigung des Bauchfells wird sich in einer erhöhten Neigung der Darmserosa zu Verklebungen in der Umgebung des Appendix äußern, während das Peritoneum in der Peripherie erst die dem geringeren Reiz entsprechende Vorarbeit geleistet hat. Ist der Zweck der Abwehr erreicht, nehmen die Verklebungen und die Umgrenzung des Entzündungsvorganges zu, so wird entsprechend dem Fortfallen des Reizes zunächst distal die geringe Fibrinausscheidung fortbleiben, die etwa vorher noch nach den Grenzen der Verklebung zu stattgefunden hat. Es wird dann weiter die rein seröse Ausscheidung ebenfalls aussetzen, und es wird dann mit Leichtigkeit die Resorption des im Überschuß ausgeschiedenen, vorwiegend oder rein serösen Exsudates erfolgen können.

Ist andererseits die Zahl und Virulenz der Angreifer eine derartige, daß das Peritoneum nicht mehr zur Entfaltung seiner vollen Abwehrbetätigung kommt, so wird zwar das seröse Exsudat noch zustande kommen, hingegen die fibrinöse Ausscheidung bereits versagen. Das Peritoneum vermag den vordringenden Angreifern keinen Wall mehr entgegenzusetzen und wird von denselben widerstandslos überschwemmt und geschädigt.

Eine derartige Erklärung der Entzündungsvorgänge am Peritoneum scheint mir zwanglos und logisch genug zu sein, um einen Beweis mehr für die Richtigkeit der auf persönliche Erfahrung am Lebenden gestützten Anschauung von Sprengel zu bilden. Sie dürfte andererseits auch vom rein biologischen Standpunkt aus der absoluten Zweckmäßigkeit der Entzündungsvorgänge am Körper am ehesten gerecht

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werden, wie sie ja neuerdings auch von Bier gelegentlich seiner Hyperämielehre besonders hervorgehoben wird.

Wohl kann man auf Grund der Annahme einer zeitlich gesonderten Abwehrbetätigung des Peritoneums einerseits und der Gefäße andererseits — entsprechend einer zeitlich verschiedenen Einwirkung der Toxine und Endotoxine auf das eine oder andere der Gewebe — zu ähnlichen Schlußfolgerungen gelangen, wie sie der Auffassung von Hägler und Sonnenburg entsprechen. Solange aber eine derartige, zunächst nur hypothetische Annahme nicht experimentell bewiesen ist und solange sie nicht eine gleiche ungezwungene und logische Erklärung der biologischen Vorgänge zu liefern vermag, wie die Sprengelsche Auschauung, dürfte der Vorzug der letzteren Anschauung unbestreitbar bleiben.

Es erscheint daher berechtigt, wenn wir den weiteren Ausführungen diese Anschauung von Sprengel zugrunde legen.

Bei der Appendicitis simplex ist nach unserer Umgrenzung dieses Krankheitsbegriffs jede entzündliche Beteiligung des Peritoneums ausgeschlossen, wobei wir absichtlich den Begriff bzw. den Unterschied zwischen einer chemischen und infektiösen Reizung des Peritoneums beiseite lassen, den man gewöhnlich unter der Bezeichnung Peritonismus und Peritonitis auseinanderzuhalten pflegt.

Es liegt danach auf der Hand, daß die äußerlich feststellbaren Merkmale und Beschwerden der Appendicitis simplex ganz geringe sein müssen, so daß sie in der Tat dem Patienten selbst und vielfach auch der Aufmerksamkeit des Arztes in ihrer eigentlichen Bedeutung entgehen können.

Demgemäß hebt u. a. auch Karewski besonders hervor, daß die eigentliche Appendicitis granulosa haemorrhagica — und naturgemäß ebenso der einfache Appendixkatarrh — ohne jeden klinisch ins Auge fallenden Anfall verlaufen kann. Wenn jedoch Karewski das gleiche bei einer Beteiligung der Serosa unter allmählich fortschreitender Tiefenwirkung des Prozesses für möglich hält, so würde einmal dieser Krankheitszustand nach unserer Auffassung bereits unter den Begriff der Periappendicitis incipiens fallen. Des ferneren dürfte bei der bekannten hohen Empfindlichkeit des Peritoneums ein derartiges Fortfallen der Schmerzerscheinungen zu den äußersten Seltenheiten gehören, die dann ein ganz allmähliches Einschleichen des Reizes bzw. der Entzündung zur Voraussetzung haben müßten.

Am häufigsten wird noch bei der Appendicitis simplex eine gewisse Neigung der Patienten zu Verdauungsstörungen ins Auge fallen, insofern häufig eine hartnäckige Obstipation besteht, die bisweilen durch anfallsweise, nur schwer zu beseitigende Durchfälle unter-

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brochen sein kann. Es kann ferner zeitweilig Neigung zu Flatulenz, Appetitlosigkeit, leichte Übelkeit, Gefühl von Völle und Aufgetriebenheit des Leibes namentlich rechts bestehen. Kurz es handelt sich meist um leichte Verdauungsstörungen allgemeiner Natur, die zunächst auf das Vorhandensein eines örtlichen Leidens hinzuweisen wenig geeignet sind.

Erheblich deutlicher kann das Bild werden, wenn es infolge Schwellung der Schleimhaut, Verschluß durch einen Kotstein oder vorübergehende Knickungen zu Stauungserscheinungen im Appendix kommt. Diese können durch eine rein mechanische Dehnung und Zerrung der bekleidenden Serosa zu deutlich hervortretenden Beschwerden und Schmerzanfällen führen, die meist nach der Magengegend hin ausstrahlen oder einen ausgeprägt örtlichen Charakter haben und in der Ileocöcalgegend gelegen sind. Es sind dieses die unter dem Namen Appendikularkoliken allgemein bekannten Erscheinungen, die stundenlang ohne Temperatursteigerung oder sonstige Störungen anhalten können und dann ebenso plötzlich und vielfach für immer schwinden, wie sie gekommen sind, sobald die Stauung eine vorübergehende ist.

Ausnahmsweise einmal wird dabei auch eine Temperaturerhöhung zustande kommen können infolge einer erhöhten Aufsaugung von Toxinen und Endotoxinen durch den erhöhten Innendruck im Appendix. Dieselbe fällt aber meistens in 24 Stunden mit der Lösung der Stauung und dem Rückgang der Schmerzerscheinungen wieder ab.

Eines derartigen Falles erinnere ich mich aus meiner Praxis, den ich hier kurz anführen möchte. Ein 13jähriger Knabe erkrankte mit typischen Schmerzen in der Gegend des Mc Burneyschen Punktes und mit deutlich ausgesprochener lokaler Druckempfindlichkeit. Am Abend desselben Tages zeigte die Temperatur einen steilen Anstieg auf 39,6, der Processus war durch die dünnen Bauchdecken deutlich fühlbar und schmerzhaft, so daß ich dem Vater des Pat. für den nächsten Tag die Operation in Aussicht stellte, falls nicht wesentliche Besserung eingetreten wäre. Am nächsten Tage war und blieb die Temperatur völlig normal, der Schmerz in der Ileocöcalgegend war nur noch auf Druck in geringem Grade vorhanden und am nächsten Tage völlig geschwunden. Der Patient ist seitdem — es ist jetzt etwa 3 Jahre her — gänzlich beschwerdefrei geblieben. Was mich trotz der hohen Temperatur bewog zu warten, war das fast völlige Fehlen der reflektorischen Bauchdeckenspannung rechts und der nur wenig beschleunigte, in seiner Stärke unveränderte Puls des Patienten. Der Fall beweist wieder den Vorteil der Empfehlung von Sprengel, erst 24 Stunden abzuwarten und nur dann operativ vor-

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zugehen, wenn innerhalb dieser Zeit nicht eine entschiedene Wendung zum Bessern eingetreten ist.

Im anderen Falle würde man eben Gefahr laufen, häufig eine gänzlich rückbildungsfähige Appendicitis simplex vorzufinden und damit eine Luxusoperation zu machen, die durch nichts gerechtfertigt erscheint. Allerdings käme man dadurch dem Vorschlag Karewskis nahe, der am liebsten bereits jede Appendizitis noch vor dem Anfall operieren möchte. Dieser Vorschlag erscheint aber schon aus der obigen Erwägung heraus viel zu weitgehend, als daß er ernstere Beachtung finden könnte.

Im allgemeinen wird also die Appendicitis simplex keine örtlichen oder allgemeinen, ins Auge fallenden Erscheinungen hervorrufen, es sei denn, daß durch Stauungsvorgänge im Appendix Schmerzempfindungen oder Resorptionsfieber von meist schnell vorübergehendem Charakter ausgelöst werden.

Das Bild ändert sich mit dem Übergreifen des Entzündungsprozesses auf das viszerale bzw. parietale Peritoneum sofort und wir haben damit die

Periappendicitis incipiens

vor uns.

Diese ist zunächst eine schneller oder langsamer fortschreitende bis zu dem Moment, wo entweder eine Umgrenzung des Entzündungsvorganges eintritt und sie zu einer Periappendicitis circumscripta wird, oder wo das unaufhaltsame Fortschreiten des Entzündungsvorganges zur Allgemeininfektion des Peritoneums führt, und sie zur Periappendicitis diffusa bzw. Peritonitis appendicularis diffusa wird. Aus der Schnelligkeit oder Langsamkeit des Vorganges noch besondere Bezeichnungen herzuleiten, wie akut fortschreitende Peritonitis, foudroyante Peritonitis, peritoneale Septhämie u. dgl., trägt wohl zur Vermehrung der Nomenklatur, nicht aber zum leichteren Verständnis und zur leichteren Unterscheidung der einzelnen Krankheitsstadien bei.

Entsprechend der allgemeinen Entzündungslehre äußert sich auch am Peritoneum der erste Grad derselben in einer starken Hyperämie der Serosa, die zu einer ödematösen Schwellung des Gewebes führt. Dieser Vorgang wird bei der Empfindlichkeit des Peritoneum bereits die ersten Schmerzempfindungen auslösen.

Je nach der Heftigkeit und Schnelligkeit des Vorganges wird der Charakter der Schmerzen ein verschiedener sein, ja er kann wie eben besprochen ganz oder fast ganz in vereinzelten Fällen ausbleiben, wenn die Entzündung sich gewissermaßen ins Peritoneum einschleicht. Es können sich dann in der gleichen schleichenden Weise

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die weiteren entzündlichen Vorgänge mehr oder weniger im Peritoneum ausbreiten und langsam in das chronische Stadium übergehen, ohne daß Patient im eigentlichen Sinne des Wortes einen Blinddarmanfall durchgemacht hätte.

Dieser gewissermaßen larvierte Verlauf der Erkrankung hat nun für Ewald die Veranlassung zu einer weiteren Vermehrung unserer Nomenklatur durch die Bezeichnung Appendicitis larvata abgegeben, womit ausgedrückt werden soll, daß es eigentlich eine Periappendizitis ist, die sich durch eine besonders langsame und leichte Art des Infektions- und Entzündungsvorganges am Peritoneum auszeichnet. Ewald hat dafür noch besondere Erkennungszeichen aufgestellt, die er als lokaler Druckschmerz, Gurren von Darmschlingen, örtliche leichte Auftreibung, Magensymptome, Flatulenz und häufig Colica mucosa bezeichnet. Diese Merkmale sind aber im allgemeinen dieselben, wie bei jeder anderen Periappendizitis auch, nur daß eben entsprechend den leichteren örtlichen Entzündungsvorgängen die örtlichen Erscheinungen mehr in den Hintergrund und dadurch die leichten allgemeinen Erscheinungen mehr in den Vordergrund treten, so daß das ganze Krankheitsbild sich mehr der Appendicitis simplex nähern kann und im großen und ganzen nach der Schwere der Erscheinungen ein Mittelding zwischen der Appendizitis und Periappendicitis incipiens bilden wird. Ob es sich darum empfiehlt, die Appendizitisnomenklatur um einen neuen Begriff mehr zu belasten, muß dahingestellt bleiben. Auch Geheimrat Garrè kann sich absolut nicht mit der Appendicitis larvata Ewalds befreunden, da schließlich fast jede Erkrankung hin und wieder einen verschleierten oder larvierten Symptomenkomplex zeigen kann, wie z. B. die Pneumonie und insbesondere die zentrale Pneumonie, die Pankreatitis, selbst die Osteomyelitis mit wenig virulenten Keimen. Nicht die Appendizitis hat nach ihm die Larve vor dem Gesicht, sondern eher der Arzt einen Schleier vor den Augen.

So häufig der Schmerz das erste Anzeichen ist, welches die Aufmerksamkeit des Patienten und des Arztes auf den Erkrankungszustand im Abdomen lenkt, so häufig kann die Bestimmung dieser Erkrankung erschwert werden durch die verschiedenartige Lokalisation des Schmerzes. Durch eine Ausstrahlung des Nervenreizes im Plexus meseraicus sup. kann es zu Schmerzempfindungen in der Magen- und Nabelgegend, ferner in der Leber- und Gallenblasengegend, der rechten und bisweilen auch der linken Nierengegend kommen. Schließlich sollen vereinzelt im frühesten Stadium von Karewski als prämonitorische Blasenstörungen bezeichnete Beschwerden beim Urinieren vorkommen, ohne daß ein krankhafter Befund zu erheben wäre.

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Die Ursache der Schmerzen erblickt Kelling in einem Reizzustand des Plexus ileocolicus durch entzündliche Vorgänge am Appendix. Von hier aus kann dann eine Ausstrahlung der Schmerzempfindungen in andere Plexusgebiete erfolgen.

Sprengel weist darauf hin, daß nur das parietale Peritoneum empfindlich ist, welches durch die Interkostalnerven, die Lumbal- und Sakralnerven versorgt wird. Das viszerale Peritoneum hält er für unempfindlich, da seine Versorgung durch das Gebiet des N. sympathicus und N. vagus erfolgt. Im übrigen nimmt auch Sprengel an, daß der Schmerz im Zusammenhang mit den krankhaften Veränderungen am Peritoneum steht, da der zuerst allgemeine Charakter desselben sich mit der Umgrenzung der Entzündung in einen mehr örtlichen umzuwandeln pflegt. Auch Sprengel sieht in der akut entzündlichen Schwellung und dem Ödem des Peritoneums die erste Ursache zur Auslösung der entzündlichen Schmerzempfindung.

Karewski und Lennander schieben nur einen Teil der Schmerzen auf den peritonealen Reiz- bzw. Entzündungszustand und erblicken die Ursache für den anderen Teil der Schmerzen in einer Lymphangitis des Mesenteriums, die ihren Ausgang von den Lymphgefäßen des Mesenteriolum aus nehmen kann.

Daß nebenbei durch etwaige Darmkoliken ebenfalls Schmerzempfindungen ausgelöst werden können, liegt auf der Hand. Dieselben werden in der Regel jedoch nur für das vorgeschrittenere Stadium der Periappendizitis in Betracht kommen.

Die eigentliche Lage der Schmerzen, sowie ihre größte Heftigkeit wird sich naturgemäß früher oder später am Ausgangspunkte der Entzündung, dem Processus, konzentrieren, insbesondere wird sich hier die Empfindlichkeit auf Druck am stärksten äußern, was bekanntlich zu der Festlegung einer typischen Schmerzstelle unter der Bezeichnung des McBurneyschen Punktes geführt hat. Diese besteht so oft zurecht, als eben der Appendix entsprechend gelegen ist. Bei der Verschiedenheit seiner Lage ist dieses jedoch durchaus nicht immer der Fall, so daß davor gewarnt werden muß, diesem Punkte eine übertriebene Bedeutung beizulegen. Kelling ist sogar geneigt anzunehmen, daß dieser Punkt einem rein nervösen Druckpunkt entspricht, und daß daher die Annahme irrig ist, man müsse bei ausgesprochener Druckempfindlichkeit an dieser Stelle immer den empfindlichen Processus unter den Fingern haben. Einen weiteren Beweis dafür erblickt er in der Tatsache, daß man auch bei einer Reihe anderer Erkrankungen diese Erscheinung gefunden hat. Eine weitere Betätigung dafür könnte man ferner in einer Beobachtung erblicken, auf die Professor Lexer öfters hinzuweisen pflegt, nämlich

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daß man bei verdächtigen Fällen oft an der korrespondierenden Stelle links die gleiche Druckempfindlichkeit seitens der Patienten angegeben erhält. Wir haben dieses oft nachgeprüft und bestätigt gefunden.

Zur frühzeitigen Erkennung des Ausgangspunktes und der Umgrenzung der Schmerzen sind nun von einzelnen Autoren besondere Untersuchungsmethoden vorgeschlagen, die namentlich in zweifelhaften Fällen von Vorteil sein können und z. T. dem Bestreben entsprungen sind, den Appendix der Palpation besser zugänglich zu machen.

So rät Lenzmann, bei der Untersuchung den Patienten das rechte Bein aktiv heben zu lassen in der Annahme, daß auf diese Weise der sich kontrahierende M. ileopsoas den Appendix gegen die Bauchdecken hin hebt und ihn so dem untersuchenden Finger näher bringt. Ob dieser Vorteil aber durch die beim Heben des rechten Beines sich verstärkende Spannung der Bauchdecken nicht aufgehoben wird, erscheint zum mindesten fraglich.

In gleicher Weise rät Meltzer zunächst einen tieferen Druck in der Ileocöcalgegend bzw. der Gegend des McBurneyschen Punktes auszuüben und während dieses Druckes das rechte Bein des Pat. zu beugen. Auf diese Weise ist dann der auf dem Ileopsoas liegende Appendix einem stärkeren Druck ausgesetzt und wird schmerzhafter.

Neuerdings weist Blumberg auf ein besonderes Frühmerkmal der beginnenden Periappendizitis hin, das auch Ewald bestätigen kann. Es soll danach bei langsamem Eindrücken der Bauchdecken am Sitz der auf das Peritoneum übergreifenden Entzündung der Schmerz auf plötzliches Nachlassen dieses Druckes stärker sein, als während des Druckes selbst. Eine weitere Bestätigung dieser Beobachtung seitens anderer Autoren steht jedoch zurzeit noch aus.

Ploenies gibt eine besondere Untersuchung der perkutorischen Druckempfindlichkeit an, die er bei vorsichtiger Anwendung für ungefährlicher hält, als die Palpation. Als Vorzüge seiner Methode führt er an, daß dadurch eine scharfe Umgrenzung des Krankheitsherdes ermöglicht werde und daß ferner damit eine Verschleierung des Bildes durch Umgehung der bestehenden Muskelspannung ausgeschaltet werde. Neuerdings gibt Rovsing ein Merkmal an, das recht zeitgemäß sogar in einer großen Anzahl von Tageszeitungen abgedruckt worden ist. Danach soll bei Druck und Aufwärtsstreichen in der linken Bauchseite, dem rückwärtigen Verlauf des Colon descendens entsprechend, durch das Zurückdrängen des Darminhaltes gegen die Ileocöcalklappe hin eine deutliche Schmerzempfindung an

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der Stelle des entzündeten Appendix bzw. in der Ileocöcalgegend ausgelöst werden. Es muß sich erst zeigen, ob es ein pathognomonisches Symptom ist.

Schließlich sei der Vollständigkeit halber noch erwähnt der Hinweis Kümmels auf umschriebene Hauthyperästhesien, welche den Headschen Zonen entsprechen und am häufigsten in der Gegend des Mc Burneyschen Punktes vorkommen sollen. Ebenso hat auch Peiser die Beobachtung von Sensibilitätsstörungen der Haut bzw. der Bauchdecken über Eiterungen in 9 Fällen machen können. Und zwar bestand in 6 Fällen eine Hypästhesie, in 2 Fällen Hyperästhesie und in einem Falle erst Hyper- und später Hypästhesie. Letztere Merkmale sind wegen ihrer Seltenheit nur von untergeordneter Bedeutung und kommen naturgemäß mehr für die Periappendicitis circumscripta, als für die beginnende Periappendizitis in Frage.

Als Folge der Schmerzhaftigkeit des Peritoneums kommt es dann zur Entstehung einer reflektorischen Bauchdeckenspannung rechts, die man wohl am einfachsten als eine reflektorische Stillstellung der Bauchdecken zum Schutz des entzündeten und bei jeder Bewegung schmerzhaften Teiles des Peritoneums auffassen darf.

Sprengel erblickt in ihr die Folge des zum serösen Exsudat führenden Reizzustandes des Peritoneums und führt sie als ausgesprochenes Merkmal für ein vorhandenes bzw. in Entstehung begriffenes Exsudat an, mit dessen Verschwinden auch die Spannung der Bauchdecken zurückgehen soll.

Die weitere Folge dieser reflektorischen Bauchdeckenspannung ist eine respiratorische Bauchdeckenstarre der rechten Inguinalgegend, die bei Besichtigung des Patienten vom Fußende her ins Auge fällt. Da von Küster besonders auf sie hingewiesen ist, wird sie auch als Küstersches Symptom bezeichnet. Küster führt sie auf eine kleinzellige Infiltration im extraperitonealen Gewebe zurück, welche die Elastizität der tiefen Bauchmuskeln aufheben und die Folge der beginnenden Eiterung sein soll. Zwangloser erscheint mir auch hierfür die gleiche Erklärung, wie wir sie für die reflektorische Bauchdeckenspannung angeführt haben.

Entsprechend dieser respiratorischen Bauchdeckenstarre Küsters weist Sprengel ebenfalls als Frühmerkmal auf eine Verringerung der Zwerchfellatmung bzw. einen vorwiegend kostalen Atmungstypus hin, der namentlich bei männlichen Patienten von Bedeutung ist, da ja bei Frauen auch im normalen Zustand infolge des Schnürens der kostale Atmungstypus zu überwiegen pflegt. Seine Entstehung bei der Periappendizitis verdankt er wohl gleichfalls dem Bestreben der

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Patienten durch Einschränkung der Zwerchfellbewegung das Peritoneum möglichst ruhig zu stellen.

Körte hält die reflektorische Bauchdeckenspannung rechts für das sicherste Zeichen der beginnenden Periappendizitis. Immerhin muß man dabei sicher sein, die bei jeder Berührung auch normaler Bauchdecken vielfach eintretende Spannung der Muskulatur ausschalten zu können. Ein Vergleich der rechten Seite gegen die linke wird dabei in der Regel entscheidend sein und darf daher nie unterlassen werden.

Ferner kommt es wohl durch Schwellung der Serosa einerseits, sowie durch toxische Einflüsse auf die Innervation der Darmschlingen andererseits häufig zu einem geringen Meteorismus, der nach Dörffler vorwiegend rechts auftritt und sich naturgemäß auf die Umgebung des beginnenden Krankheitsherdes beschränkt. Dieser Meteorismus ist so gering, daß er zunächst nicht weiter ins Auge fällt. Er bewirkt jedoch nach Sprengel eine Kantenstellung der Leber, bei welcher die Leberdämpfung in der Mammillarlinie bisweilen fast vollständig fehlen kann. Auch Oppenheim weist auf dieses Fehlen der Leberdämpfung als besonderes Erkennungszeichen einer vom Processus ausgehenden örtlichen Entzündung hin und führt diese Erscheinung ebenfalls auf einen äußerlich meist nicht zur Wahrnehmung gelangenden Dickdarmmeteorismus zurück.

Daß entsprechend der örtlichen Herabsetzung der Darmtätigkeit eine meist ziemlich hartnäckige Verhaltung von Stuhl und bisweilen auch von Winden eintreten kann, ergibt sich nach dem obigen von selbst.

Die Temperatur antwortet in der Regel auf den Einbruch der Entzündung in das Peritoneum mit einem deutlich erkennbaren Anstieg und bewegt sich entsprechend dem Fortschreiten der Entzündung weiter in aufsteigender Linie. Vereinzelt können ausnahmsweise auch Schüttelfröste auftreten, während andererseits bei geringer Virulenz und sehr langsamem Fortschreiten der Infektion und des Entzündungsreizes auf das Peritoneum die Steigerung der Temperatur bisweilen gänzlich fortbleiben kann.

Als ein besonderes Zeichen drohender bzw. ausgebrochener Peritonitis weist Krogius darauf hin, daß in solchen Fällen die Mastdarmtemperatur nicht mehr in dem gewöhnlichen Verhältnis zur Achselhöhlentemperatur steht, sondern wesentlich höher ist als sonst. Krogius selbst führt an, daß er bei annähernd normaler Achselhöhlentemperatur bis 40° C im After gemessen habe. Dieses Zeichen ist einerseits um so wertvoller, als es jederzeit leicht nachzuweisen ist. Andererseits ist dabei zu bedenken, daß es von der Entfernung des Entzündungsprozesses bzw. der Hyperämie vom Rektum abhängig

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ist. Es wird daher bei hochsitzendem Processus um die Zeit später zum Ausdruck kommen, welche die Entzündung braucht bis zu ihrer Entwicklung in die Gegend des Rektums hin.

Rostowzew hebt als weiteres Vorzeichen seitens der Temperatur für den weiteren Verlauf der Erkrankung hervor, daß bei der Periappendizitis die höchste Temperatur in der Regel nicht von 4—6 Uhr nachmittags, sondern von 9—10 Uhr abends gemessen wird. Nach der Häufigkeit dieser späteren Höchsttemperatur am Abend will Rostowzew die Schwere des Falles bemessen und sucht hierfür den Beweis an der Hand einer selbstgefertigten Tabelle zu erbringen. Weitere Bestätigungen dieser Beobachtung bleiben einstweilen abzuwarten.

Der Puls ist meist entsprechend der Temperatur von größerer Frequenz als normal, jedoch in der Regel nicht kleiner, sondern eher noch etwas voller als vorher. Sprengel erblickt in einer wesentlichen Zunahme der Pulsbeschleunigung eine erhöhte Indikation für die Dringlichkeit des operativen Eingriffs.

Ferner kommt es entsprechend der fortschreitenden entzündlichen Reizung auf das Peritoneum zu stärkerer Übelkeit, Aufstoßen und Brechreiz, sowie in meist größeren Zwischenräumen zu Erbrechen. In ganz vereinzelten Fällen ist Blutbrechen beobachtet worden, das dann wohl als Ausdruck einer besonders schweren Allgemeinvergiftung stets von der übelsten Vorbedeutung gewesen ist.

Als Ergänzung zu der perkutorischen Druckempfindlichkeit nach Plönies sei noch darauf hingewiesen, daß man auch in frühen Stadien der Entzündung eine leichte perkutorische Dämpfung erhalten kann, welche auf den vermehrten Perkussionswiderstand durch die geschwellten und ödematösen Gewebe zurückgeführt wird. Ein Widerspruch gegen diese Annahme ist in dem oben erwähnten geringen Dickdarmmeteorismus wohl kaum zu erblicken, da durch letzteren nur die Höhe oder Tiefe des Tones, hingegen durch den vermehrten Widerstand des Gewebes die Stärke des Perkussionstones geändert wird.

Entsprechend dieser Schwellung der Gewebe bei geringem örtlichen Meteorismus kann man bisweilen an der Stelle der Entzündung das Gefühl einer undeutlichen, schmerzhaften Schwellung unter den Fingern haben, falls man nicht in der Lage ist, den geschwellten und meist äußerst empfindlichen Processus selbst nachweisen zu können. Letzteres ist jedoch vielfach wegen der reflektorischen Bauchdeckenspannung mit Schwierigkeiten verknüpft.

Bei sehr tiefem Sitz des Appendix in der Nähe der Blase kann es ausnahmsweise auch früh bereits zu Blasenstörungen kommen,

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die sich von den sog. prämonitorischen Blasenstörungen Karewskis dadurch unterscheiden, daß sie einer pathologisch-anatomischen Grundlage nicht entbehren. In der Regel werden jedoch Blasenstörungen, wenn es überhaupt dazu kommt, erst nach einer Zeit einsetzen, welche dem Vorrücken der Entzündung bis in die Blasengegend entspricht.

Kommen wir nun schließlich zu der in neuerer Zeit mehr und mehr in Aufnahme gelangenden Bestimmung der Leukozytenwerte, so leistet nach dem übereinstimmenden Urteil fast sämtlicher Kliniker diese Methode gerade in den ersten 48 Stunden, wo es am meisten darauf ankäme, fast nichts zur Klärung der Sachlage. Ebenso ist auch für die späteren Stadien der Erkrankung die Methode nicht als absolut zuverlässig zu bezeichnen.

So hält Rehn die Leukozytenzählung nicht nur für überflüssig, sondern auch für gefährlich, weil man sich durch derartige Zählungen in Sicherheit wiegen kann und dadurch unter Umständen den richtigen Zeitpunkt zur Operation versäumt. Auch er bezweifelt die Zuverlässigkeit der Methode für eine Anzahl von Fällen auf Grund seiner Erfahrungen.

Nach Ch. Juillard ist die Leukozytenzählung in den ersten 48 Stunden ohne Bedeutung, dagegen soll sich bei eiterbildenden Formen die Kurve 2—3 Tage lang über 25000 erheben.

Wassermann hat ebenfalls einige Mißerfolge dieses Merkmals gesehen, insofern er Fälle beobachten konnte, bei denen trotz vorhandener Eiterung keine Leukozytose da war.

Russel kann ebenfalls in der Leukozytosenbestimmung kein ausschlaggebendes Merkmal erblicken. Auch Körte kann die Leukozytenzählung nicht für die Frühdiagnose verwerten, da er dadurch oft Enttäuschungen erlebt hat.

Nach Nordmann leistet die Leukozytenbestimmung in den ersten 48 Stunden gar nichts. Auch er fand trotz erhöhter Leukozytenzahl einen normalen Wurmfortsatz. Ebenso konnte trotz starker Vermehrung der Leukozyten in anderen Fällen der operative Eingriff ohne Schaden hinausgeschoben werden. Im Gegensatz hierzu bestand in einem Falle eine normale Leukozytenzahl trotz des Vorhandenseins einer ausgedehnten Eiterung.

Nach Hochenegg kann auch bei Douglasabszessen die Leukozytose vollständig fehlen.

Federmann sieht nicht in einer einzelnen Zählung, sondern in der Anlegung einer Kurve die Bedeutung der Leukozytose. Damit spricht er ihr bezüglich ihres Wertes für die Indikationsbestimmung der absoluten Frühoperation ebenfalls das Urteil. Das Ansteigen und

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Fallen der Leukozytose bewegt sich nach seiner Ansicht parallel der Temperatur, demnach kann in den ersten 48 Stunden die Leukozytose wohl das Zeichen einer Infektion, aber noch keiner Eiterung sein. Die Appendicitis simplex, womit wohl die Periappendicitis incipiens gemeint ist, hat nach ihm in den ersten 2—3 Tagen stets unter 20000, weswegen er für das Frühstadium die Leukozytose ausdrücklich für wertlos erklärt. Dagegen kann bei einem Empyem des Appendix der Leukozytenwert bis auf 25000 steigen, während wieder umgekehrt bei einer derartigen Leukozytenzahl nicht unbedingt ein eitriger Prozeß vorhanden zu sein braucht. Nach den ersten 5 Tagen hält Federmann eine Leukozytenzahl über 20000 für ein sicheres Anzeichen einer wachsenden Eiterung, während andererseits das Fehlen der Leukozytose in diesem Stadium nicht gegen eine vorhandene Eiterung zu sprechen braucht. Mit anderen Worten spricht also im Frühstadium nach Federmann eine hohe Leukozytose nicht unbedingt für, und im späteren Stadium das Fehlen der Leukozytose nicht gegen das Vorhandensein einer Eiterung.

Sprengel faßt seine Ansicht und Erfahrung über die Leukozytose in 5 Thesen zusammen, die ich nachfolgend wörtlich anführe:

1. Die Leukozytose ist bei den Frühformen geringer, als bei den Spätformen.

2. Die Leukozytenwerte steigern sich anscheinend sowohl in Abhängigkeit von den Veränderungen am Wurmfortsatz, als auch von denen am Peritoneum. Demnach erreichen sie ihren höchsten Grad bei den Fällen von Appendicitis destructiva im Spätstadium.

3. Für die Frühformen sind die Ergebnisse weniger regulär, als für die Spätformen. Es kommen hohe Leukozytenwerte bei der Appendicitis simplex und bei geringen Veränderungen am Peritoneum vor, und umgekehrt niedrige Leukozytenwerte bei schweren Veränderungen am Wurmfortsatz und am Peritoneum.

4. Ein so charakteristischer Unterschied zwischen den Leukozytenwerten bei Appendicitis circumscripta serofibrinosa und purulenta, wie manche Autoren annehmen, ergibt sich nach meinen Zahlen nicht.

5. In allen Stadien der Appendizitis gibt es Fälle, bei denen die Leukozytenwerte in keiner Weise die nach den lokalen Befunden zu erwartenden Änderungen aufweisen.

Sein Urteil faßt Sprengel ablehnend kurz zusammen: Die Methode ist unzuverlässig und läßt uns gerade in den Fällen im Stich, in denen es außerordentlich darauf ankäme, für den Kranken etwas daraus schließen zu können.

Wertvoll kann jedoch die Leukozytenbestimmung zuweilen sein zur Unterscheidung der Periappendizitis gegen andere Krankheits-

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zustände. So will Berndt sie zur Unterscheidung zwischen Typhus und Periappendizitis heranziehen, da sie am Anfang des ersteren vermindert, bei letzterer hingegen erhöht sei. Das gleiche gilt von der Unterscheidung der Peritonitis gegen Ileus, worauf von Federmann besonders hingewiesen wird. Daß sie im übrigen bei sehr schwerer Peritonitis ebenfalls fehlen kann, kommt später noch zur Sprache.

Günstig für die Leukozytose sind anscheinend die Erfahrungen von Curschmann, der die Leukozytenvermehrung als Merkmal vorhandener Eiterung noch über die Temperatur stellt, da letztere leicht täuschen könne, die erstere hingegen zuverlässig sei.

Auch G. Nilson hält nächst dem Ergebnis von Eiter bei einer Probepunktion (!) die Leukozytose für das sicherste Zeichen des Vorhandenseins von eitrigem Exsudat in der Bauchhöhle. Allerdings meint er dann mit vorsichtiger Einschränkung, daß dieses Merkmal den Ausschlag geben könne, wenn die übrigen Erscheinungen entsprechend festgestellt sind. Daß im übrigen die Probepunktion als diagnostisches Hilfsmittel bei einer periappendizitischen Eiterung heute nur noch historischen Wert beanspruchen kann, darüber dürfte man einem Zweifel wohl von keiner Seite mehr begegnen.

Fassen wir nun unsere bisherigen Ausführungen über die Periappendicitis incipiens noch einmal kurz zusammen, so ist für die Erkennung derselben auf folgende Anzeichen besonders zu achten:

1. Anamnestisch: Vorangegangene Magen- und Darmbeschwerden allgemeiner Natur, Neigung zu Verstopfung, bisweilen plötzliche Durchfälle.

2. Allgemeine, später meist in der Ileocöcalgegend umgrenzte Leibschmerzen mit entsprechender Druckempfindlichkeit des Abdomen.

3. Reflektorische Bauchmuskelspannung, vornehmlich rechts.

4. Respiratorische Bauchdeckenstarre, vornehmlich rechts.

5. Überwiegend kostaler Atmungstypus, bzw. etwas oberflächliche und entsprechend beschleunigte Atmung.

6. Kantenstellung der Leber mit Schwinden der Leberdämpfung in der rechten Mammillarlinie.

7. Andauernder, langsamer oder schneller Temperaturanstieg, vereinzelt Schüttelfrost.

8. Vergrößerter Unterschied zwischen Achselhöhlen- und Mastdarmtemperatur.

9. Andauernde, schnelle oder langsame Vermehrung der Leukozytenwerte, sobald sie mit dem übrigen Krankheitsbilde übereinstimmt.

10. Übelkeit und Brechreiz, vereinzelt Erbrechen.

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11. Mehr oder weniger ausgesprochene Stuhlverhaltung, bisweilen heftige Durchfälle.

12. Leichte, dem Verhältnis zur Temperaturerhöhung entsprechende Beschleunigung des in der Stärke unveränderten Pulses.

Es liegt auf der Hand, daß diese Merkmale sowohl im einzelnen wie in ihrer Gesamtheit mit dem weiteren Vordringen der Entzündung im Peritoneum stärker hervortreten werden. Andererseits wird namentlich in den ersten Anfängen der Entzündung das eine oder andere von ihnen nur angedeutet sein oder auch gänzlich fehlen können. Immerhin wird bei genügender Beachtung der angeführten Merkmale und entsprechender Bewertung derselben in ihrem Zusammenhang als geschlossenes Krankheitsbild die Erkennung der Periappendizitis auch in ihrem frühesten Stadium nicht allzuviel Schwierigkeiten bieten, namentlich, wenn man sich stets den Satz von Karewski gegenwärtig hält, daß jeder Mensch mit chronischen, schmerzhaften Darmstörungen der Periappendizitis verdächtig ist, solange keine andere Ursache dafür nachgewiesen ist.

Wesentlich klarer durch die ausgesprochenere Lokalisierung der Erscheinungen bietet sich im Falle einer Umgrenzung des Entzündungsvorganges das Bild der

Periappendicitis circumscripta

dem Untersucher dar.

Vor allem wird fast immer ein abgeschlossenes Exsudat in Gestalt einer mehr oder minder deutlich durch Perkussion und Palpation nachweisbaren Resistenz in der Ileocöcalgegend vorhanden sein. Die Lage dieses Exsudats kann je nach der Lage des Processus eine erheblich verschiedene sein. Es kann nach oben bis unter die Leber, nach innen bis über die Linea alba, nach unten bis in den Douglas und bei retrocöcaler Entwicklung in die Lumbalgegend verlagert sein, woran in zweifelhaften Fällen zu denken ist.

Die am Anfang recht ausgesprochene Schmerzhaftigkeit und Druckempfindlichkeit des Exsudates kann mit der Dauer seines Bestehens mehr und mehr zurückgehen und bei länger bestehenden Abszessen schließlich völlig fehlen.

Die Temperaturerhöhung gelangt in der Regel mit der Umgrenzung der Entzündung zum Stillstand und kann sich dann noch eine gewisse Zeit lang in ziemlich gleichbleibender Höhe bewegen. Sie kann aber auch bei großen Abszessen nach einiger Zeit wieder zur Norm abfallen, da die immer stärkeren Verwachsungen und fibrinösen Beläge der Abszeßwandungen eine Verminderung und schließlich eine Aufhebung der Resorption von Giftstoffen durch den Körper herbeizuführen vermögen. Bei längerem Bestehen kann dann der

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Inhalt der Abszesse völlig steril werden. So hat Noetzel etwa 75% der alten periappendizitischen Abszesse als steril nachweisen können.

Der Puls ist entsprechend der Temperatur meist etwas beschleunigt, in seiner Spannung jedoch nicht wesentlich beeinträchtigt.

Als Unterscheidungsmerkmal gegen die noch nicht abgegrenzte Periappendizitis ist nach Sprengel besonders auf das normale Verhalten der Atmung, sowie den Rückgang bzw. das völlige Fehlen der reflektorischen Bauchdeckenspannung hinzuweisen. Dieses erklärt sich aus dem Fortfallen des nunmehr umgrenzten entzündlichen Reizes auf das freie Peritoneum, das seinen weiteren Ausdruck in dem Fehlen bzw. in dem Rückgang des freien Exsudates in der Bauchhöhle findet.

Aus dem gleichen Grunde hört in dem Stadium der umschriebenen Periappendizitis auch das Erbrechen auf und fängt erst wieder an bei weiterem Fortschreiten und neuen Nachschüben der Entzündung.

Zu achten ist auch auf die oben erwähnten Sensibilitätsstörungen der Haut, die Peiser über Eiterungen beobachtet hat.

Besondere Schwierigkeiten für die Erkennung können bisweilen die retrocöcalen bzw. retroperitoneal gelegenen Abszesse bilden, da sie durch ihre Lage der Palpation und Perkussion schwerer zugänglich sind. Hier wird meist eine willkürliche Haltung des rechten Oberschenkels in leichter Beugung und Außenrotation, sowie starke Schmerzhaftigkeit bei Bewegung des rechten Oberschenkels ein deutliches Merkmal zur Klärung der Sachlage bilden. Das gleiche Zeichen bieten naturgemäß auch die retrofascial gelegenen Abszesse dar, deren Unterscheidung gegen die retrocöcalen und retroperitonealen Abszesse sich im weiteren Verlauf durch ihr Austreten unterhalb des Poupartschen Bandes auf den Oberschenkel ermöglichen läßt. Bei entzündlicher Verlötung des Processus mit dem Peritoneum parietale retrocöcal wird sich schon frühzeitig diese Schmerzhaftigkeit bemerkbar machen infolge einer Zerrung des gereizten Peritoneums auf dem M. ileopsoas durch Bewegungen des rechten Beines im Hüftgelenk. Dadurch kann bisweilen schon im Stadium der Periappendicitis incipiens ein Hinweis auf die retrocöcale Lage des Processus gegeben sein. Verstärkter Mastdarm- und Blasentenesmus, sowie schleimige Beimengung im Stuhl werden häufig auf eine Lokalisierung der Eiterung im Douglas hinweisen, welche ihre Bestätigung durch eine digitale Untersuchung vom Mastdarm aus findet. Letztere sollte bei der Häufigkeit der Douglasabszesse in keinem Falle von Periappendizitis unterlassen werden, wenn die Untersuchung begründeten Anspruch auf Vollständigkeit machen will.

Ferner kann es in vereinzelten Fällen durch Druck großer

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Abszesse gegen ein Darmlumen, seltner durch Knickungen von Darmschlingen infolge breiter oder strangförmiger Verwachsungen zu ausgesprochenem Ileus kommen, über dessen Ursache die Heranziehung der übrigen Krankheitsmerkmale einen Zweifel kaum übriglassen wird. Auf der gleichen mechanischen Grundlage kann bei unvollkommenem Darmverschluß ein entsprechender Meteorismus in die Erscheinung treten.

Auf die etwas unsichere Rolle der Leukozytose auch in diesem Stadium der Erkrankung ist bereits oben hingewiesen worden. Man wird sie hier in der Regel nachweisen können, solange einerseits Giftstoffe der Bakterien vom Blute aufgenommen werden, und solange andererseits der Körper genügend Kraft besitzt, um sich in entsprechender Weise gegen die Schädigung zur Wehr setzen zu können. Aus diesem Grunde darf man wohl mit Lennander das Vorhandensein der Leukozytose als ein günstiges Vorzeichen für den weiteren Verlauf der Erkrankung betrachten.

Mit dem Nachlassen des Übertritts von Toxinen in das Blut oder auch mit dem Versagen der Widerstandskraft des Körpers wird naturgemäß parallel dem Fallen der Temperatur auch die Leukozytenmenge wieder zurückgehen. Der Rückgang der Leukozytose kann daher sowohl auf eine Besserung, wie eine Verschlimmerung des Leidens hinweisen und sie kann daher auch hier nur einen bedingten Wert im Zusammenhang mit den übrigen Krankheitserscheinungen zur Ergänzung des gesamten Krankheitsbildes beanspruchen.

Kurz zusammengefaßt wird demnach die Periappendicitis circumscripta durch den deutlichen Rückgang der vorausgegangenen allgemeinen Entzündungserscheinungen des Peritoneums, durch die mehr oder weniger ausgesprochene Umgrenzung der örtlichen Krankheitserscheinungen, welche meist leicht den Nachweis einer umschriebenen intraabdominalen Eiterung gestatten, für die Erkennung von allen Stadien der Periappendizitis die geringsten Schwierigkeiten darbieten.

Wesentlich schwieriger können die Verhältnisse bei der

Periappendicitis diffusa

liegen, um so mehr als sie sich je nach Zahl und Virulenz der Erreger einerseits und Widerstandskraft des Peritoneums andererseits so schnell entwickeln kann, daß fast explosionsartig eine Infektion des gesamten Peritoneums stattfindet. Auf diese Weise kann man bisweilen im Augenblick der Periappendicitis incipiens schon vor einer Periappendicitis diffusa stehen, gleichwie andererseits aus einer Periappendicitis circumscripta durch äußere Insulte oder sonstige Gelegenheitsursachen jederzeit sekundär eine Allgemeininfektion des Peritoneums zustande kommen kann.

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Man ist daher vom Beginn der Periappendizitis ab keinen Augenblick sicher, plötzlich vor der Tatsache einer Allgemeininfektion des Peritoneums zu stehen und wenn je, so hängt hier das Leben des Patienten von einer möglichst frühzeitigen Erkennung des Krankheitszustandes und der sofortigen Einleitung des einzig möglichen Heilverfahrens, nämlich des operativen Eingriffs, ab. Man wird daher gut tun, sich jederzeit bei Abwägung der Merkmale gegenwärtig zu halten, daß es für den Patienten und den Arzt von geringerem Nachteil ist, wenn gelegentlich ein Fall auf Grund einer allzu vorsichtigen Diagnose noch vor der Peritonitis zur sofortigen Operation gelangt, als wenn in dem Bestreben eine möglichst exakte Diagnose zu stellen, der Zeitpunkt zum Eingriff versäumt und der Kranke einem sicheren Ende überliefert wird.

Jeder einer Peritonitis auch nur verdächtige Fall ist demnach für die Therapie als eine vollgültige Peritonitis zu behandeln.

Die Erkennung der peritonealen Allgemeininfektion kann sich am Anfang gerade der schwersten Fälle um so schwieriger gestalten, als durch die Schwere der Infektion, durch die Übermacht der Angreifer die zunächst als gegebene Größe zu betrachtenden Verteidigungskräfte des Körpers über den Haufen gerannt werden, so daß eine Entfaltung derselben gleich von Anbeginn völlig ausgeschlossen wird. Auf diese Weise kann der größte Teil der sonst klinisch wahrnehmbaren Kennzeichen in Fortfall geraten, und von der Erfahrung und Kenntnis des Arztes wird es abhängen, auch ohne dieselben bzw. gerade wegen des Fehlens derselben die Schwere des Krankheitszustandes richtig zu würdigen.

In der Mehrzahl der Fälle wird jedoch der Körper erst nach einer kürzeren oder längeren Zeit des Kampfes seiner Schutzkräfte und Schutzstoffe gegen den andringenden Feind erliegen. In diesem Stadium werden sich dann naturgemäß die Anzeichen des Kampfes nach außen in der gleichen Weise, wie bei der Periappendicitis incipiens, nur in entsprechend höherem Grade kenntlich machen.

Der Schmerz ist entsprechend der größeren Ausdehnung der Entzündung des Peritoneums in verstärktem Grade im ganzen Leibe vorhanden und wird bereits bei leisem Druck auf die Bauchdecken, sowie bei geringen Bewegungen des Patienten sich besonders heftig bemerkbar machen. Bisweilen kann daneben noch eine erhöhte Empfindlichkeit der Ileocöcalgegend bestehen, die namentlich bei aktiven und passiven Beugungen des rechten Beines zum Ausdruck kommt. Ganz zuverlässig ist aber auch dieses Merkmal nicht, insofern als es bei sehr schweren Fällen wohl durch eine toxische Schädigung der

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Nerven zu einem fast völligen Fortfall der Schmerzempfindung kommt, und man wird daher gut tun, diese Möglichkeit immer im Auge zu behalten.

Ebenso kann auch bei einem Ausgang der Allgemeininfektion von einem tief im kleinen Becken liegenden Appendix die Empfindlichkeit der Bauchdecken verhältnismäßig lange fehlen. Auch hier kann man, wenn man sich zu sehr auf dieses Merkmal verläßt und die erforderliche Untersuchung per rectum unterläßt, leicht die ersten 48 Stunden ungenützt verstreichen lassen und damit die Prognose für die Heilung des Patienten nahezu völlig ungünstig gestalten.

Ganz besonders möchte ich darauf hinweisen, daß auf den sogenannten Perforationsschmerz wenig oder nichts zu geben ist, da er in einer großen Anzahl von Fällen völlig fehlen kann, während er in zahlreichen anderen Fällen durch die vorausgegangenen Schmerzen und Beschwerden derart verdeckt wird, daß er dem Patienten nicht besonders auffällt.

Allerdings wird sich namentlich die schnelle Ausbreitung der Entzündung auf ein größeres Gebiet auch durch schnelle Zunahme der Schmerzen nicht nur an Ausdehnung, sondern auch an Heftigkeit bemerkbar machen. Da aber diese allgemeine Schmerzhaftigkeit gerade am Beginn der Periappendizitis ebenfalls in gewissem Grade vorhanden ist, so wird in diesem Stadium eine Unterschätzung derselben in ihrer Bedeutung bisweilen vorkommen können. Dagegen dürfte in einem späteren Stadium die plötzliche Zunahme der bereits im Abklingen begriffenen Beschwerden schon eher den Fortgang der Entzündung ins Auge fallen lassen.

Ein Frühzeichen der diffusen Periappendizitis bildet ferner die entsprechend der Verallgemeinerung der Entzündung beiderseits bestehende Rektusspannung im Gegensatz zu der nur rechtsseitigen Spannung bei der Periappendicitis incipiens.

Diese Spannung kann sich mit der Zunahme der entzündlichen Veränderungen derart verstärken, daß die Bauchdecken eingezogen und bei der Betastung bretthart erscheinen können. Man hat dann den typischen Anblick des kahnförmig eingezogenen Abdomens vor sich. Infolge Fehlens der Schmerzhaftigkeit des Peritoneums kann gerade bei den schwersten Fällen auch die beiderseitige Rektusspannung in Fortfall kommen.

Um eine weitere Ruhestellung des Peritoneums zu ermöglichen, wird ferner wie bei der Periappendicitis incipiens die Bewegung des Zwerchfells reflektorisch eingeschränkt und wir werden daher neben dem Stillstand der Bauchdecken gleichzeitig auf den vorwiegend oder rein kostalen Atmungstypus zu achten haben. Entsprechend der

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geringeren Tiefe der Atembewegung fällt eine erhebliche Beschleunigung der Atmung ins Auge.

Die Temperatur wird in der Regel auf den erhöhten Reiz durch einen entsprechenden Anstieg, seltner mit Schüttelfrösten antworten. Sie zeichnet sich im übrigen durch einen völlig atypischen Verlauf aus und kann bei den schwersten Fällen, gleich den übrigen Anzeichen völlig versagen. Das ist besonders zu beachten bei denjenigen schweren Allgemeininfektionen des Peritoneums, welche plötzlich ohne besondere Vorboten oder vorausgegangene Schmerzanfälle einsetzen und infolge des Fehlens der üblichen Warnungszeichen bisweilen zu der Annahme einer leichten Periappendicitis incipiens führen können, bis der unaufhaltsame Kollaps zu spät die Schwere des Krankheitszustandes erkennen läßt.

Im weiteren Verlauf der Erkrankung bildet der Abfall der Temperatur bei Fortbestand der übrigen schweren Allgemeinerscheinungen ein übles Vorzeichen als Merkmal beginnenden Kollapses und führt zu der bekannten Kreuzung der Temperatur- und Pulskurve, der fast ausnahmslos das Kreuz als Abschluß der Krankengeschichte nachzufolgen pflegt.

Steiler, plötzlicher Temperaturabfall bei länger bestehender Periappendizitis soll nach W. H. Bennet das häufigste Zeichen einer Appendixperforation sein. In der Regel wird dieser Abfall aber von einem um so höheren Temperaturanstieg bisweilen auch mit Schüttelfrost gefolgt sein.

Auf die oben erwähnte Differenz zwischen Mastdarm- und Achselhöhlentemperatur nach Krogius ist besonders in zweifelhaften Fällen als wertvolles Hilfsmittel zu achten.

Das sicherste, stets zuverlässige Zeichen bildet allein die Beschaffenheit des Pulses, der sich durch zunehmende Beschleunigung bei geringer und immer schwächer werdender Spannung der Gefäßwand auszeichnet. Durch die Regelmäßigkeit seiner Veränderung kann er in zweifelhaften Fällen allein ausschlaggebend sein und verdient daher die ebenso zweckmäßige, wie bezeichnende Benennung des Peritonealpulses in vollem Maße. Auf die besonders üble Vorbedeutung seines Mißverhältnisses zur Temperatur ist oben hingewiesen. Erwähnt sei noch, daß nach Kümmel bei Perforation des Appendix gleich dem Abfall der Temperatur auch eine vorübergehende Verlangsamung des Pulses auftreten kann, die dann aber gleich dem folgenden Temperaturanstieg einer zunehmenden Beschleunigung des Pulses Platz macht.

Übelkeit und Erbrechen nimmt zunächst entsprechend dem stärkeren Peritonealreiz an Häufigkeit und Heftigkeit zu, kann aber

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im weiteren Fortschritt der Erkrankung nach Sprengel schließlich aufhören. Diese Erscheinung ist wohl ebenfalls als Folge einer toxischen Schädigung bzw. Lähmung der sensiblen Nervenfunktionen aufzufassen, welche vermutlich ebensowenig den zentripetalen Reiz nach dem Brechzentrum zu übermitteln in der Lage sind, wie die motorischen Nerven den zentrifugalen Reiz bei der schwindenden Peristaltik der Därme. Daß in schweren Fällen diese Schädigung bereits sehr früh einsetzen und daher auch das Erbrechen fast gänzlich in Fortfall kommen kann, liegt auf der Hand. Auf die besonders üble Vorbedeutung des Blutbrechens ist bereits oben hingewiesen. Es verdankt seine Entstehung bisweilen außerordentlich zahlreichen punktförmigen Blutungen auf der Magenschleimhaut, welche von Nietzsche und Dieulafoy auf die Wirkung mit dem Magensaft ausgeschiedener Toxine zurückgeführt werden. Ob diese Erklärung für alle Fälle zutreffend ist, muß dahingestellt bleiben, daß der Erscheinung in jedem Falle rein toxische Ursachen zugrunde liegen, ist wohl zweifellos.

Der bei der Periappendicitis incipiens bereits erwähnte, sehr frühzeitig einsetzende lokale Meteorismus führt auch hier schnell zu der Kantenstellung der Leber, so daß das Frühzeichen des Leberhochstandes naturgemäß für diese Fälle erhöhte Geltung behält.

Entsprechend der toxischen oder auch entzündlichen Schädigung der motorischen Nerven durch eine Lymphangitis des Mesenteriums kommt es dann schnell zu einer fortschreitenden Lähmung mit nachfolgenden Meteorismus sämtlicher Darmschlingen. Da nun mit der fortschreitenden Lähmung der motorischen und sensiblen Nerven auch die Bauchdeckenstarre entsprechend nachläßt, so werden schließlich die einzelnen geblähten, meist unbeweglichen Darmschlingen durch die schlaff gewordenen Bauchdecken deutlich sichtbar, und wir haben dann das typische Bild des paralytischen oder dynamischen Ileus vor uns. Solange dieser Ileus auf einzelne tiefer gelegene Darmbezirke beschränkt ist, wird er sich gleichzeitig durch periodisches, grünlich-galliges, später kotiges Erbrechen kenntlich machen. Bei weiterem Fortschreiten der Lähmungserscheinungen hört dieses ebenfalls auf und allein die völlige Verhaltung von Stuhl und Winden weist neben dem meist vorhandenen allgemeinen Meteorismus auf das Versagen der Darmtätigkeit hin.

Fälle in diesem Stadium lassen die Operation als aussichtslos erscheinen, da die allgemeine Intoxikation des Körpers dann bereits zu sehr fortgeschritten ist. Es ist daher nicht Sache des Operateurs, der solche Fälle noch operiert um dem Kranken die letzte Rettungsmöglichkeit zu bieten, wenn sie trotzdem tödlich verlaufen, sondern

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Sache des Arztes, der den Fall zu spät der rettenden Hand des Chirurgen überliefert.

Die Leukozytose zeigt auch hier ein der Temperatur ziemlich parallel laufendes Verhalten. Sie weist eine deutliche Erhöhung auf, solange der Körper noch verteidigungsfähig ist. Der Abfall der Leukozytose ist stets als Zeichen beginnender Allgemeininfektion aufzufassen und spricht bei dem Fortbestehen der übrigen schweren Erscheinungen gleich dem Temperaturabfall für einen ungünstigen Ausgang der Erkrankung. Demgemäß ist eine hohe Leukozytose bei klinischer Annahme einer Allgemeininfektion für den weiteren Verlauf der Erkrankung bzw. für die Heilungsaussichten auf operativem Wege als günstiges Vorzeichen aufzufassen. In ganz schweren Fällen wird gleich der Temperatur auch die Leukozytose von Anfang an versagen und kann dann gänzlich in Fortfall kommen. Erwähnt sei noch, daß Russel in normalen Leukozytenwerten bei Kranken mit schweren Allgemeinerscheinungen einen sicheren Hinweis auf eine stattgehabte Perforation erblicken will.

Im Urin kann nach Lennander vereinzelt eine geringe Albuminurie auf eine allgemeine Infektion bzw. Intoxikation hinweisen, gleichwie bisweilen Indikan als Folge eines tiefsitzenden dynamischen Ileus nachweisbar sein kann. Beide Merkmale können wegen der Seltenheit und des späten Auftretens derselben für die Erkennung vom Gesichtspunkt der Therapie aus nur untergeordnete Bedeutung beanspruchen.

Desgleichen kann es in vereinzelten Fällen zu allgemeinem Ikterus kommen, der auf hämatogener Grundlage entstanden, als Zeichen schwerer Allgemeinintoxikation stets von übler Vorbedeutung sein wird.

Schließlich möchte ich neben der bekannten Facies abdominalis, die je nach der Schwere des Falles früher oder später auftreten wird, und neben der Trockenheit der Zunge noch auf eine Injektion der Konjunktiven hinweisen, die mir bisweilen am Beginn schwerer Allgemeininfektionen des Peritoneums aufgefallen ist, selbst in Fällen, wo die Temperatur, die Spannung der Bauchdecken und im ersten Beginn auch der Puls in gewissem Grade im Stich ließ.

Kurz zusammengefaßt ist also für die Erkennung der Allgemeininfektion des Peritoneums vom Appendix aus auf folgende Merkmale das Augenmerk zu richten:

1. Peritonealpuls mit zunehmender Beschleunigung unter dauernder Abnahme der Spannung der Gefäßwand.

2. Hoher Temperaturanstieg mit atypischem Verlauf, Differenz zwischen Achselhöhlen- und Mastdarmtemperatur.

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3. Trockenheit der Zunge, bisweilen Injektion der Konjunktiven.

4. Spontane Schmerzhaftigkeit des ganzen Leibes namentlich bei Bewegungen und vornehmlich solchen des rechten Beines.

5. Starke Druckempfindlichkeit des ganzen Leibes, namentlich in der Ileocöcalgegend.

6. Reflektorische Bauchdeckenstarre beiderseits.

7. Beschleunigter, vorwiegend oder rein kostaler Atmungstypus.

8. Fehlen der Leberdämpfung in der rechten Mammillarlinie.

9. Übelkeit mit periodischem, heftigen Erbrechen.

10. Örtlicher, später allgemeiner Meteorismus, dynam. Ileus.

12. Facies abdominalis.

13. Albuminurie, Indikanurie in seltenen Fällen.

14. Hämatogener Ikterus bei schwerer Intoxikation.

15. Vereinzelt Schwellung und schmerzhafte Vorwölbung der vorderen Rektalwand.

In ganz schweren Fällen kann fast reaktionslos, allein unter entsprechender Veränderung des Pulses schneller Verfall und Tod eintreten.

Kommen wir schließlich noch zur Erkennung der chronischen Periappendizitis, der Periappendizitis im Sicherheitsstadium, so wird für diese in der überwiegenden Mehrzahl der Fälle die vorangegangene Anamnese bereits entscheidend sein. Die subjektiven Beschwerden sind meist geringer Natur und äußern sich in mangelhafter Verdauung bzw. Neigung zu chronischer Obstipation, bisweilen unterbrochen von plötzlichen Durchfällen nach Diätfehlern oder auch spontan, ferner in einem Gefühl von Druck und Völle in der Ileocöcalgegend, sowie leichtem Meteorismus der rechten Bauchseite. Objektiv ist vielfach gar nichts, in vielen Fällen jedoch die Reste früherer Entzündungen in Gestalt einer undeutlichen Resistenz in der Ileocöcalgegend nachweisbar, die bisweilen auf Druck, seltener auch spontan etwas schmerzhaft ist.

Wirklich heftige und sogar das Leben bedrohende Beschwerden können in vereinzelten Ausnahmefällen einsetzen, wenn es durch Abknickung und Abschnürung von Dünndarm- oder Dickdarmschlingen auf Grund alter Verwachsungen zu einem rein mechanischen Ileus kommt, der sich dann meist durch entsprechende Unruhe, Steifung und Auftreibung der höher gelegenen Darmschlingen bei gleichzeitigem Fehlen eines Temperaturanstiegs leicht in seinem ursächlichen Zusammenhang erkennen läßt.

Derartige Fälle von chronischem Adhäsionsileus hat Federmann in einer Anzahl von 6 auf 500 Appendizitisoperationen gefunden, was einem Prozentsatz von 1,2 % entsprechen würde. Er weist

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darauf hin, daß der Stuhlgang bei diesen Fällen lange Zeit erhalten sein kann, und rät prophylaktisch zur frühzeitigen Eröffnung von periappendizitischen Abszessen zwecks Verhütung größerer Adhäsionen.

Selbst 10 Jahre nach einer überstandenen Periappendizitis hat Subbotitsch noch einen derartigen Fall von Ileus beobachten können, der infolge Abschnürung einer Darmschlinge durch einen alten peritonitischen Strang entstanden war. Auch in der Breslauer Klinik ist unter Geheimrat Garrè ein ähnlicher Fall zur Operation gelangt, der in der nachfolgenden Zusammenstellung der operativen Fälle eine entsprechende Würdigung gefunden hat.

Die Vermeidung derartiger, wenn auch seltener, so doch um so schwererer Folgeerscheinungen dürfte einen Grund mehr bilden zur prinzipiellen Vornahme der Frühoperation oder, wenn die Patienten erst später zur Behandlung gelangen, zum sofortigen Eingriff auch im Zwischenstadium, um damit Abszeß- und Adhäsionsbildung möglichst von vornherein zu verhindern.

Für die Differentialdiagnose der Periappendizitis möchte ich mich an dieser Stelle nur mit einigen kurzen Hinweisen begnügen, da es ja auf der Hand liegt, daß bei der Verlagerungsmöglichkeit des Appendix dafür in vereinzelten Fällen fast jedes Organ der rechtsseitigen, ja sogar der gesamten Bauchhöhle in Betracht kommen kann.

Hinsichtlich der Nierenerkrankungen wird bei Wanderniere, Nierensteinen und Nierentumor in der Regel das Fehlen der Temperatursteigerung vor Verwechselungen schützen, und wenn neuerdings die am falschen Orte festgewachsene Niere nach Riedel bisweilen Fieber machen kann, so dürfte das doch nur für außerordentlich seltene Ausnahmefälle in Betracht zu ziehen sein. Bei Nierensteinen, Tuberkulose und Tumor der Niere werden gelegentliche Blutungen im Urin vielfach das erste Anzeichen der Erkrankung bilden. Bei Pyelitis und entzündlichen Erkrankungen der Niere wird der mikroskopische und chemische Urinbefund die Unterscheidung leicht ermöglichen, abgesehen von den zystoskopischen Untersuchungsmethoden, die ja schließlich dem praktischen Arzt nicht ohne weiteres zugänglich sind. Bei einer großen Mehrzahl der Nierenerkrankungen ist ferner ganz besonders auf die eigenartigen, in dem Ureter der erkrankten Niere zur Blase ausstrahlenden Sensationen zu achten, die in den meisten Fällen den Patienten auch als solche zum Bewußtsein gelangen und auf Befragen sei es als schmerzhafte, sei es als eigenartige Empfindungen angegeben werden. Schließlich sei noch auf den vielfach deutlich gürtelartigen Charakter der Schmerzempfindung

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hingewiesen, der gerade bei Nierenerkrankungen häufig in stär
oder geringerem Grade in die Erscheinung zu treten pflegt.

Hinsichtlich der Unterscheidung von Leberaffektionen se
kurz auf das Ausstrahlen der Schmerzempfindung in den Rücke
vornehmlich die rechte Schulter, auf die meist nachweisbare
größerung oder Druckempfindlichkeit der Leber bzw. Gallenblas
den meist nach der Einnahme von Speisen einsetzenden un
Abend nach dem Schlafengehen periodisch sich steigernden Cha
der Beschwerden, auf die Verschieblichkeit der etwa fühlbare
sistenz mit der Atmung, auf den vielfach ausgesprochenen V
willen gegen Fleischnahrung, auf ev. Ikterus, acholischen Stuh
Hautjucken hingewiesen, alles Merkmale, deren teilweises od
samtes Vorhandensein eine Klärung des Krankheitsbildes in der
nicht allzu schwierig gestalten dürfte.

Die rechtsseitigen Adnexaffektionen bei Frauen werde
vielfach bereits anamnestisch durch voraufgegangene Menstrua
störungen, sowie den Nachweis vorangegangener Genitalinfekti
kennen lassen. Klinisch wird hierfür als besonderes Unterschei
merkmal das oben erwähnte Blumbergsche Symptom von s
Autor empfohlen, wofür jedoch weitere Bestätigungen auße
Ewald z. Z. noch ausstehen. Tatsächlich können diese Fälle kl
noch am ehesten zu Fehldiagnosen Anlaß geben, wobei alle
nicht zu vergessen ist, daß bei der nahen Nachbarschaft der
Organe eine Kombination von Appendizitis mit rechtsseitiger A
erkrankung nicht gerade als Seltenheit zu betrachten ist.

Daß im übrigen vereinzelt eine fieberhafte Enteritis und
Typhus zu Fehldiagnosen führen kann und auch geführt ha
noch nebenher erwähnt. Bei ersterer werden entsprechende s
mige oder zellige Beimengungen im Stuhl, bei letzterem die bakt
Diagnose vor Verwechslungen schützen können. Daß ander
gerade auf der Grundlage einer infektiösen Enteritis sich äußers
artige Erkrankungen des Appendix entwickeln können, ist oben
des näheren hervorgehoben worden.

Schließlich sei der Vollständigkeit halber noch an die Ma
und Pankreasaffektionen gedacht, die gleichfalls bisweilen
Zweifel in der Diagnose entstehen lassen können, aber in der
durch den ausgesprocheneren und lokalisierteren Charakter de
schwerden für die Unterscheidung von Appendixaffektionen
allzu großen Schwierigkeiten bieten werden.

Wenden wir uns nun der

Therapie der Appendizitis

zu, so liegt es in der Natur der Sache, daß wir uns der he

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Anschauung entsprechend vornehmlich mit der chirurgischen Seite derselben zu beschäftigen haben werden. Hierbei wird es sich zunächst um die Indikationsstellung und Prognose nach der Anschauung der einzelnen Autoren in den verschiedenen Stadien der Erkrankung handeln und wir werden uns daher am besten unserer vorausgegangenen Einteilung gemäß im einzelnen mit der Früh-, Zwischen-, Spät-, Not- und Sicherheitsoperation zu beschäftigen haben.

Von diesen allen steht heute im Brennpunkt des Interesses die Frage der absoluten Frühoperation und der Zwischenoperation, während bezüglich der Anschauung über die Zweckmäßigkeit der Spät-, Not- und Sicherheitsoperation wesentliche Differenzen unter den Autoren kaum noch zu verzeichnen sind. Es erscheint daher gerechtfertigt, wenn wir über die ersteren beiden Operationen die Ansichten der Autoren einer etwas eingehenderen Würdigung unterziehen, wobei manche Änderung derselben nach der radikaleren Richtung hin auch bei den sich zuerst ablehnend verhaltenden Klinikern im Laufe der letzten Jahre namentlich hinsichtlich der Frühoperation zu verzeichnen ist.

So macht Kümmel, der sich vorher abwartend verhielt und nur im äußersten Notfall eingriff, seit März 1904 wegen des stets unberechenbaren Verlaufes der Erkrankung prinzipiell die Frühoperation, sobald nach 12—24 Stunden die Erscheinungen nicht geschwunden sind oder sich gesteigert haben. Er steht auf dem präzisen Standpunkt, jede als solche erkannte Appendizitis zu operieren und zwar je eher, je besser.

Interessant ist auch die Änderung im Standpunkt von Sonnenburg, wenn er auch nicht ganz so entschieden, wie die anderen Autoren für die Frühoperation eintritt. Er versuchte bis 1905 nach Möglichkeit die Operation stets in das Sicherheitsstadium zu verlegen. Noch 1905 führt er gegen die Frühoperation den etwas theoretischen Einwurf an, daß bei hochgradiger Infektion und Toxinwirkung selbst die früheste Operation versagen kann, und daß es zum mindesten zweifelhaft ist, ob die Frühoperation immer eine schnellere Heilung der Krankheit, wie ein später vorgenommener Eingriff erzielen kann. Trotzdem ist er seit diesem Zeitpunkt zwar nicht zum ausgesprochenen Anhänger der Frühoperation geworden, betätigt aber dennoch in der Praxis die Gepflogenheit, alle nicht ganz leichten Fälle innerhalb der ersten 48 Stunden zu operieren. Merkwürdigerweise glaubt er diese Erweiterung seiner Indikationsstellung zum operativen Eingriff auf eine Änderung des Materials zurückführen zu sollen, das ihm in den letzten Jahren zugegangen ist.

Helferich hält bedingsweise die Frühoperation für berechtigt, da

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es besser sei, zu sagen, der Patient wäre auch ohne Operati
rettet worden, als der Patient wäre noch durch die Operat
retten gewesen.

Sprengel operiert jede Appendizitis, sobald nicht innerha
ersten 24 Stunden ein entscheidender Rückgang aller Sympto
folgt, und zwar in jedem Stadium.

Rehn hält bereits 1901 als einer der ersten neben Spreng
Entfernung des Appendix im akuten Anfall für das einzig
Mittel, die Mortalität der Erkrankung auf ein Minimum he
drücken, indem er ganz besonders hervorhebt, daß die Operat
Anfall ebenso leicht, oft leichter und sicher nicht gefährlicher
Sicherheitsstadium ist. Auch 1905 tritt er wiederum warm f
Frühoperation ein, indem er den akuten Anfall nur für eine
der meist chronischen Erkrankung erklärt, deren Blitz nicht nur
sondern auch zünden kann, ohne daß man sagen kann, was da
Flammen aufgeht. Als besonderen Vorzug der Frühoperation
er die sichere und radikale Heilung unter Vermeidung späterer
zustände.

Karewski hält die frühzeitige Entfernung des Appendix eb
für das einzig rationelle und ist daher dafür, in jedem Falle so
operieren. Da man den Verlauf der Appendizitis nie mit Sic
voraussagen kann, erscheint ihm die Appendektomie angezeigt,
irgendwelche Krankheitserscheinungen mit Sicherheit auf diese
des Darmtraktus hinweisen, und zwar möglichst sofort nach
Übergreifen der Erkrankung auf das Peritoneum. Wenn möglic
man sogar auch bei leichteren Beschwerden noch vor dem
also gewissermaßen vor dem Übergreifen der Entzündung au
Peritoneum operieren, eine Forderung, auf die wir später noch
zurückkommen werden.

Nach v. Eiselsberg ist die Frühoperation in allen rechtze
Behandlung kommenden Fällen auszuführen. Den gleichen
punkt vertritt naturgemäß auch sein Assistent Haberer, we
andererseits auch die Frühoperation nicht für ebenso ungefä
halten möchte, wie die Sicherheitsoperation: „Alles in allem
Frühoperation sicher die idealste Behandlungsmethode der Peri
dizitis, weil sie, ohne die weitere Entwicklung des Prozesses
warten, denselben mit einem Male abschneidet."

Lennander hält es gleichfalls für den Kranken am besten,
er im Verlauf der nächsten Stunden zur Operation gelangt,
die Diagnose auf akute Appendizitis gestellt ist und der ge
Grund zur Beunruhigung vorhanden ist. Namentlich bei dem z

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Anfall rät er zum Eingriff, da die Diagnose dann klar ist und der Anfall damit sofort abgeschnitten wird.

Carl Beck ist ebenfalls dafür, lieber früher, wie zu spät zu operieren und hält für die sicherste Therapie die frühzeitige Entfernung des Appendix, besonders da die Operation als solche gänzlich ungefährlich ist.

Riese führt als weiteren Vorzug der Operation an, daß den Patienten im Schmerzanfall der Entschluß zur Operation wesentlich leichter fällt, während sich manche in dem schmerzfreien Sicherheitsstadium nachträglich eines anderen besinnen und die Operation immer weiter hinausschieben, bis der nächste schwere Anfall sie in die Hände des Chirurgen zurücktreibt.

Riedel hält außer bei ganz leichten Fällen die absolute Frühoperation, womöglich bereits 5—10 Stunden nach dem Beginn des Anfalls für weitaus am besten.

Payr ist für die absolute Frühoperation, da man entgegen der Ansicht von Sonnenburg aus dem klinischen Bilde nie mit Sicherheit auf den anatomischen Befund schließen kann, und rät besonders bei medianer oder nach unserer Benennung mesocöliacaler Lage des Appendix zwischen losen Darmschlingen möglichst frühzeitig einzugreifen.

Dörffler ist von der Überlegenheit der Frühoperation fest überzeugt und glaubt damit dem Patienten das Sicherste zu raten. Als einen besonderen Vorzug der Frühoperation führt er an, daß sie in einem Zeitpunkt stattfindet, wo das Peritoneum noch widerstands- und leistungsfähig ist, daß man dabei eine leichtere Übersicht des Operationsfeldes und damit auch eine leichtere Entfernung des Appendix hat. Er ist überzeugt, daß mit dem Augenblick der Frühoperation in allen nicht ganz leichten Fällen die Mortalität der Appendizitis auf 0% herabsinken wird.

Tilmann empfiehlt die Frühoperation bei allen vorher gesunden, mit akutem Erbrechen erkrankten Appendizitispatienten, während er bei chronischen und rezidivierenden Formen nur im Falle einer akuten Verschlimmerung eingreift und sonst im Sicherheitsstadium operiert.

Müller (Hildebrand) drückt sich etwas gewunden aus, wenn er sagt, daß jede Periappendizitis dem Chirurgen gehört, ob sie operiert werden muß oder nicht, daß ferner am besten eine exakte Beobachtung und möglichst frühe, aber nicht Frühoperation sei, denn die letztere könne zwar manches Leben retten, aber auch manches zerstören, eine Behauptung, für welche Müller in ihrem Nachsatz wohl schwerlich Beweise erbringen kann. Namentlich warnt er vor

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der Frühoperation bei bestehender Nephritis, die er für sehr
lich hält und von der vielfach Todesfälle in der Literatur be
würden, eine Frage, auf die wir bei der zusammenhängend
örterung der Gegenindikationen der Frühoperation noch näher
kommen werden.

Körte hält die Frühoperation in 48 Stunden nur in w
schweren Fällen für angezeigt, ein etwas dehnbarer Begriff,
von dem klinisch projizierten Krankheitsbild des Kranken,
eventuell gänzlich verschiedenen anatomischen Befund und der
individuellen Beurteilung des mehr oder minder erfahrene
wissenschaftlich durchgebildeten Arztes abhängig ist, und so
seinen Grenzen kaum festgelegt werden kann.

Gussenbauer meint kurz und treffend, man müsse mehr
Leben des Patienten, denn um seinen Wurmfortsatz besorgt s

Hahn hält im Gegensatz zu der etwas pessimistischen Ansc
Sonnenburgs, daß bisweilen auch die früheste Operation ve
könne, nach eigener 10jähriger Erfahrung dafür, daß die Op
im frühesten Zeitpunkt und bei guter Dränage selbst bei einget
Perforation ebenso gut ist, wie ohne eine Perforation, da er
nicht einen Todesfall hatte. Entscheidend dafür ist naturgem
möglichst geringe Zeitraum zwischen dem Perforationsvorgan
der nachfolgenden Operation.

Borelius hebt als einen weiteren Vorzug der Frühoperatio
vor, daß man innerhalb 48 Stunden stets primär nähen kann,
wenn der Appendix gangränös aussieht und sich freies Exsu
der Bauchhöhle vorfindet, da dieses in der Regel rein serös un
als steril zu bezeichnen ist.

Landau rät auch bei gleichzeitigen Frauenleiden möglich
48 Stunden zu operieren.

Stanton führt als einen Grund mehr für die Frühoperati
daß man am ersten Tage des Anfalls bereits die Erkrankung
allein auf die Schleimhaut des Appendix beschränkt, sondern i
Schichten der Wand verbreitet vorfinden könne.

Hoffmann (Küster) operiert ebenfalls, sobald bei der typ
Erkrankung das Fieber länger als 36 Stunden hoch bleibt.

Von weiteren Vertretern der prinzipiellen Frühoperation i
nicht ganz leichten, bzw. in 48 Stunden zurückgehenden Fälle
noch genannt: Mynter, Lanz, Bloß, Wette, Dieulafoy, C
W. Meyer-New York, O. Hartmann, Legueu, Poirier, Se
Chaput, Championnière, Routier, Tuffier, Russel, D
Roux, Rotter, Schnitzler, v. Mosettig, S. Perman,
Holmes, Ochsner, v. Bergmann, A. C. Bernays, F.

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G. Heaton, W. Hagen, Trendelenburg, Ceccherelli, Kleman, Webster und Dowd.

Dieser kurze Literaturüberblick, der im übrigen durchaus keinen Anspruch auf Vollständigkeit erheben will, dürfte immerhin bereits zur Genüge beweisen, eine wie große Übereinstimmung unter der überwiegenden Mehrzahl der Autoren über den unbedingten Wert der absoluten Frühoperation herrscht, mit dem einzigen mehr individuellen Unterschied, daß der eine sie bei etwas leichteren, der andere sie bei etwas schwereren Erscheinungen bzw. bei fehlender Rückbildung derselben in den ersten 24—48 Stunden zum Prinzip erhebt. Allen gemeinsam ist jedenfalls die Erkenntnis und das Bestreben, dem Patienten, sobald die Krankheit als solche erkannt ist, durch eine möglichst frühzeitige und gefahrlose Ausschaltung der Causa nocens eine sichere und schnelle Heilung zu garantieren, ehe man ihn den unsicheren und niemals vorherzusehenden Zufällen des weiteren Krankheitsverlaufes aussetzt.

Den radikalsten Standpunkt von allen vertritt zweifelsohne Karewski, wenn er rät, die Operation wenn möglich noch vor dem Anfall zu machen, ein Vorschlag, der wegen allzu großer Möglichkeit eventueller Luxusoperationen auf eine prinzipielle Annahme von irgendwelcher Seite wohl kaum jemals zu rechnen haben dürfte. Immerhin darf man sich aber nicht verhehlen, daß man bisweilen dennoch heute vor die Frage gestellt werden kann, in gewissem Sinne vor dem Anfall zu operieren, vorausgesetzt, daß man unter dem eigentlichen Anfall das übliche Bild der Periappendizitis versteht, eine Auffassung, die wohl auch der Ansicht von Karewski entspricht, wenn er ursprünglich zum sofortigen Eingriff bei den ersten Anzeichen des Übertrittes der Erkrankung auf das Peritoneum rät. Es kommen nämlich heute tatsächlich gar nicht so selten Fälle vor, in denen allzu ängstliche und vorsichtige Patienten, die eine oder mehrere bisweilen ganz kurze, vielleicht nur 1—2 Stunden dauernde Appendikularkoliken ohne jedes Fieber durchgemacht haben, an den Chirurgen mit der Frage oder mit dem direkten Verlangen herantreten, sie von dem vermutlichen Friedensstörer zu befreien, ehe er ihnen zur Quelle eines ernsten Erkrankungszustandes wird. Dabei ist zu beachten, daß bei der heute fast allgemein verbreiteten Kenntnis von der Bedeutung und Lage des sog. Blinddarms im Publikum fast jeder Schmerz von kolikartigem Charakter in der rechten Unterbauchgegend als Blinddarmentzündung, und vielfach gar nicht mit Unrecht verdächtigt wird. Daß in solchen Fällen eine nachträgliche objektive Untersuchung kein positives Ergebnis zu liefern braucht und daher absolut nicht als Gegenbeweis von tatsächlich vorausgegangenen Appendikularbe-

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schwerden aufgefaßt werden darf, liegt auf der Hand. Vielfac man allerdings vielleicht eine gewisse Bestätigung dafür durc behandelnden Arzt erhalten können, wenn eben während des ein Arzt dabeigewesen ist, was bei der Kürze der Schmerzdaue aus Mangel an Zeit oder aus anfänglicher Indolenz seitens d tienten nicht der Fall zu sein pflegt.

Ich pflege mich in solchen Fällen damit zu behelfen, daß i bald die Patienten am Ort sind und im Falle eines richtige appendizitisanfalles jederzeit sofort in chirurgische Behandlung k können, es ihrem eignen Ermessen freistelle, sich später bei d ringsten Fiebererscheinungen mit entsprechenden Beschwerd sofortigen Operation einzufinden oder auf ihren eigenen Wuns die Operation sofort vornehmen zu lassen. Denn es ist men durchaus verständlich, daß vereinzelte Patienten, ehe sie dauer Damoklesschwert eines nach mehreren Appendikularkoliken nur ausbleibenden Periappendizitisanfalles mit der Notwendigkeit operativen Eingriffs über sich fühlen wollen, sich lieber ein fortigen Operation zwecks sicherer Ausschaltung einer der Möglichkeit unterziehen, die dann fast ausnahmslos durch die skopisch nachweisbaren Veränderungen am Appendix, wie Ko Schwellung und Injektion der Mukosa oder der ganzen Wa Knickungen, Retention des Inhaltes u. dgl. den Beweis erbringe und auch bei uns tatsächlich im Laufe der letzten Jahre erbra daß die Gefahr einer Luxusoperation in derartig vereinzelten nicht allzuhoch einzuschätzen ist. Anders liegt in derartigen schon die Sache, wenn die Patienten auf dem Lande wohnen größere Reise vorhaben, kurz, wenn sie sich in Lebensverhäl befinden, wo sie nur schwer oder gar nicht sich jederzeit di des Chirurgen zugänglich machen können. Da erscheint es si bereits in diesem Stadium, also gewissermaßen noch vor dem lichen Anfall, zur Operation zu raten, da man nicht wissen unter welchen Umständen sie später einem etwa eintretend möglicherweise recht schweren Anfall ausgesetzt sind. Es sind eben Fälle, die individuell beurteilt sein wollen und in dene ausnahmsweise aus den Lebensverhältnissen oder der ausgespro Willensäußerung der Patienten heraus die Operation berecht scheinen kann. Derartige Fälle werden sich sicher auch bei a Chirurgen vorfinden, sobald sie ihr Material danach sichten v Daraus nun aber die Forderung einer prinzipiellen Operatio vor dem Anfall herzuleiten, liegt eine Veranlassung nicht vo lange man sicher ist, daß einem Patienten durch die Ablehnung Forderung kein Schaden erwachsen kann.

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Um noch kurz die Indikationsstellung für die Frühoperation der Periappendizitis bei gleichzeitiger Gravidität zu streifen, so sind sämtliche Autoren sich darin einig, daß die Gravidität an sich eine erhöhte Indikation zum möglichst frühzeitigen operativen Eingriff bildet, da sie in der Regel einen sehr ungünstigen Einfluß auf den Verlauf und die Prognose der Erkrankung im Sinne einer wesentlich erhöhten Gefahr der peritonealen Allgemeininfektion ausübt. Rosner geht deswegen so weit, die Operation sogar in denjenigen Fällen anzuraten, die nicht ganz sicher sind und einer Periappendizitis nur verdächtig erscheinen. Ebenso raten Riese, Labhardt, G. Heaton u. a. zum möglichst frühzeitigen Eingriff in derartigen Fällen mit Rücksicht auf die erhöhte Infektionsmöglichkeit des Peritoneums. Kümmel allein rät, wenn es nicht möglich ist, frühzeitig nach Beginn der Erkrankung zu operieren, lieber bis zum Sicherheitsstadium zu warten, immerhin ein etwas riskanter Vorschlag, da eben bis zu diesem Stadium die Patienten dauernd der Gefahr einer Allgemeininfektion des Peritoneums ausgesetzt bleiben. Gerade hier dürfte vielmehr stets eine möglichst weitgehende Indikationsstellung zum operativen Eingriff, sobald die Patientin in chirurgische Behandlung gelangt, am empfehlenswertesten sein.

Als Gegenindikationen werden bezüglich der Frühoperation von Ball besonders Tuberkulose, Herzfehler und Nierenerkrankungen hervorgehoben. Namentlich bei letzteren glaubt auch Müller ganz besonders vor einem operativen Eingriff im akuten Stadium warnen zu müssen. Treten wir der Frage etwas näher, so werden wir bei schweren Herzfehlern oder Nierenerkrankungen der Anschauung Balls ohne weiteres beipflichten können, da hier der operative Eingriff an sich gewissermaßen der Tropfen sein kann, welcher des Maß der aufs äußerste herabgesetzten Lebens- und Leistungsfähigkeit des Körpers zum Überfließen bringt. Für die leichteren und mittelschweren Fälle kann man aber Ball darin doch nicht ganz beistimmen, insofern gerade Patienten mit Herz- oder Nierenerkrankungen um so eher vor den üblen Zufällen der Periappendizitis zu bewahren sind, als der an sich schon in seiner Leistungsfähigkeit herabgesetzte Körper um so schwerer mit einer Peritonealinfektion fertig werden und dann um so leichter einer Allgemeininfektion des Peritoneums mit Herz- oder Niereninsuffizienz erliegen wird. Man könnte daher in nicht allzu schweren Herz- oder Nierenerkrankungen bei gleichzeitiger Periappendizitis eher eine Indikation mehr zur möglichst frühzeitigen Ausschaltung der Causa nocens zwecks Verhütung weiterer Komplikationen erblicken, welche für den an sich in seiner Widerstandskraft erheblich herabgesetzten Patienten eine wesentlich größere Gefahr

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involvieren können, als die vitale Schädigung, welche d
dauernde und ungefährliche operative Eingriff ev. unter Äthe
oder Lumbalanästhesie mit sich bringt. Auch hier wird es s
mindesten dringend empfehlen, zu individualisieren und nic
weiteres in jeder Herz- oder Nierenerkrankung eine absolute In
gegen einen möglichst frühzeitigen operativen Eingriff zu e
da die Zufälle des Krankheitsverlaufes wesentlich schlimmere
als der operative Eingriff an sich zeitigen können.

Fassen wir nun zum Schluß noch einmal die Gründe,
zugunsten der Frühoperation innerhalb der ersten 48 Stunden s
kurz zusammen, so haben wir dieselben in folgendem zu e

1. Gegenüber dem stets unberechenbaren Verlauf der Pe
dizitis stellt die Frühoperation das einzige Mittel dar zur s
Beendigung des Krankheitsprozesses und zur sicheren, schne
dauernden Wiederherstellung des Patienten.

2. Die Frühoperation allein vermag dem Patienten mit S
die Vermeidung sämtlicher Früh- oder Spätfolgezustände d
appendizitis, wie Thrombosen, Embolien, metastatische A
Pleuriten einerseits, sowie Adhäsionsbeschwerden und Narbe
andererseits in Aussicht zu stellen.

3. Die Frühoperation ist für den Patienten ebenso ung
und für den Operateur vielfach leichter, wie die früher a
bevorzugte Sicherheitsoperation, da sie wegen des Fehlens
häsionen fast stets eine gute Übersicht des gesamten Operatio
und somit dem Chirurgen die Möglichkeit gewährt, mit
Schnitten auszukommen.

4. Die Frühoperation hat gegenüber der Spätoperation de
einer radikaleren und schnelleren Heilung für den Patienten
einerseits stets die radikale Entfernung des Appendix und
seits fast ausnahmslos den primären Verschluß der Bauchwun
Naht gestattet. Selbst bei Einlegen eines kleinen Sicherheits
in zweifelhaften Fällen wird die Heilung immer noch schn
sich gehen, als wenn man zur Inzision von Abszessen gezwu
nach welcher der Patient in der Regel den Appendix behält
meist noch einer weiteren Operation zur Entfernung desselbe
ziehen muß.

5. Der Entschluß zur Operation fällt dem Patienten w
leichter im Schmerzanfall selbst, als später in dem schmerz-
schwerdefreien Sicherheitsstadium.

In Erwägung dieser zahlreichen und unbestreitbaren Vor
absoluten Frühoperation in den ersten 48 Stunden erscheint
nicht nur berechtigt, sondern dringend geboten, den Praktike

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wieder darauf hinzuweisen, daß er in der Frühoperation das sicherste Mittel in der Hand hat zur schnellen und dauernden Wiederherstellung seiner Patienten in allen Fällen, bei denen nicht in 24 Stunden ein völliger oder zum mindesten doch deutlich merkbarer Rückgang sämtlicher Erscheinungen zu verzeichnen ist.

So groß nun die Übereinstimmung der Autoren über die Indikationsstellung und Zweckmäßigkeit der Frühoperation ist, so groß ist andererseits zurzeit noch die Divergenz der Anschauungen über die Vorzüge und Nachteile der

Zwischenoperation.

Es ist dieses so ziemlich das einzige Gebiet der Therapie, auf dem heute noch nicht eine gewisse Übereinstimmung hat erzielt werden können. Wie oben bereits erwähnt, verstehen wir unter Zwischenoperation diejenige Operation, welche zeitlich in das Zwischenstadium fällt, das vom 3.—8. Tage der Erkrankung gerechnet wird und demjenigen Zeitraum entspricht, welchen das Peritoneum erfahrungsgemäß braucht, um durch feste Verwachsungen eine sichere Umgrenzung und Abkapselung des Entzündungsherdes entstehen zu lassen. Der Streit der Meinungen dreht sich nun darum, ob in diesem Zeitraum der operative Eingriff an sich durch die leichtere Lösung der noch frischen Adhäsionen und nachfolgende Infektion des freien Peritoneums einerseits oder die Möglichkeit einer spontanen Verschlimmerung und Allgemeininfektion des Peritoneums bei exspektativem Verhalten andererseits die größere Gefahr für den Patienten darstellt.

Die Gegner der Zwischenoperation wie Sonnenburg, Körte, v. Eiselsberg, Haberer, v. Bergmann, Lanz, Büngner, Israel, Nordmann, Holmes, A. C. Bernays, W. Hagen, Landau, Treves u. v. a. vertreten naturgemäß den ersteren Standpunkt. Sie halten die Gefahr einer Lösung der Adhäsionen mit nachfolgender Allgemeininfektion des Peritoneums in dem Zwischenstadium für so groß, daß nur eine heftige Verschlimmerung der Erkrankung bzw. die drohende Gefahr des spontanen Eintritts einer Allgemeinperitonitis das Risiko eines derartig gefährlichen Eingriffs zu rechtfertigen vermag. Sie suchen daher prinzipiell alle Fälle nach 48 Stunden ins Sicherheitsstadium oder zum mindesten ins Spätstadium überzuführen, um dann die etwa notwendige Spaltung der nunmehr sicher abgekapselten Abszesse vorzunehmen.

Allerdings stellen auch einige der prinzipiellen Gegner der Zwischenoperation die Indikation zum Eingriff in der Praxis weiter, als die anderen und nehmen dadurch eine gewisse Mittelstellung zu der Frage ein. Wenn z. B. Kümmel rät, im späteren Stadium nach

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48 Stunden nur bei abklingendem Fieber oder fieberfreiem E
abzuwarten, so klingt das schon ganz anders, als wenn er ga
gemein den Lehrsatz aufstellt, nach 48 Stunden lieber Eis und
zu geben, falls nicht die Schwere des Anfalles den Eingriff not
macht. Im letzteren Fall ist gewissermaßen eine Verschlimm
im ersteren Fall bereits das Ausbleiben eines deutlichen Rück
der Erscheinungen für ihn genügend als Indikation für den ope
Eingriff im Zwischenstadium. Körte, v. Eiselsberg und T
spalten Abszesse sofort nach der Diagnose auch im Zwischens
ohne ihre weitere Abkapselung abzuwarten. Andere wiederum g
auch bei Abszessen die festere Abkapselung wenn irgend mögli
warten zu müssen mit dem Messer in der Hand und unter exa
Beobachtung auf die Möglichkeit einer Spontanperforation de
frischen Verwachsungen hin.

Zu den Vertretern der radikalen Richtung haben wir alle die
zu rechnen, welche die Periappendizitis prinzipiell in jedem S
und zu jeder Zeit, also auch im Zwischenstadium operativ anz
pflegen, da sie teils auf Grund persönlicher Erfahrungen, te
Grund wissenschaftlicher Erwägungen die Gefahr der perito
Allgemeininfektion als Folge des Eingriffs nicht allzuhoch eins
und die Bestätigung dafür in den guten Resultaten ihrer Sta
erblicken zu können glauben.

So hat, wenn wir die vorliegende Literatur einer kurzen Dur
unterziehen, u. a. Dörffler seiner eigenen Angabe nach einer
zenden, von ihm nie zuvor erreichten Erfolg erzielt, seitden
seiner Praxis jeden Fall von Periappendizitis sofort operier
irgendwie ernstere Erscheinungen zeigt. Und zwar versteht er
letzteren peritonitische Symptome im weitesten Sinne des W
Er hält jede Operation vor der Etablierung einer allgemeiner
tonitis für gefahrlos, leicht und sicher ausführbar und mein
durch eine rechtzeitige Operation bei entsprechender Sachk
und Sorgfalt das Auftreten einer tödlichen Allgemeinperitoni
mit absoluter Sicherheit vermieden werden kann. Selbst in z
haften Fällen ist er dafür, lieber eine Probelaparotomie zu n
als ein Menschenleben zu riskieren.

Auch Rehn stellt die Forderung auf, jede Blinddarmentz
operativ anzugehen, ehe sich die verderblichen Folgen der Erkr
ausgebildet haben. Sein Schüler Nötzel steht naturgemäß a
gleichen Standpunkt, indem er sagt: „Wir kennen keinen Zei
in dem wir abwarten. Wir operieren jeden Appendizitisanfa
seiner Einlieferung ins Krankenhaus zu jeder Tages- und Nac

Karewski meint, daß die Überleitung in das kalte Stadiu

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häufig gelingen kann, daß sie aber nicht selten ein Spiel mit dem Leben des Patienten ist. Da überhaupt jede Zeitbestimmung, jede Beurteilung der Schwere oder Leichtigkeit der Symptome trügerisch sein kann, hält er es für das richtigste in jedem Falle sofort einzugreifen.

Worcester hält die chirurgische Behandlung für das einzig richtige, fügt dann aber etwas diplomatisch hinzu, die Zeit der Operation bestimme der Gang der Erkrankung, ein Rat, der bei der Allgemeinheit seines Ausdrucks für den Praktiker wohl kaum von Wert sein kann.

Krecke spricht sich für die Operation unter allen Umständen auch ohne sichere Symptome der Eiterung aus.

Ebenso rät Delagénière stets sofort zu operieren, in welchem Stadium auch die Erkrankung bei der ersten Konsultation zur Behandlung kommt. Desgleichen operieren Rinne, v. Zwalenburg, Hahn, I. C. Stinson, A. H. Cordier jeden Fall zu jeder Zeit, da Abwarten immer gefährlich sein kann.

Der Hauptverfechter des operativen Vorgehens in jedem, also auch im Zwischenstadium ist Sprengel, nach dem die Periappendizitis am besten sofort nach gesicherter Diagnose operiert wird. Denn „findet man dabei Eiter, so war die Operation gerechtfertigt, findet man keinen, so ist sie unschädlich“. Im übrigen begründet Sprengel seinen radikalen Standpunkt des näheren damit, daß auch ohne Operation der Zeitraum des Zwischenstadiums, absolut betrachtet, die höchste Zahl an Todesfällen aufweist, da innerhalb dieser Zeit infolge des freien bzw. in Abgrenzung erst begriffenen Entzündungsprozesses die Gefahr einer peritonealen Allgemeininfektion am größten ist und am leichtesten eine entscheidende Wendung zum Schlimmeren eintreten kann. Gerade deswegen habe man ja, um dieses gefährliche Stadium zu vermeiden, den Vorteil der Frühoperation allgemein anerkannt. Sprengel vermag daher um so weniger einzusehen, weshalb man den Patienten bei späterer Aufnahme im Zwischenstadium erst den weiteren Gefahren desselben aussetzen solle, ohne diesen durch einen sofortigen Eingriff mit Sicherheit vorzubeugen. Absolute Klarheit über den Vorteil bzw. die Überlegenheit der Zwischenoperation kann eben nur eine vergleichende Statistik über die Mortalität einer größeren Anzahl von Fällen bringen, die während des Zwischenstadiums teils ohne, teils mit prinzipieller Operation behandelt worden sind und nicht nur auf besonders schwere Indikationen hin operiert sind. Solange eine solche Aufstellung fehlt, ist ein abgeschlossenes Urteil über diese Frage wenigstens vom praktischen Standpunkt aus nicht möglich und wird immer mehr oder

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weniger von den subjektiven Eindrücken und Erfahrungen de zelnen abhängen.

Vom theoretischen Standpunkt aus möchte ich eine Erwägu vorheben, die von Professor Lexer, als überzeugtem Anhäng Zwischenoperation, häufig in seiner Vorlesung angeführt wir die mir doch ganz erheblich zugunsten der Zwischenoperat sprechen scheint. Wenn man nämlich den Entzündungsvorga Appendix in seiner reaktiven Wirkung auf die Umgebung m Entzündungsvorgängen am Furunkel vergleicht, so ist bei le einwandsfrei der experimentelle Nachweis durch Impfung gefüh die durch Rötung, Infiltration und Schmerzhaftigkeit ausgezei sogenannte reaktive Entzündungszone der Haut um den eigen Infektionsherd herum durch eine erhöhte Produktion von bakte und bakteriolytischen Schutzstoffen gegen eine Infektion mit den des eigentlichen Herdes als immun zu bezeichnen ist.

In gleicher Weise bildet sich nun auch um den eigentliche zündungsherd am Appendix eine reaktive Entzündungszone aus, wir makroskopisch an der stärkeren Injektion und Schwellu viszeralen Peritoneums der umgebenden Darmschlingen deutl grenzen können. Soweit diese Injektion sich erstreckt, ist da toneum analog dem experimentellen Nachweis am Furunkel als zu bezeichnen gegen eine sekundäre Infektion vom primären I dungsherd aus, da es bereits durch verstärkte Hyperämie u zeugung entsprechender Schutzstoffe gewissermaßen seine Vo als Sicherung gegen einen Überfall der feindlichen Truppen stellt hat. Solange also der Operateur sich im Zwischens innerhalb dieser reaktiven Entzündungszone der Darmserosa h solange eine Verbreitung des infektiösen Virus über diese Zone vermieden wird, ist eine Allgemeininfektion des Peritoneu Sicherheit auszuschließen. Dieses zu erreichen wird im allge Sache der Erfahrung und Technik des Operateurs sein. Daß erreichen läßt, dürften die bisher erreichten, durchaus ermut Erfolge der oben angeführten Vertreter der Zwischenoperat Genüge beweisen. Man darf daher die Operation als solch kaum als besonders gefahrvoll bezeichnen, sobald der Operate die nötige Erfahrung und Technik verfügt.

Ziehen wir nach dieser theoretischen Erwägung noch in E daß das Spätstadium oder Abszeßstadium einerseits gerade de Zeitraum darstellt, in welchem es am ehesten zur Ausbildu bekannten schweren Folgezustände der Periappendizitis, Thro Embolien, metastatischer Abszesse u. dgl. kommt, und daß s seits bei dem so häufigen Gegensatz der klinischen Erscheinu

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dem anatomisch-pathologischen Befund trotz genauester und sorgfältigster Beobachtung gar nicht so selten bei der exspektativen Behandlung der dringendste Zeitpunkt zur Operation noch vor dem Eintritt einer peritonealen Allgemeininfektion versäumt werden kann, so dürften wir wohl mit Recht zu dem Schluß gelangen, daß die tatsächlichen Gefahren der Zwischenoperation doch wesentlich geringer einzuschätzen sind, als die Zufälle und Folgezustände, welchen die Patienten gerade durch das Zwischenstadium bzw. durch die prinzipielle Überleitung der Erkrankung ins Spätstadium oder Sicherheitsstadium ausgesetzt sein können.

Es ist daher wohl anzunehmen, daß nach weiteren günstigen Erfahrungen der radikaleren Operateure auch bei den heutigen Gegnern der Zwischenoperation die Furcht vor den Gefahren des Eingriffs an sich mehr und mehr schwinden wird, so daß sie selbst allmählich ihre Indikationen weiter und weiter stellen und dann auf Grund eigener Erfahrungen ihre Anschauung nach der radikaleren Richtung hin revidieren werden. Alles in allem scheint mir die Zwischenoperation ein Eingriff zu sein, über dessen Indikationsstellung und Zweckmäßigkeit die Meinungen zwar noch geteilt sind, der aber mit der häufigeren Anwendung und Erprobung auch seitens seiner heutigen Gegner den Beweis seiner Berechtigung selbst erbringen dürfte, um so mehr als bei ihm gleichwie bei der Frühoperation die radikale Entfernung des erkrankten Appendix die Regel bildet und damit die Patienten gleichzeitig der Notwendigkeit eines zweiten operativen Eingriffs zwecks definitiver Heilung enthoben werden.

Hinsichtlich der Technik ist dabei zu bemerken, daß durch frische Verwachsungen und ev. in der Bildung begriffene Abszesse um den Appendix herum die Übersicht des Operationsfeldes erheblich erschwert und daher die Anlegung eines größeren Schnittes zur Eröffnung der Bauchhöhle von vornherein erforderlich ist. Da man nun aber bei direktem Vorgehen auf den Krankheitsherd durch die Bauchdecken hindurch Gefahr laufen würde, durch absichtliche oder unabsichtliche Lösung der Adhäsionen die freie und gänzlich ungeschützte Bauchhöhle zu infizieren, so wird von Rehn, Riedel, Sprengel, Dörffler, Murphy, Moskowicz u. a. empfohlen, prinzipiell von der freien Bauchhöhle aus gegen den Entzündungsherd vorzugehen und zu diesem Zwecke die Bauchhöhle nicht direkt über, sondern dicht neben dem Krankheitsherd zu eröffnen. Man kann dann unter guter Übersicht über die gesamte Sachlage das freie Peritoneum durch entsprechende Tamponade um den Entzündungsherd abschützen, um so in Sicherheit an die Lösung der frischen Adhäsionen und die radikale Entfernung des erkrankten Organes heranzugehen.

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Wesentlich kürzer können wir uns fassen bezüglich der Ope
im Spät-, Not- und Sicherheitsstadium, über deren Zweckmäß
bzw. Notwendigkeit heute volle Einstimmigkeit herrscht. Erg
sich doch von selbst, daß für

die Spätoperation

im Stadium der abgeschlossenen intraabdominalen Eiterung de
Lehrsatz „ubi pus, ibi evacua" mehr als irgendwo anders seine
Geltung behalten muß. Allerdings haben auch hier in den l
Jahren die günstigen Erfahrungen der radikaleren Operateure m
Sicherung der Bauchhöhle durch Tamponade und die dadurch l
geminderte Furcht vor der peritonealen Allgemeininfektion die
kationsstellung zum operativen Eingriff noch erheblich erw
Während man sich nämlich früher aus Angst vor einer weiteren
breitung der Infektion nach Möglichkeit auf die Eröffnung der
flächlich, dicht unter den Bauchdecken gelegenen Abszesse z
schränken suchte und womöglich wartete bis sie einer di
Eröffnung von außen her zugänglich waren, gehen heute die
genannten Autoren in gleicher Weise wie im Zwischenstadium
die abgekapselten Abszesse im Spätstadium prinzipiell durch di
Bauchhöhle an, da sie in der größeren Übersichtlichkeit und
tigen Abtamponierung der Umgebung des Abszesses bei diesem
gehen eine bessere Sicherung des freien Peritoneums erblick
müssen glauben, als in der direkten Eröffnung der Abszesse dur
Bauchdecken hindurch, bei welcher unabsichtliche Eröffnunge
freien Peritoneums infolge geringerer Übersicht viel leichter de
obachtung entgehen und infolge Fehlens der schützenden Tamp
auch leichter zur nachfolgenden Allgemeininfektion des Perito
Anlaß geben können.

So erhält man nach Dörffler die besten Resultate, wenn
sowohl in den frischen, wie in den verschleppten Fällen ausnah
von der freien Bauchhöhle aus gegen den entzündlichen Tumo
geht. Er eröffnet daher nach dem Vorgange von Rehn, Riede
Sprengel prinzipiell von vornherein die freie Bauchhöhle, um
unter Abdichtung des freien Peritoneums mit Gazekompressen s
gegen die Resistenz vorzudringen, den Abszeß möglichst in Seit
zu eröffnen und wenn möglich die Radikaloperation anzuschl
Sodann Dränage der Höhle durch Tamponade, Auswechsel
Schutztampons am Peritoneum durch frische sterile Gaze und Ve
Der nächstgrößere Verbandwechsel bzw. die Entfernung der
ponade wird nicht vor 8 Tagen vorgenommen, um eine genü
Festigkeit der abschließenden Verwachsungen zu garantieren
übrigen glaubt Dörffler, daß trotz der Tamponade in dera

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Fällen die Gefahr eines späteren Narbenbruches nicht allzugroß sei, da es bei frühzeitiger Operation noch nicht zu erheblichen Ernährungsstörungen der Fascie gekommen ist.

Rehn handelt auch hier nach dem Grundsatz, daß stets in der Erkrankung und nie in der Operation die eigentliche Gefahr zu erblicken ist. Er empfiehlt sogar den Processus, wenn möglich, aufzusuchen und zu amputieren und rät auch leichte Verwachsungen vorsichtig zu lösen, um sämtliche Buchten des Abszesses freizulegen. Wenn er dann aber nur unter Einlegen eines Dräns in die Höhle rät, die Bauchhöhle primär zu schließen bis auf die relativ kleine Öffnung für das Dränrohr, so mag das ja wohl eine Berechtigung auf Grund seiner persönlichen guten Erfahrungen haben. Wenn er vom theoretischen Standpunkt jedoch dafür anführt, daß gerade dieser exakte Schluß der Bauchhöhle eine wichtige Vorbedingung für eine gute Dränage sei, da dadurch der normale intraabdominale Druck wieder hergestellt werde, so darf man dem vielleicht entgegenhalten, daß es fraglich und zum mindesten noch nicht bewiesen ist, daß dieser intraabdominale Druck das Hauptagens bei der Entleerung des Sekretes ist, sondern daß dabei ein großer Anteil, wenn nicht der Hauptanteil auf die kapillare Ansaugung seitens der Gazedränage einerseits und auf die Vis a tergo der von hinten her nachdrängenden mehr und mehr serösen Exsudation andererseits zu schieben ist. Wenigstens scheint mir eine derartige Frage nicht unberechtigt, wenn man erwägt, daß bei analogem Vorgehen an den Extremitäten auch nicht der Gewebsdruck an sich, sondern die künstlich durch die Biersche Stauung noch gesteigerte Vis a tergo des nachdrängenden serösen Exsudates das eigentliche, rein mechanische Agens bei der Austreibung und Entleerung der schädlichen Stoffe darstellt. Des weiteren ist dagegen vielleicht noch anzuführen, daß, rein mechanisch gedacht, ein geringerer Querschnitt der Dränageöffnung unmöglich dasselbe leisten kann, wie ein größerer Querschnitt, und daß diese Differenz wohl kaum durch die in ihrer Wirkung noch etwas unsichere Erhöhung bzw. Wiederherstellung des alten intraabdominalen Druckes ausgeglichen wird. Im übrigen dürfte sich auch bei der geringsten Retention an irgendeiner Stelle der Abszeßhöhle die sekundäre Entleerung derselben nach der Methode von Rehn wesentlich schwieriger gestalten, eine solche Retention aber von vorneherein auszuschließen dürfte gerade bei dieser auf das geringste Maß beschränkten Anwendung der Dränage ein genügender Grund nicht vorliegen. Ein besonderer Anlaß zur Empfehlung der Versorgung der Abszeßhöhle nach dem Vorgang von Rehn scheint mir danach nicht vorzuliegen, solange nicht eine tatsächliche Überlegenheit dieser Methode über die alte,

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ausgiebigere Art der Dränage praktisch und wissenschaftlich wandsfrei nachgewiesen ist.

Trotz der guten Erfolge, welche die genannten Autoren mit i radikalen Vorgehen zu verzeichnen haben, steht die Mehrzah Chirurgen noch auf dem älteren Standpunkt, bei der Inzision abgeschlossenen Abszessen, wenn irgend möglich, die Eröffnun freien Bauchhöhle zu vermeiden und den Appendix nur dann z fernen, wenn er sich sozusagen dem Messer von selbst darbiet bleibt dann die definitive Entfernung des Appendix einer zw Operation vorbehalten, die man bis zum nächsten Anfall versch kann, wenn der Patient in der Lage ist, sich bei den ersten Anze desselben zur sofortigen Frühoperation einzufinden. Im anderen wird man bereits vorher im Sicherheitsstadium die Appendek vornehmen, um den Patienten nicht unnötig der Möglichkeit un Zufällen eines weiteren Anfalles ohne sofortige chirurgische auszusetzen.

Hinsichtlich der Indikationsstellung zum Eingriff kann für Praktiker nicht genug betont werden, daß der Eingriff am besten findet, sobald eine fühlbare und schmerzhafte Resistenz auch nu Verdacht einer umschriebenen Eiterung nahelegt. Wenn man nä in Betracht zieht, daß immer noch und zwar gar nicht so selten zur Operation eingeliefert werden, in denen bisweilen der A bereits die Bauchdecken vorwölbt und in denen dann di zision eine Unmenge von Eiter entleert, so fragt man sich i wieder, wie es heutzutage noch möglich ist, daß ein Arzt an di sorptionsmöglichkeit derartiger Exsudatmengen auch nur denken andererseits eine festere Abkapselung des dauernd anwachs Abszesses bei konservativer Behandlung erwarten kann, ohne darüber klar zu werden, daß er auf diese Weise geradezu ein mit dem Leben seiner Patienten treibt. Solche Fälle haben nachträglich für den betreffenden Arzt noch das Bedauerliche an daß sich die mißhandelten Patienten spontan die gleiche Frage legen und trotz der gut gemeinten Beruhigungsversuche des Chir in einer für die wissenschaftlichen Fähigkeiten des Arztes schmeichelhaften Weise selbst zu beantworten pflegen.

Es muß daher immer wieder hervorgehoben werden, daß in Falle der praktische Arzt am sichersten geht, wenn er bei jeder A dizitis überhaupt, und um so mehr wenn er sie erst im Spätst zu Gesicht bekommt, möglichst frühzeitig einen Chirurgen zu zieht, um sich auf dessen größere Erfahrung hinsichtlich der I tion zum operativen Eingriff zu stützen.

Kommen wir nun schließlich zu demjenigen Stadium, in we

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durch die eingetretene Allgemeininfektion des Peritoneums die Not am größesten ist, und welches daher mit Recht die Bezeichnung als Notstadium verdient, so ist bezüglich des operativen Eingriffs in demselben, der

Notoperation,

zu bedenken, daß die Aussichten derselben naturgemäß um so günstiger sind, je früher dieselbe erfolgt, und daß der entscheidende Wendepunkt für eine günstige Prognose bereits nach Ablauf der ersten 48 Stunden gegeben ist. Es wird daher für dieses Stadium der alte Grundsatz, lieber zu früh, wie zu spät zu operieren, eine doppelte Geltung haben müssen, da man besser einen Patienten noch vor der Allgemeinperitonitis operiert, als daß man in dem Bestreben, eine Klärung und Sicherstellung der Diagnose herbeizuführen, die kostbaren Stunden ungenützt verstreichen läßt. Es ist daher mit Recht jeder einer Allgemeininfektion des Peritoneums auch nur verdächtige Patient stets so zu behandeln, als wenn tatsächlich eine solche bereits vorhanden wäre.

Mit Rücksicht auf den grundsätzlichen Unterschied bei der Prognose der Notoperation vor und nach den ersten 48 Stunden wird der Eingriff innerhalb der ersten 48 Stunden seit Beginn der Allgemeininfektion des Peritoneums auch als Frühoperation schlechtweg bezeichnet. Da man aber darunter gemeinhin auch die absolute Frühoperation in den ersten 48 Stunden der Erkrankung überhaupt verstehen kann, habe ich im Gegensatz zu der letzteren die Benennung der relativen Frühoperation gebraucht, da es sich dabei ja nicht um eine absolut, sondern nur relativ frühzeitige Operation handelt, nämlich hinsichtlich des zeitlichen Beginns der peritonealen Allgemeininfektion, und nicht der Erkrankung an sich.

Bezüglich der Indikationsstellung für den Eingriff herrscht mit Rücksicht auf die günstige Prognose desselben innerhalb der ersten 24 Stunden eine absolute Einigkeit über die strikte Notwendigkeit der relativen Frühoperation. Über die eigentliche Notoperation nach 48 Stunden sind die Ansichten jedoch etwas geteilt, indem einzelne Autoren auch dann prinzipiell stets operativ vorgehen, während andere die Indikation zum Eingriff von Fall zu Fall stellen und sie von dem Allgemeinzustand der Patienten in erster Linie abhängig machen wollen. Letztere neigen zu der Annahme, daß der an sich bereits sehr geschwächte Patient entweder dem operativen Eingriff direkt erliegen könne, oder daß er zum mindesten dadurch allzusehr geschwächt werde, um mit den im Körper bereits vorhandenen Giftstoffen fertig zu werden und so mittelbar den Folgen der Operation erliegen müsse.

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In diesem Sinne macht u. a. auch Dörffler den Vorschlag 48 Stunden die Indikation für den Eingriff lieber von Fall zu F entscheiden und den Eingriff zu unterlassen bei schlechtem rascher Atmung und hochgradigem Meteorismus, kurz bei den üb Anzeichen einer weit fortgeschrittenen Peritonitis. Zu erwägen es seiner Meinung nach, ob der Eingriff sich noch lohnt, bei Puls und leidlichem Allgemeinbefinden des Patienten.

Nordmann will bei allgemeiner Peritonitis erst nach dem 5. mit Auswahl operieren, da sich dann vielleicht eine Reihe vo tienten noch erholen könne, welche sonst der Operation er würden, und namentlich da bei hochgradiger Darmlähmung die sichten auf den Erfolg eines Eingriffs sich wesentlich verschlech wenn nicht gänzlich in Fortfall kämen.

Wieder andere wollen bei der Perforationsperitonitis über erst das Schwinden der vorhandenen Shokerscheinungen abw da in diesem Zeitpunkt die Operation zu gefährlich sei. Diese schauung darf man wohl mit Lennander entgegenhalten, d derartigen Fällen das beste Mittel gegen den Shok die Operati denn cessante causa, cessat effectus.

Auch Kocher hält die operative Behandlung der allgemeine Zwerchfell bis zum Becken sich erstreckenden Peritonitis für ä undankbar, und zwar ebenfalls wegen des Shoks und der gestei Resorption von Giftstoffen, welche der operative Eingriff zur habe. Da nun nach experimentellen Untersuchungen von Cir plötzliche Abfall des Blutdruckes, welcher als eigentliche Ursac Shokes aufzufassen ist, durch eine vorübergehende Ausschaltu arteriellen Blutzufuhr zu den Därmen zu verhindern sein s meint Kocher, daß danach die Frage diskussionsberechtigt ersc könne, ob zu dem gleichen Zweck die vorübergehende Anlegung Klemmzange am Mesenterium für die kurze Zeit der Eventrati Reinigung der Därme in Betracht zu ziehen sei. Die Entsch der Frage dürfte davon abhängig sein, ob sich jemand entschließ Vorschlage Kochers praktische Folge zu geben.

Trotz dieser Einwände der mehr individualisierenden Ri darf man wohl doch die z. Z. herrschende allgemeine Anschauun die Indikationsstellung zur Notoperation dahin definieren, daß in aussichtslos erscheinenden Fällen von allgemeiner Peritonit Darmlähmung der Shok einer unter Lokal- oder Lumbalanäs oder Äthertropfnarkose ausgeführten Operation nicht ein der ist, daß man dem Patienten deswegen die letzte Chance zur R entziehen müßte, selbst auf die Gefahr hin, die eigene Oper statistik mit einem Todesfall mehr zu belasten. Denn die

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welche im Stadium der Allgemeinperitonitis mit Darmlähmung auch ohne Operation tatsächlich durchkommen, dürften zu den Ausnahmen zu zählen sein, welche die Regel von der absoluten Aussichtslosigkeit auch der konservativen Behandlung unter derartigen Umständen bestätigen.

Im übrigen ist selbst in diesen Fällen der operative Eingriff um so mehr in Betracht zu ziehen, als man ja in der namentlich von Barth und Heidenhain empfohlenen mehrfachen Enterostomie gleichzeitig ein Mittel zur Hand hat, die bereits geblähten und paralytischen Darmschlingen zu entlasten und ihnen dadurch eine Möglichkeit mehr zur Erholung und Erneuerung der geschwundenen Peristaltik zu gewähren.

So aussichtslos auch der operative Eingriff im vorgeschrittenen Stadium der allgemeinen Peritonitis zu sein pflegt, so darf man ihn mit gutem Gewissen doch wohl kaum unterlassen, da in der Regel der Nutzen der Operation eine mögliche Shokwirkung derselben weit überwiegen dürfte, um so mehr als wir heute in den intramuskulären oder subkutanen Kochsalzinfusionen ein zuverlässiges Mittel gegen letztere stets zur Verfügung haben.

Hinsichtlich des eigentlichen therapeutischen Prinzipes bei der allgemeinen Peritonitis sei nur kurz hervorgehoben, daß dasselbe im allgemeinen drei verschiedenen Indikationen zu genügen hat, nämlich dem Bestreben 1. eine möglichst weitgehende Entlastung des Körpers von Infektionserregern und deren Produkten zu erreichen, 2. eine Kräftigung und Hebung der gesamten Körpervitalität im allgemeinen und damit die Möglichkeit einer verstärkten Produktion von Abwehrstoffen herbeizuführen und 3. eine Kräftigung und Hebung der Gewebsvitalität des Peritoneums und der gesunkenen bzw. gänzlich geschwundenen Darmbetätigung im besonderen herbeizuführen.

Der ersten Indikation läßt sich nur durch den operativen Eingriff gerecht werden, welcher in ausgiebigen und je nach der Schwere des Falles mehr oder weniger zahlreichen Inzisionen der Bauchdecken mit nachfolgender Drainage zu bestehen hat. Diese Inzisionen werden in der Regel beiderseits oberhalb und parallel dem Poupartschen Bande angelegt, wobei der Appendix wenn möglich gleichzeitig entfernt wird. Dazu können dann nach Bedarf eine Inzision in der Linea alba, Gegeninzisionen in der Lumbalgegend beiderseits, sowie Inzision und Dränage durch Douglas oder Scheide hinzukommen, je nachdem eine Verbreitung bzw. Lokalisierung der Entzündungsprodukte zu konstatieren ist.

Von einer gleichzeitigen mechanischen Reinigung durch Tupfer oder Spülung mit antiseptischen Lösungen ist man längst zurückge-

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kommen, da erstere naturgemäß zu einer mechanischen und
zu einer chemischen Schädigung der an sich in ihrer Vitalität
gesetzten Gewebe führen muß. Die Mehrzahl der Autoren b
sich vielmehr damit, durch ausgiebige Spülung der Därme mit p
logischer Kochsalzlösung eine vorsichtige mechanische Entfernu
Erreger und ihrer Produkte bzw. eine Verdünnung derselben
zuführen, wobei man in schweren Fällen so weit geht, sämtliche
schlingen vor die Bauchhöhle zu lagern und im einzelnen einer
lichen Spülung mit Kochsalzlösung zu unterziehen.

Eine geringere Anzahl von Autoren will auch auf diese S
verzichten, da sie glauben, daß der Nutzen der mechanische
fernung einer Anzahl von Infektionserregern überwogen werde
die Gefahr einer weiteren Verbreitung der Erreger vermittel
Spülflüssigkeit in noch freie Buchten und Falten der Bauchfell
in denen sich diese dann ansiedeln und weitere Schädigunge
ursachen könnten. Beide Teile behaupten mit ihrem Vorgehe
Erfahrungen gemacht zu haben, was an sich nicht unverständl
wenn man nämlich annimmt, daß das Spülverfahren praktis
wenig mehr nützt, aber auch zum mindesten ebensowenig sc
wie das rein konservative Verhalten gegenüber dem infi
Bauchfell.

Vom theoretischen Gesichtspunkt aus will mir die Annahme
scheinlicher vorkommen, daß das Spülverfahren doch eine g
Überlegenheit haben müßte auf Grund folgender Erwägung.
leugbarer Vorteil desselben besteht darin, daß es fragelos die a
Zahl der im Körper vorhandenen Infektionserreger verminde
dadurch die Produktion von Toxinen und Endotoxinen in erheb
Maße herabsetzen muß.

Des weiteren werden die noch am Bauchfell übrigbleibenden, zu
in gewissem Grade in größeren Haufen, bzw. konzentriert gel
Bakterienansammlungen zwar fragelos in bisher nicht infizierte Ge
fortgerissen, aber doch andererseits auch auf eine viel größe
zum Teil in ihrer Widerstandskraft noch nicht wesentlich herabg
Fläche des Peritoneums verteilt, das logischerweise durch die
derte Verteilung der auch an absoluter Zahl verminderten An
auf eine gewissermaßen längere und entsprechend dünnere
gegenüber der nunmehr zur vollen Ausnutzung gelangenden und s
weise mit frischen Truppen besetzten Verteidigungslinie der
sicherlich mehr Chancen hat, über die nunmehr in kleinere Ge
einheiten zerlegten Angreifer Herr zu werden, als die letzte
ihrer geringeren Anzahl eine Bresche in die Festung zu
Praktisch mag das vielleicht nicht von allzugroßer Bedeutun

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etisch will mir die Entscheidung der Frage in diesem Sinne
h als das wahrscheinlichere bedünken.
er zweiten Indikation, der Kräftigung und Hebung der Lebens-
und physiologischen Abwehrbetätigung des Körpers im allge-
en, dient vor allem die reichliche Verabfolgung von intramusku-
oder subkutanen Kochsalzinfusionen, von denen die ersteren
größerer Schmerzhaftigkeit für die Patienten weitaus den Vorzug
enen, da sie schneller resorbiert und daher auch in weit größeren
titäten gegeben werden können, ohne doch wie die letzteren
zeitweilige Ernährungsstörungen zu den bekannten Fascienne-
n zu führen, die dann von einzelnen wegen ihrer Resorptions-
eraturen auf eine Infektion von außen her bzw. mangelhafte
nik bei der Infusion geschoben zu werden pflegen. Andererseits
er auch nicht zu vergessen, daß durch diese Infusionen überhaupt,
durch die subkutanen Infusionen bei ihrer langsameren Resorp-
ganz besonders, im Gewebe gegenüber den im Blut kreisenden In-
nserregern ein Locus minoris resistentiae geschaffen wird, und daß
f diese Weise sehr wohl an den Infusionsstellen zu Abszeßbildung
rund einer Autoinfektion des Körpers kommen kann, die gleichfalls
iner Infektion von außen her gar nichts zu tun hat. Vielfach wird
bereits der für Bact. coli typische, fäkulente Geruch des Abszeß-
diese Vermutung zur absoluten Sicherheit erheben können, ja ich
ere mich eines Falles, in dem bereits makroskopisch der Abszeß-
t den vollkommenen Eindruck eines dünnflüssigen Stuhles machte.
iese Autoinfektion ist eine Möglichkeit, an welche man gegebenen-
wohl denken muß; sie ist aber andererseits bei der Seltenheit
Auftretens kein Grund, deswegen die geradezu lebensrettenden
salzinfusionen zu unterlassen, welche wie oben erwähnt gleich-
als das sicherste und zuverlässigste Mittel zur Beseitigung einer
orhandenen Shokwirkung zu bezeichnen sind.
on den früher beliebten intravenösen Kochsalzinfusionen ist man
schen gänzlich abgekommen, da sie bei wesentlich umständlicherer
nik absolut nichts mehr zu leisten vermögen, als die erheblich
cheren intramuskulären Infusionen.
es ferneren wäre dann an eine Stärkung und Hebung der Herz-
eit und Blutzirkulation durch die üblichen Exzitantien, wie Alkohol,
pfer und Digitalis zu denken, die man bei häufigerem Erbrechen
eder per rectum in Gestalt von kleinen Einläufen oder auch sub-
, soweit Kampfer und Digitalis in Betracht kommt, verabfolgen kann.
wieweit man durch die Anwendung spezieller oder polyvalenter
era, wie sie heute fabrikmäßig erzeugt und teils per rectum teils
utan verabfolgt werden, eine weitere Erhöhung der Abwehrbetäti-

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gung tatsächlich erzielen kann, ist eine Frage, die heute noch ni
spruchreif zu bezeichnen und im allgemeinen von den Erfah
der einzelnen Autoren abhängig zu machen ist. Allerdings sin
Erfahrungen zurzeit noch so wenig ermutigend, daß die Mehrz
Autoren auf die Anwendung des Mittels von vornherein ver

Das gleiche wäre von der perkutanen, intravenösen od
rectum empfohlenen Anwendung der von manchen ebenfalls
gepriesenen Silbersalze in Gestalt der Kollargolzusammenset
zu sagen. Als praktisch wirksam haben sich bisher nur die Ko
infusion und die bekannten Herzexzitantien erwiesen.

Der dritten therapeutischen Indikation zur Hebung der lokal
websvitalität des Peritoneums und der herabgesetzten oder g
aufgehobenen Peristaltik der Därme werden — abgesehen v
eventuellen Enterostomie nach Barth — am ersten hohe und
oder permanente Einläufe mit physiologischer Kochsalzlösu
rectum gerecht, die heute von allen Autoren ausnahmslos em
und angewandt werden und an Menge in der Regel 1—1½ Lit
mehr betragen sollen. Wenn einzelne Autoren diesen Einläufe
besondere Zusätze zwecks einer erhöhten Wirkung geben wolle
beispielsweise Lennander mit 5% Traubenzucker und 3% A
und Weir mit 15 Tropfen einer 0,1%igen Adreanlinlösung st
Alkohols, so liegen über den tatsächlichen Wert derartiger Z
zu wenig Erfahrungen vor, um ein abschließendes Urteil darü
ermöglichen. Der Hauptwert der Einläufe dürfte jedenfalls
der besseren Ernährung und Durchspülung der Darmwandung i
rein mechanischen Einwirkung auf die Anregung der Darmt
zu erblicken sein, indem sie gleichzeitig die Entstehung vo
wachsungen bzw. fibrinösen Verklebungen der Därme unter s
verhindern, wie ev. auch leichtere Verklebungen zu lösen geeigne

Von medikamentösen Mitteln zur Anregung der gesunkenen
peristaltik sei der Vollständigkeit halber noch hingewiesen a
von Nordmann empfohlenen Koffeininjektionen, ferner auf di
kutanen Injektionen von Atropin. sulfuricum und die neuerdings v
in Aufnahme gelangten Injektionen von Physostigmin, das man
Maximaldosis von 0,001 dreimal pro Tag anzuwenden pflegt, u
dem viele Autoren gute Erfolge gesehen haben.

Kurz zusammengefaßt hängt also bei der Allgemeininfekti
Peritoneums in erster Linie das Leben des Patienten von einer
lichst frühzeitigen Vornahme der lebensrettenden Notoperati
welche jedoch nur als relative Frühoperation wirklich gute Au
für die Heilung des Patienten zu bieten vermag. Nach Verla
48 Stunden verschlechtert sich die Prognose sehr schnell und

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t proportional der zeitlichen Entfernung des Eingriffs vom Beginn
llgemeininfektion des Peritoneums. Immerhin ist darin kein Grund
rblicken, selbst in verzweifelten Fällen dem Kranken diese letzte
ce zur Rettung zu versagen, da nach dem heutigen Stande der Tech-
ie etwaige Shokwirkung eines derartigen Eingriffs die Vorteile des-
n selbst in den schwersten Fällen kaum jemals überwiegen dürfte.
s erübrigt sich schließlich noch die Besprechung der Behandlung
cherheitsstadium, dem Stadium der abgelaufenen bzw. chronischen
ppendizitis. Dieselbe hat heute in der Regel ebenfalls in der
alen Entfernung des Appendix durch den als

Sicherheitsoperation

uns bezeichneten Eingriff zu bestehen. Es ist dieses diejenige
ation, welche sozusagen den Anfang der so überaus segens-
rfolgreichen chirurgischen Behandlung der Periappendizitis ge-
hat, und welche aus der Einsicht heraus entstanden ist, daß
definitive Heilung der Erkrankung erst mit der Entfernung des
ndix möglich ist, da man ohne dieselbe nie vor Rezidivanfällen
ist und der Patient sonst, wie Sprengel, glaube ich, sich
ückt, sein Totenhemd in Gestalt des erkrankten Appendix stets
ch trägt. Es kann daher auch ohne die Entfernung des Appen-
ie von einer absoluten, sondern immer nur von einer relativen
ng des Patienten bezüglich des letzten akuten Anfalles, nicht
bezüglich der Erkrankung überhaupt die Rede sein.

eses ist denn auch der Grund gewesen, weswegen zunächst bei
Eingriffen im Sicherheitsstadium in gleicher Weise, wie später
bei den Eingriffen im akuten Stadium die Indikationen immer
r gezogen sind. Während früher nämlich die Mehrzahl der
en auf dem Standpunkt verharrte, nur nach einem schweren
l den Appendix zu entfernen, und bei einem leichteren Auftreten
Erkrankung, der sog. chronisch rezidivierenden Periappendizitis,
—3 Anfälle abzuwarten, ehe man sich zum operativen Vorgehen
hloß, ist heute hierin ebenfalls ein Umschlag nach der radikaleren
ung hin eingetreten insofern, als die Mehrzahl auch der konser-
ten Chirurgen bereits nach dem ersten, als solcher deutlich ge-
zeichneten Anfall von Periappendizitis in allgemeiner Übereinstimmung die Entfernung des Appendix empfehlen, da man nie wissen
wie schwer sich der nächste Anfall gestalten wird.

ne Ausnahme davon kann man, wie oben erwähnt, nur bei den-
en Patienten machen, welche durch ihren Wohnsitz jederzeit in
age sind, bei den ersten Anzeichen des nächsten Anfalls sich
rühoperation einzufinden, und von denen man überzeugt sein
daß sie dieses dann auch tatsächlich tun werden. Man kann auf

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diese Weise dem Einwand begegnen, daß die betreffenden Pa
sonst vielleicht gar keinen Anfall mehr bekommen hätten, was ja s
lich ausnahmsweise einmal zutreffen kann. Jedoch ist auch fü
Fälle zu bedenken, daß der betreffende Patient selbst nach vielen
nicht vor einem neuen und schwereren Anfall sicher ist, da fra
den meisten Fällen durch die vorausgegangenen pathologische
änderungen am Appendix eine gewisse Prädisposition zum n
Anfall bzw. zu einer neuen Erkrankung des Appendix zurüc
von der man nie voraussagen kann, unter welchen mehr oder
ungünstigen Verhältnissen der Patient von ihr betroffen werde

Das beste Beispiel dafür, wie spät ein derartiger zweiter
eintreten kann, bietet Geheimrat Garrè selbst dar, der mir geg
sich häufig als Beispiel dafür anzuführen pflegte, daß eine A
zitis auch bei konservativer Behandlung dauernd ausheilen kö
er als Knabe von 13 Jahren einen Anfall durchgemacht hal
seitdem dauernd von Beschwerden verschont geblieben sei
zweite Anfall trat dann in typischer Weise im Herbst 1907 a
erst die im Dezember 1907 erfolgte Sicherheitsoperation biet
mehr die sichere Gewähr einer dauernden Heilung. Es ist j
los, daß auch hier in dem zweiten Anfall gewissermaßen ein
vom ersten Anfall unabhängige Erkrankung des Appendix n
langer anfallsfreier Zeit zu erblicken ist, es ist aber auch ebens
los, daß durch eine gewisse Prädisposition auf der Grundl
Residuen des ersten Anfalls ein mittelbarer ätiologischer Zus
hang mit demselben gegeben sein kann.

Im übrigen bietet die Sicherheitsoperation nach dem erste
den weiteren Vorteil, daß sie technisch in der Regel äußerst
ist, da die Verwachsungen dann naturgemäß noch nicht so z
zu sein pflegen, wie nach mehreren Anfällen, und daß sie dah
dem Patienten die größte Sicherheit bietet für eine gefahrlo
dauernde Wiederherstellung.

Trotz ihrer unbestreitbaren Vorzüge hat aber die Sicherh
ration gegenüber der Frühoperation im ersten Anfall den N
daß man mit gewissen Verwachsungen im Sicherheitsstadium
siduen der vorausgegangenen Entzündung fast immer zu rechn
Diese pflegen ja in der Regel nicht derart zu sein, daß si
wesentlichen Einfluß auf den Verlauf und die Technik der Op
auszuüben vermöchten. Sie können aber auch vereinzel
schweren Anfällen in solcher Anzahl, Ausbreitung und Stär
handen sein zwischen den um den Appendix herum gelegenen D
daß dadurch einerseits die Orientierung und das Aufsuch
Appendix innerhalb derselben ganz enorm erschwert wird und a

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s bei dem Versuch einer stumpfen Lösung derselben eher die
mwandung selbst nachgibt, als die breiten schwartigen und
vieligen Verwachsungen zwischen den Därmen selbst. Man ist
n wohl oder übel gezwungen, diese Verwachsungen mit dem Messer
durchtrennen und schwebt so bei der schwierigen Orientierung
ich in doppelter Gefahr, eine Verletzung des Darmes selbst her-
uführen. Es können auf diese Weise in Ausnahmefällen Verhält-
e gegeben sein, welche den Eingriff technisch zu einer der schwie-
en Operationen der gesamten Abdominalchirurgie gestalten können,
en Prognose dann natürlich auch eine entsprechend zweifelhafte ist.
ch selbst erinnere mich an drei derartige Fälle, deren einer in
Privatklinik von Professor Lexer und die beiden anderen in
ier Privatklinik zur Operation gelangten. Alle drei Patienten
en nach verlängertem Krankenlager durch, jedoch stellte die
ration in allen drei Fällen einen derartig schwierigen Eingriff dar,
sie zum mindesten den Namen einer Sicherheitsoperation kaum
fertigen würde, wenn nicht eben derartige Fälle als Ausnahmen
ezeichnen wären, welche im übrigen nur die Regel bestätigen.
iese Besorgnis vor stärkeren Verwachsungen hat nun zwei ver-
dene Richtungen bezüglich des Zeitpunktes für den Eingriff nach
ereren Anfällen gezeitigt. Während nämlich die einen raten,
ichst frühzeitig nach dem Abklingen der akuten Symptome ein-
eifen, da dann die Verwachsungen noch nicht so fest und relativ
t zu lösen sind, wollen die anderen möglichst bis zu einem Jahr
darüber hinaus mit dem Eingriff warten, da dann erfahrungsge-
ein Teil der peritonealen Adhäsionen durch Resorption ge-
unden sein kann, oder sie ziehen, falls ein neuer Anfall da-
chen kommt, die absolute Frühoperation im zweiten Anfall vor,
nach ihrer Meinung infolge der entzündlichen Schwellung und
htränkung der Gewebe während des akuten Stadiums sich die
ng der festen Adhäsionen weniger schwierig gestalten dürfte.
he von den beiden Anschauungen in diesen Fällen von „Ein-
rung des Appendix", wie v. Eiselsberg sie bezeichnet, vorzu-
n ist, dürfte von den individuellen Erfahrungen des einzelnen ab-
ig sein. Sicherer will mir persönlich die letztere mehr abwartende
ode erscheinen, da man bei einem allzu frühzeitigen Vorgehen nach
Anfall doch zu sehr mit der Möglichkeit des Vorhandenseins voll-
enter Exsudatreste und entsprechend erhöhter Gefahr einer opera-
Allgemeininfektion des Peritoneums zu rechnen haben dürfte.
n ganz besonders schweren Fällen dieser Art kann sich daher
eilen der Operateur intra operationem vor die Alternative gestellt
n, entweder, wenn er den Appendix unter allen Umständen ent-

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fernen will, eine ausgedehnte Darmresektion in Gestalt der Entf
des untersten Ileum mit Cöcum und dem Beginn des Colon asc
zu machen, oder, falls er den Patienten nicht der Gefahr eine
artigen Eingriffs aussetzen will, die Bauchhöhle zunächst unverri
Dinge zu schließen und bis zu dem nächsten Anfall abzuwarten
vermittelnde Stellung hierzu nimmt der Vorschlag v. Eiselsber
in derartigen Fällen wenigstens durch die Anlegung einer Ente
stomose zwischen Kolon und Ileum den Verwachsungsbezirk der
schlingen auszuschalten und so eine Entlastung desselben neben
zeitiger Vorbeugung eines späteren Verwachsungsileus herbeizuf

Wenn nun im übrigen auch die Möglichkeit derartiger Fäl
schweren Verwachsungen fraglos als ein gewisser Nachteil f
Sicherheitsoperation gegenüber der absoluten Frühoperation
zeichnen ist, so sind doch andererseits diese Fälle im Siche
stadium so außerordentlich selten, daß sie einen Einfluß auf die g
Mortalitätsziffer der Sicherheitsoperation nicht auszuüben, dahe
die Sicherheitsoperation als solche nicht zu diskreditieren ver

Auf die technische Seite der an sich ja äußerst einfachen S
heitsoperation näher einzugehen, würde über den Rahmen u
Ausführungen hinausführen. Es genügt darauf hinzuweisen, daß
an Stelle des früher hauptsächlich bevorzugten pararektalen Sc
der sogenannte Zickzack- oder Wechselschnitt nach Riedel de
zug verdient, welcher lateral von der Rektusscheide angelegt
stumpfer Durchtrennung des Musc. obl. externus, internus und
versus in der Faserrichtung auf das Peritoneum vordringt und
eine möglichst ideale Restitutio ad integrum der Bauchdecke
Vorteil einer absolut sicheren Ausschaltung der Möglichkeit sp
Narbenhernien bietet. Bezüglich der Amputation des Append
der Stumpfversorgung sei noch erwähnt, daß die Methoden
heute so vielseitige sind, daß es nicht zu viel gesagt sein dürfte
man behauptet, daß sich heute dazu fast jeder Chirurg seiner e
Methode bedient. Der einzige Unterschied dieser Methoden
meist nur in der technisch leichteren oder schwierigeren Ausf
derselben, wobei für den einzelnen noch der Umstand in Frage
auf welche Methode er gewissermaßen besonders eingeschoss
Bezüglich des Heilungsvorganges selbst läßt sich wohl keiner
zahlreichen Methoden ein besonderer Vorzug und Nachteil nac
da sie in der Hand eines geübten Operateurs jederzeit gleic
Erfolge gezeitigt haben.

Hinsichtlich der medikamentösen Behandlung in den
Fällen, wo chirurgische Hilfe nicht zu erreichen ist, oder der
— heute ein ziemlich seltener Fall — nicht zu der Erlaubnis

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rgischen Eingriffs zu bewegen ist, kann nicht genug vor der
lich mechanischen und kritiklosen Verabfolgung von Abführmitteln
rnt werden, wie sie noch heute vom Publikum und leider auch
nicht so selten von Ärzten in allen Fällen von Leibschmerzen,
orbenem Magen und sonstigen Verdauungsstörungen in Gestalt
vorwiegend Rizinusöl und Kalomel verabfolgt zu werden pflegen,
ch sogar in den Fällen, welche vom Arzt bereits als Blinddarm-
ndung richtig erkannt worden sind. Wohl kann ausnahmsweise
al in einem ganz leichten Falle mit chronischer Obstipation eine
tige Medikation den Anfall kupieren, wie man die generelle
ndung dieses Mißbrauchs zu motivieren pflegt, in schwereren
n kann sie dafür aber um so sicherer zur Beschleunigung der
nden Perforation und nachträglichen Verhinderung der schützen-
Verklebungen in der Umgebung der Perforationsstelle neben
n vermehrten Austritt von Darminhalt durch die Perforations-
in die Bauchhöhle führen und so zur direkten Veranlassung der
olgenden tödlichen Allgemeininfektion des Peritoneums werden.
a man nun anerkanntermaßen keineswegs immer aus dem nach
n projizierten Krankheitsbild auf die tatsächlichen pathologischen
nderungen am Wurmfortsatz selbst schließen kann, so ist meines
ntens jede Verabfolgung von Abführmitteln per os in derartigen
n unter allen Umständen als ein Vabanquespiel mit dem Leben
atienten zu bezeichnen, dem eine wissenschaftliche Berechtigung
zugestanden werden darf, solange nicht diagnostisch eine sichere
einstimmung des äußeren und inneren Krankheitsbildes bei der
ppendizitis herbeizuführen ist.

erselben Indikation, den Darm zu entlasten, können vorsichtige
ufe mit reinem Öl, Wasser mit Öl und Glyzerin oder ähnlichen
zen in gleicher Weise gerecht werden, ohne gleichzeitig ein der-
s Risiko quoad vitam des Patienten nach sich zu ziehen.

gleicher Weise ist auch vor der üblichen Verabfolgung von
otizis, insbesondere des so beliebten Opiums zu warnen, welches
seits nur geeignet ist, durch Verdeckung der so überaus wichtigen
onealen Schmerzreaktion eine Verschlimmerung des Krankheits-
s in gefährlicher Weise zu verschleiern und andererseits die an sich
twendige physiologische Darmtätigkeit in unnötiger Weise schwächt
herabsetzt. In besonders schmerzhaften Fällen oder bei besonders
blen Patienten können wohl ausnahmsweise kleine Morphium-
n zur Erleichterung des Patienten angewandt werden, bei denen
wenigstens die lokale Einwirkung auf den Darm in Fortfall kommt.
n übrigen ist die beste Entlastung des Darmes in der Entziehung
Beschränkung der Nahrungsaufnahme auf das irgend zulässige

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Mindestmaß zu erblicken, so daß in der ersten Zeit am beste dünner Tee, Schleimsuppe oder geeiste Milch c. stdl. teelöffe und von manchen Autoren in den ersten Tagen überhaupt nicht abfolgt wird. Die Schwächung, welche die Patienten im allgen durch diese vorübergehende Nahrungsentziehung per os er können, steht in keinem Verhältnis zu dem Vorteil, welche die wissermaßen rein physiologische Entlastung und Ruhigstellun Darmes von jeder Arbeit für den weiteren Entwicklungsverla Erkrankung selbst bietet, und kann außerdem durch kleine und Nähreinläufe von c. 2stdl. 200 g oder permanente. Kochsalzin per rectum bis zu einem gewissen Grade ausgeglichen werden

Fassen wir nun nach den obigen Ausführungen das Prinz heutigen Anschauung über die Behandlung der Erkrankung des V fortsatzes und ihrer Folgezustände kurz zusammen, so könn dieselbe in folgenden wenigen Sätzen präzisieren:

1. Das sicherste und gefahrloseste Mittel zur Beherrschun sofortigen Kupierung des Krankheitsprozesses und damit zur mö schnellen und dauernden Wiederherstellung des Patienten ist d solute Frühoperation in allen Fällen, welche nicht innerhalb der 24 Stunden einen entschiedenen Rückgang bzw. völliges Schw sämtlicher Krankheitssymptome aufweisen.

2. Aber auch in den Fällen, welche für die Frühoperation z in die Hand des Arztes geraten, ist der sofortige Eingriff stet zuziehen, da die Gefahren auch des späteren Eingriffs in keiner hältnis zu den Zufällen und Komplikationen des Krankheitsve stehen, von denen der Patient bei der Überführung in das Siche stadium dauernd bedroht bleibt.

3. Bei abwartender Behandlung ist am besten eine rein p logische Entlastung und Ruhigstellung des Darmes durch eine lichst weitgehende, wenn nicht vollständige Entziehung der Nah aufnahme per os in den ersten Tagen der Erkrankung anzus Der Ausfall an Nährstoffen kann durch entsprechend konzen und häufige, kleine Nährklistiere in gewissem Grade ersetzt w

4. Vor einer medikamentösen Entlastung des Darmes durch A mittel per os kann nicht dringend genug gewarnt werden, da dieselben eine erhöhte Möglichkeit zur Beschleunigung der Perf und nachfolgenden Allgemeininfektion des Peritoneums gegeben

5. Vor einer medikamentösen Ruhigstellung des Darmes Opium und ähnliche Derivate muß gleichfalls gewarnt werden, durch gleichzeitig eine gefährliche Verschleierung des Krankheits und eine Beeinträchtigung der physiologischen Gewebsvitalität u tätigung der Darmwand herbeigeführt wird. (Schluß folgt als Nr.

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492/93.

(Gynäkologie Nr. 179/80.)

Der praktische Arzt und die Händegefahr.

Von

Prof. Dr. **F. Ahlfeld,**
Marburg a. d. Lahn.

Mit acht Tafeln.

Kommt der angehende Arzt von der Universität, nach absolviertem ktischem Jahre, aus den mit dem ganzen modernen Komfort aus-isteten Kliniken und Krankenhäusern in die Praxis, dann wird ihm l bange, wie das in Zukunft werden soll. Naturgemäß muß er in Alltagspraxis vielerlei missen, wovon der gute Erfolg eines Ein-'s in der Klinik abzuhängen schien. — Nur darf er im Unterlassen hylaktischer Maßregeln nie unter das absolut Notwendige hinunter-en.

Ganz besonders groß sind die Unterschiede der äußeren Bedingungen einer geburtshilflichen Tätigkeit. Nicht zum geringsten beruhen die Schwierigkeiten auf der in den meisten Kliniken und Lehr-nern gepredigten Lehre vom Unvermögen, die Arzthand ungefährlich machen, eine Lehre, auf der sich notgedrungen ein ganzes Gebäude lerner Vorsichtsmaßregeln aufbauen muß, eine Lehre, die den ge-senhaften Arzt zu öfteren Unterbrechungen seiner geburtshilflichen gkeit zwingt, wenn er nicht gar an der Möglichkeit, Geburtshilfe en seiner anderen Praxis treiben zu können, verzweifelt.

Meine Erfahrungen, die ich in dieser Abhandlung niedergelegt habe, sich gleichzeitig auf zahlreiche Experimente im Bezug auf Hände-nfektion stützen, sollten den Kollegen Mut machen, einer sorg-en Händedesinfektion zu vertrauen, wo andere Schutzmaßregeln unlich sind.

Es würde meinen Auseinandersetzungen die Beweiskraft fehlen, lte ich einfach den jetzt gebräuchlichen Lehren andere entgegen-en. Es war nötig, die Einwände der Gegner einzeln zu besprechen damit auch dem Leser zu ermöglichen, sich selbst ein Urteil zu en, auf wessen Seite das Recht sei.

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Nimmt der Leser zu dieser Arbeit noch die Nummer 310/1 Klinischen Vorträge hinzu, — Die Desinfektion der Hand des rurgen und Geburtshelfers — dann kann er sich recht wohl übe Fragen, die in der Lehre von der Händedesinfektion und der ge hilflichen Abstinenz haben besprochen werden müssen, Aufklärun schaffen, und ich hoffe gewiß, er wird sich meiner Überzeugu schließen, daß bei gewissenhafter Händedesinfektion eine Schä der dem Arzte anvertrauten Frauen nicht zu erwarten ist.

Ist es nicht verlorene Zeit und Mühe, immer und immer v den Versuch zu machen, die führenden Persönlichkeiten, Chi und Gynäkologen, von der außerordentlichen Wirkung des Al als Händedesinfiziens zu überzeugen? Ist zurzeit Aussicht vorh eine Umstimmung der Meinungen herbeizuführen, wo die überwie Mehrzahl der Fachkollegen sich auf eine bestimmte Ansicht, di zulänglichkeit der Heißwasser-Alkohol-Desinfektion, festgelegt ha nicht gerade der jetzige Zeitpunkt besonders ungeeignet, wo in gangenen Jahre, auf dem Dresdener Gynäkologenkongreß, die Referenten über das Thema „Asepsis bei gynäkologischen Operati Fritsch und Küstner, sich abweisend gegen meine Untersuch und Resultate ausgesprochen haben, der erstere in einer ganz ders entschiedenen Form, und die Anwesenden sich mit diese schauungen einverstanden erklärt haben?[1])

Hätte ich nicht auf der anderen Seite die Beweise, daß trotz mächtigen Koalition, die, wie ich früher nachgewiesen habe, Hauptsache einer Schule entspringt, der sich die anderen füge die Mehrzahl der Urteilenden selbst keine eigenen Studien g hat, doch die Stimmen sich mehren, die dem Alkohol sein Re gestehen, und hätte ich nicht selbst, bis in die neueste Zeit, Versuche angestellt, die in ihren Endresultaten mit meinen fr übereinstimmen und durch die ich, unter Benutzung überzeu neuer Methoden, die Gegengründe widerlegen zu können glau würde ich wenig Hoffnung haben, mit einem neuen Appell Kollegen etwas auszurichten.

Aber ich will aus der Tätigkeit eines Lehrers für angehend nicht scheiden, ohne nochmals meine Überzeugung auszusprech zu begründen, daß von allen Methoden, die Hand für chirurg

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1) Die Hinweise auf meine Publikationen über Händedesinfektion gebe Wiederholungen zu vermeiden, nur mit Zahlen. Die Zahl in Kursivziffern bezi auf das am Schlusse angefügte Verzeichnis, die andere bedeutet die Seiten

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geburtshilfliche Eingriffe geeignet zu machen, bisher Heißwasser-Alkohol-Desinfektion diesen Zweck am einsten und besten erfüllt und daß, wenn auch der Gummischuh für Anstaltsärzte und Spezialisten bis zu einem gewissen e einen Ersatz für ungenügende Händedesinfektion bietet, er für Gros der Ärzte, für Hebammen und das niedere Heilpersonal Errungenschaft minderen Wertes bleibt.

h beginne mit dem Referate über Versuche der Reinigung frisch infizierten Hand.

n Sommersemester 1907 habe ich mich an die Beantwortung Frage gemacht, die im Verhältnis zu ihrer eminenten Wichtigbisher nur sehr stiefmütterlich behandelt ist. Meine Versuche n Aufschluß darüber geben, ob die mit virulenten Keimen erührung gekommene Hand alsbald nach der Beschmut- in einem Grade zu reinigen sei, daß eine weitere chiische und geburtshilfliche Tätigkeit unbedenklich geet werden könne.

ırzeit lautet die Antwort nein. Der Arzt soll eine Handinfektion ngehen versuchen; war sie aber dennoch erfolgt, so soll er eine che Abstinenz von chirurgischer und geburtshilflicher Tätigkeit lten, im Notfalle Gummihandschuhe gebrauchen. Eine, auch die lichste, Händedesinfektion genügt unter solchen Umständen ; das ist die verbreitete Ansicht der Fachgenossen.

uf die Frage, worauf sich diese Ansicht stützt, folgt unfehlbar Behauptung von der Unmöglichkeit einer absoluten Händekeimachung, und auf die Frage, welche Unterlagen für diese Behaupvorhanden sind, wird man von „unzähligen“ Versuchen sprechen, lle zum gleichen Resultate geführt hätten.

eshalb meine Resultate nicht die Berücksichtigung gefunden, ie verdienen, daran soll ich, wie mir Kollege Schäffer sagt, die Schuld tragen: „Indem Sie den Unterschied zwischen einer ität in praktisch-chirurgischem Sinne und einer ‚absoluten‘ Sterinicht anerkennen, machen Sie es den Gegnern der Alkoholfektion leicht, Gegenbeweise zu erbringen und damit scheinbar die Methode zu diskreditieren.“[1])

u meiner Rechtfertigung und zur ferneren Berücksichtigung in r Abhandlung folgendes:

ie Frage, ob eine Hand mittels der Heißwasser-Alkoholmethode lut keimfrei gemacht werden kann, muß in zwei Unterfragen nnt werden:

[1]) Aus einem Briefe.

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1. Dringt nach einer Vorbereitung mit Heißwasser und S
Alkohol so tief in die Haut ein, daß er die in den tieferen S
befindlichen Keime erreicht, um sie abtöten zu können? und
2. werden bei einer geübten und gewissenhaften Ausführ
Methode alle Stellen der Hand vom Alkohol derart erreicht,
Alkohol gegenüber den dort befindlichen Bakterien seine Schu
tun kann?

Da ich die erste Frage mit ja beantwortet habe und mit al
schiedenheit der Annahme einer Scheindesinfektion entgegen
bin — eine Überzeugung, die von den Gegnern nicht geteilt
so habe ich nach dieser Seite hin von einer absoluten
gesprochen.

Die zweite Frage habe ich niemals mit ja beantwortet, da
führung der Desinfektion von der subjektiven Tätigkeit, auch
mehr oder weniger günstigen Beschaffenheit der Hände abhä
kann daher nach dieser Seite hin niemals von einer Gara
Keimfreimachung reden. Die zu erreichen, liegt, günstige Bedi
vorausgesetzt, nur in der Macht des Ausführenden.

Daß, wenn alle nötigen Bedingungen zusammenkommen, d
wirklich absolut steril werden kann, muß ich annehmen und
ich an. Wie oft dies in Wirklichkeit eintrifft, das zu überseh
wir außerstande. Können wir doch, wie Schäffer ganz ric
merkt, nicht einmal für die ohne Zweifel sterilisierbaren Gege
als Instrumente, Verbandmaterial, Gummihandschuhe usw., im
falle sagen, sie seien tatsächlich steril. Und doch benutzen
vertrauensvoll unter Annahme des positiven Erfolges.

Wenn eine absolute Sterilität aber auch nur im Einzelfalle
so hätte ich nicht zu viel gesagt, denn ich habe nur die Mög
einer solchen behauptet. Dabei wahre ich mich ausdrücklich,
ich hiermit einen Rückzug eingeschlagen hätte, und füge deshalb
Die Bedingungen, eine wirkliche Sterilität zu erre
sind für viele Hände gegeben.

In einer früheren Arbeit[1]) habe ich, im bezug hierauf,
ausgesprochen: „Mit der Behauptung, mittels der Heißwasser-
Alkoholmethode läßt sich die Hand bis in die Tiefe der Hau
machen, deckt sich nun keineswegs eine Garantie für diesen
Der Prozentsatz der erreichten absoluten Sterilisierung de
hängt von vielen Faktoren ab, immerhin aber von Faktoren,
nicht zu schlechter Hautbeschaffenheit, bei gutem Willen des H
den vorhanden sein können, Ausnahmen zugegeben."

1) *26.* 1839.

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n weiterer Grund, weshalb meine Methode sich nicht, wie es all sein müßte, allgemeine Geltung verschafft hat, liegt in dem nde, daß zur Zeit, als sie sich Terrain erobern wollte, die Fabri- von Gummihandschuhen gelang. Wären diese nicht erfunden, irde sich jetzt jeder gewissenhafte Operateur mittels Heißwasser-Alkohol desinfizieren. Vielleicht, daß er noch irgend etwas lüssiges hinzufügte.

r dritte Grund liegt in persönlichen Verhältnissen, deren ich rschiedenen meiner Publikationen[1]) genügend Erwähnung getan Wäre die Empfehlung und das Eintreten für den Alkohol als desinfiziens von einer anderen Seite ausgegangen, dann würde ethode längst schon Gemeingut aller Chirurgen und Geburts- sein, und den Hebammen würde sie als einzige Desinfektions- de verordnet werden.

es vorausgeschickt, gehe ich nun zu der Frage über, zu deren wortung ich die angegebenen Versuche gemacht habe, inwie- die jetzige Anschauung von der Gefahr einer Auto- tion berechtigt ist und ob Abstinenz und Gummihand- n wirklich die einzig erlaubten Gegenmittel sind.

n der Beantwortung dieser Fragen hängt meines Erachtens die nz des praktischen Arztes ab. Fällt die Antwort dahin aus, daß risch infizierte Hand in Bälde nicht gereinigt werden kann, so der praktische Arzt, der sich vor derartigen Verunreinigungen Hand nicht schützen kann, chirurgische und geburtshilfliche in der Hauptsache an Krankenhäuser verweisen.

e Konsequenzen einer derartigen Beantwortung machen sich geltend. Unter dem Titel „Septicus" macht Poges in der kologischen Rundschau, 1907, Nr. 1 den Vorschlag, der Privat- olle sich so verhalten, wie die Ärzte von gut geleiteten Anstalten , die, um nicht mit septischem Material in Berührung zu kommen, wa erforderliche Behandlung eines verdächtigen Falles dem vor- nen septischen Assistenten, dem Septicus, überlassen. Um Abstinenz auch in der Außenpraxis sicher durchzuführen, sollen n größeren Städten wohlausgebildete Spezialisten vorhanden an die jeder infektiöse und infektionsverdächtige Fall abgetreten n müßte.

gen diesen Vorschlag lassen sich viele Einwände bringen. Vor daß er nur für große Städte in Frage kommen würde, während ros der Ärzte sich selbst behelfen müßte. Weiter wäre es wohl

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Verhandl. der Deutsch. Gesellsch. für Gyn. 1901, IX, S. 237. — Deutsche Wochenschr. 1904, Nr. 50, S. 1839. — Zentralbl. für Gyn. 1906, Nr. 3, S. 76.

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für das Publikum höchst fatal, wenn es von dem gewünschten
und Helfer an den „Kindbettfieberarzt“ gewiesen würde und
zugleich eine bedenkliche Prognose ausgesprochen wäre. W
weiter, zumal im Beginn einer Erkrankung, sagen, ob ein
septischer ist oder nicht? Sind wir doch gerade zurzeit in ein
haften Diskussion, welche Fieberfälle man Kindbettfieber nenn
welche nicht.

Und so könnte ich mit Gegengründen fortfahren. Ich glau
Vorschlag hat keine Aussicht auf Verwirklichung, wenn au
Düsseldorfer Kollege, Herr Dr. Wederhake, in einem Refer
erwähnten Vorschlags uns mitteilt, daß Düsseldorf derartige
listen schon aufweise, und sein Urteil dahin abgibt: „Man kann
Vorschlag nur mit Freuden begrüßen und im Interesse der le
Menschheit wünschen, daß von ihm recht ausgedehnt Gebra
macht würde.“

Geht man auf die Materie genauer ein, kritisiert man die ei
Behauptungen, die als Grundlage von der Forderung der No
Infektion und Abstinenz dienen sollen, so kommt man auf s
unbewiesene Annahmen, daß man sich verwundern muß, wie
ungenügend fundierte Lehre die Zustimmung unserer hervor
sten Fachvertreter gewonnen hat.

Machen wir uns erst klar, was man unter Auto-Noninfekt
unter Abstinenz versteht. Im Lehrbuche der operativen Gyni
von Doederlein-Krönig, 2. Auflage, finden wir, Seite 18,
Definition: „Unter Noninfektion verstehen wir die möglichst
haltung oder Vermeidung des Kontakts unserer Hände mit infe
Material, z. B. Eiter, Lochialsekret infizierter Wöchnerinnen
„Unter Abstinenz verstehen wir die zeitweise Suspension vo
tischen Operationen, wenn unsere Hände mit septischen oder
schen Stoffen in Berührung gekommen sind.“

„Beide Maßnahmen, Noninfektion und Abstinenz, müsse
als gleichwertige Faktoren bei der Unsicherheit der chemische
infektion neben dieser für die Asepsis der Hände verlangt w

Menge verschärft diese Anschauungen noch wesentlich.
Winckels Handbuch der Geburtshilfe, 1. Band, 2. Hälfte, 19
er, Seite 1196: „Durch die in der Berufstätigkeit sich immer
holenden Verunreinigungen mit Infektionsstoffen ist dafür geso
die Geburtshelferhand ihren drohenden Charakter so gut wie
liert.“ „Der Arzt, besonders der allgemeine Praktiker, der sic
tischer Arzt, Wundarzt oder Chirurg, und Geburtshelfer ne
vielleicht noch gefährlicher wie die Hebamme.“

Geht schon aus diesen beiden Zitaten hervor, daß der pr

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t, nach Menges Meinung, kaum in der Lage sein kann, jemals e ärztlich brauchbare Hand zu besitzen, so geht der folgende, e 1197 ausgesprochene Satz noch weit über diese Zitate hinaus: ch das Badewasser spielt als heterogene Infektionsquelle überhaupt ne oder eine nur ganz untergeordnete Rolle, „vorausgesetzt, daß Bad für die Kreißende nicht von der gefährlichen Hand der urtsleitenden Person gerichtet wird". Also selbst die wohldesinerte Hand eines Arztes, einer Hebamme darf nicht in Berührung dem für eine Gebärende zurechtgemachten Badewasser kommen, die Gefahr besteht, die Hand könne Keime in das Badewasser übergen, die dann auf dem Wege per vaginam Unheil anrichten könnten. Da Fritsch, im Anschluß an Doederlein, Krönig und Menge, in allerneuester Zeit als ein Hauptvertreter dieser Lehre bekennt in präziser Weise seinen Standpunkt gelegentlich eines Referats r Asepsis bei gynäkologischen Operationen niedergelegt hat, so e ich mich im weiteren an diese neueste Publikation und setze kurze Referat[1]) verbotenus meinen Ausführungen voran:

„Abstinenz. Schwieriger liegt die Frage, wie wir die Hände die Wunde, die sie anlegen und berühren, ungefährlich machen en. Dazu ist die erste, jetzt überall anerkannte Bedingung, daß Operateur septische Kranke oder Leichenteile überhaupt ht anfaßt, daß er ferner unsaubere Sachen nie mit unbedeckten, eschützten Händen resp. ohne Gummihandschuhe berührt. Allein gilt diese Regel: Die Forderung der Noninfektion, wie es önig nennt, und die Abstinenz, d. h. daß, wenn man zufällig oder Bewußtsein septische Gegenstände doch berühren mußte, der erateur eine Zeitlang, 3 oder 4 Tage, das Operieren unterläßt bzw. ein Arzt, der dergleichen gefährliche Sachen berührt, bei einer eration nicht assistiert, ja nicht einmal zuschaut. Selbstverständ- werden dann Bäder und verschärfte Desinfektion gefordert.

Diese Forderung ist übrigens so alt als die Worte oder Begriffe: ktion und Desinfektion. Schon Semmelweis hat ja beides verlangt. Ein Operateur soll sich von infektiösen Menschen und Dingen nzipiell fernhalten. Kam doch schon vor Jahren ein Operateur in llagezustand, weil er nach Spaltung eines Furunkels eine Laparotomie gemacht hatte, obwohl er vor der Laparotomie sich sorgfältig nfizierte. Diese prinzipielle Noninfektion und Abstinenz gilt jetzt rall als Grundsatz."

Soweit Fritsch. Setzen wir nun statt „Operateur" „Arzt, der zu rieren gedenkt oder operieren muß", übertragen wir, wie es ja

1) Verhandl. der Deutschen Gesellsch. für Gynäkologie 1907, XII, S. 472.

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auch Fritsch tut, die Frage aus der Klinik heraus auf die allge
Praxis, so stehen wir zuerst vor der Frage: Welches sind „sep
Kranke", was sind „unsaubere Sachen", was „septische Gegenst
„dergleichen gefährliche Sachen", vor deren Berührung sich der
tische Arzt hüten soll?

Nun sind ja einige der gefährlichsten Krankheiten bekannt,
Giftstoffe, auf frische Wunden übertragen, mit einer gewissen
scheinlichkeit wieder Blutvergiftung erzeugen. Aber können wir
lich diese Fälle in ihrem Beginn, vor der Berührung mit dem Kr
immer als solche zeitig genug erkennen, um Vorsichtsmaßreg
treffen, um eine Behandlung abzulehnen? Genügt nicht scho
Besuch in der Krankenstube, das Reichen der Hand, die der Kr
den Gruß bietet, Giftstoffe aufzunehmen? Soll man die Krank
ihre Hoffnung auf den Arzt gesetzt hat, mit der Weisung wiede
lassen, ein anderer Arzt, der Herr Septicus, solle die Behan
übernehmen? Kann nicht die dadurch entstehende Verzögerung
Eingriffs, auch wohl die seelische Erregung, unheilvolle Folgen h

Und weiter, wenn nun der praktische Arzt sicher in letzte
es mit keinem der manifesten septischen, ich will sagen gefähr
Krankheitsfällen zu tun gehabt hat, kann er sich dann wirkli
ruhigem Gewissen darauf verlassen, daß er nicht doch irge
und wie septische Stoffe berührt habe? Ist es nicht vielleich
tiger, jede Ärztehand zu jeder Zeit als eine solche anzusehe
ohne die sorgfältigste Händedesinfektion keinen chirurgischen,
geburtshilflichen Eingriff wagen soll, bei dem eine Berührun
Wunden stattfindet?

Ich mag die Frage ansehen von welcher Seite ich will, es
sich für den praktischen Arzt die Unmöglichkeit, operative (auch ge
hilfliche) Praxis zu treiben, wenn diese extremen Anschauunge
sie im obigen Referate vertreten werden, sein Handeln bedingen m

Ebensowenig komme ich mit Befriedigung über die Forderu
Abstinenz hinweg.

Was ihre Dauer anbetrifft, so spricht Fritsch von 3 oder 4
Doederlein-Krönig stützen sich auf Zweifel und schreiben:
Zweifel wird auf Grund klinischer Erfahrungen eine 3 Tage wä
Abstinenz verlangt und von einer großen Zahl Operateure geb
Die deutsche Prüfungsordnung für das ärztliche Staatsexame
8 Tage fest. In der Hebammenpraxis werden 8—14 Tage Kar
angeordnet.[1])

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1) Hauptversammlung des Preußischen Medizinalbeamten-Vereins, Beric
26. Sept. 1898, S. 25.

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Vie soll nun diese Zeit händereinigend wirken? Findet in diesem aume eine Abschuppung statt, die alle septischen Keime mit fort-t? Oder soll die Zeit dazu dienen, durch häufige gründliche gungen die „gefährlichen" Keime zu beseitigen? Ließe sich dies gutem Willen und Verständnis nicht in einer kürzeren Zeit aus-n?

Wir gehen", schreiben Doederlein-Krönig, „hier von der Vor-tzung aus, daß durch die stets vor sich gehende Abschuppung bersten Epidermisschichten die anhaftenden pathogenen Bakterien anisch entfernt werden, oder daß sie durch die ihnen auf der tten Hautoberfläche aufgezwungene saprophytische Lebensweise Virulenz verlieren."

sführlicher geht Menge auf diesen Punkt ein. Seite 1296 im ten Teile des v. Winckelschen Handbuchs setzt er auseinander, elche Vorgänge der Haut die Reinigung zurückzuführen sei. keiner dieser Publikationen ist angegeben, auf welche Unter-ngen oder praktischen Erfahrungen sich diese Annahmen stützen. ie überhaupt richtig sind, ist wiederum eine der zu beantwor-n Fragen.

nn — eine andere Frage — der Arzt in der Abstinenzzeit nicht wußt von anderer Seite wieder eine Körperinfektion erfahren? ierigkeiten über Schwierigkeiten, Fragen über Fragen, an deren wortung man doch hätte eher gehen müssen, ehe man solche se Forderungen an den Arzt stellt.

eser Vorhalt scheint mir um so berechtigter, da ja Fritsch die Möglichkeit zugibt, durch eine besonders sorgfältige Hände-fektion wäre der praktische Arzt imstande, sich derart zu rei-, daß er operative Eingriffe aseptisch und mit gutem Erfolge hren könne. Im Fortlauf seines Referats heißt es: „Ich möchte aber doch einschieben, daß ich auch nach zufälliger Verun-gung der Hände eine erfolgreiche Desinfektion für möglich halte. ich doch Jahrzehnte hindurch mit unbedeckten Händen allerhand che Operationen gemacht und unmittelbar danach, allerdings sorgfältigster, wenn ich so sagen darf, doppelter und dreifacher fektion, nach Überdesinfektion, wieder aseptische Operationen gutem Erfolge ausgeführt. Ich würde es nicht wieder tun, weil s für prinzipiell unrichtig halte, ich habe es auch seit Jahren wieder getan; aber für möglich halte ich es auch heute noch, septisch verunreinigte Hand schnell wieder in dem Grade keim-zu machen, daß sie ungestraft eine aseptische Operation ausführen Das muß man zur Beruhigung der Ärzte sagen, die doch oft anders handeln können."

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Mit der „Beruhigung“ kann sich aber der praktische Ar
zufrieden geben. Er muß Sicherheit haben. Er muß auch Ge
haben, daß, wenn er sich mittels einer bewährten Methode un
nauer Berücksichtigung aller zum Erfolg nötigen Punkte des
hat, ihm eine Anklage keine Verurteilung bringen kann.

Auch Menge gibt bis zu einem gewissen Grade die Mög
zu, eine frisch infizierte Hand sofort zu reinigen: „Nachgewi
maßen lassen sich die infektiösen Massen am leichtesten un
kommensten von der Haut mechanisch wieder entfernen, w
noch nicht eingetrocknet waren.“

Gleich im nächsten Satze schränkt Menge aber den A
„vollkommensten“ erheblich ein: „Niemals wird aber die mit
infektiösem Materiale in Berührung gewesene Handhaut du
sofort angeschlossene mechanische und chemische Desinfektio
gefährlichen Charakters absolut sicher entkleidet. Sie bleib
nach diesen Reinigungsprozeduren verdächtig und stellt, be
wenn sie mit streptokokkenhaltigem Materiale in Kontakt getre
den Geburtsleiter vor die Frage von der sogenannten Absti

Versuche über Frischdesinfektion. Die obengestellt
habe ich auf folgende Weise zu beantworten versucht:

Meine eigenen Finger, die meist 48 Stunden mit keinem
fiziens in Berührung gekommen waren, habe ich, wie es die
ergibt, mit den verschiedensten Stoffen beschmutzt, die man al
als „infektiöse Stoffe“ bezeichnet. Ich habe Mastitiseiter, Ka
jauche, Eiter parametraner Abszesse, Zystitiseiter, Lochien vo
peralkranken Frauen, Darmkot, Lochien nicht fiebernder Person
an die Hand gebracht und mich nach Beendigung der ärztliche
pulation zunächst mit warmem Wasser, Seife und Bürste g
wie man es in der Praxis ausführen kann. Dieser Reinigu
ich eine Alkoholwaschung mit Bürste oder Flanell folgen las
dann wiederum die Oberfläche der Hand auf Bakteriengehal
sucht.

Vorversuche bezogen sich auf die Beschaffenheit der
hand, d. h. der Hand, die absichtlich die Berührung „infe
Stoffe vermieden hatte.

Zu diesen Versuchen benutzte ich Petrischalen, da es dar
kam, die Zahl und Art der Kolonien zu bestimmen. In diese
Serie begnügte ich mich damit, die Fingerkuppen auf der Vol
auf der Nagelseite strichweise über den Agar weggleiten zu l

Schon seit Jahren benutze ich diese Methode, um im Begi
Hebammenunterrichts den Schülerinnen zu zeigen, welch gro
von Keimen sie an ihrer Tageshand haben, selbst wenn sie n

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nken und Wöchnerinnen zu tun hatten. Die so gewonnenen Bilder
derart überzeugend, daß sie weit besser wirken als lange Reden
Auseinandersetzungen.

Für meine jetzigen Versuche war es aber nötig festzustellen, daß
die gepflegte Arzthand, in diesem Falle die meinige, auch wenn
nicht in direkte Berührung mit „gefährlichen Fällen" gekommen
stets durch einfaches Bestreichen der Agarplatte eine reiche Aus-
bewirkt.

Freilich auch in dieser Beziehung muß mit einiger Vorsicht das
ultat beurteilt werden, denn tatsächlich sind nicht alle Kolonien,
auf der Platte wachsen, Handkeime.

Obwohl ich die verwendeten Petrischalen mit einem kräftigen
nmiring derart versah, daß die überstehende Platte fest an die
rragte angedrückt wurde, obwohl das Öffnen beim Bestreichen des
r mit dem Finger nur wenige Sekunden Zeit brauchte, kamen doch
einem nicht geringen Prozentsatz Luftverunreinigungen vor. Vor
r Täuschung schützte ich mich, wenigstens in etwas, daß ich die
saat mit dem Finger in ganz bestimmten Figuren machte, die ich
dann im Protokoll einzeichnete. Wenn Keime weitab von diesen
ihrungsstellen sich entwickelten, so war ich berechtigt, sie als
t vom Finger stammend anzusehen.

Auch Sarwey[1]) hatte bei seinen Experimenten, obwohl er sich
ßere Mühe gegeben hat, das Einfallen von Luftkeimen zu verhüten,
chschnittlich auf vier bis fünf sterile Platten eine mit je ein bis
aufgefallenen Luftkeimen.

Als Endresultat dieser Vorversuche ergibt sich, was zu erwarten
: Die Tageshand, wenn sie nicht vorher mit einem Des-
ziens in Berührung gekommen, bewirkte auf unseren
rplatten durch einfaches Bestreichen ausnahmslos eine
che Aussaat von Keimen.

Ich kann daher in den weiteren Experimenten mit gutem Recht
auffallende Abnahme der Keime als Folge einer Reinigungsmaß-
l ansehen.

Um dem Leser ein Urteil zu ermöglichen, in welchem Grade die
erfläche der Hand ihren Keimgehalt ändert, wenn diese oder
Behandlung vorausgegangen ist, benutzte ich die Photographie,
damit, was Zahl und Verteilung, bis zu einem gewissen Grade
die Art der Kolonien anbetrifft, jede subjektive Deutung wegfällt.
Ich beginne mit der Darstellung der Bilder der Aussaat von der

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1) Bakteriologische Untersuchungen über Händedesinfektion. Berlin 1905,
5 u. f.

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Tageshand von Frauen, die nichts mit Kranken zu tun hatten. wählte ich die Hände von Schülerinnen, die frisch eingetreten und die ersten 8 Tage des Kursus sich — ohne Desinfiziens — r gelernt hatten. Ich ließ auf die Agarplatte eine 3 schreiben.

Das Resultat zeigen die Abbildungen 1 und 2.[1]) Jedes Se wo ich dieses Experiment als Unterrichtsmittel benutzte, erg der gleiche Erfolg. Ich mache auf die abseits der Zahl befin Luftkeime aufmerksam.

Auch mit meiner Hand, die am Morgen mit Seife und Was waschen, mit keinem Desinfiziens in Berührung gekommen wa keine Stoffe angefaßt hatte, die als septische oder pyogene aufz waren, machte ich das gleiche Experiment und erzielte ähnlic sultate, wenn auch die Kolonien nicht in der Massenhaftigk zeigten wie auf den Platten der Schülerinnen. Siehe Fig. 3.

Nach diesen Vorversuchen nahm ich nun die Experimen künstlicher Beschmutzung und gleich darauffolgender gung vor. Was Plan und Ausführung anbelangt, muß ich die ve denen Serien zunächst zusammen besprechen, um dann an C Stelle auf die Abweichungen einzugehen. Hingegen möchte ic unerwähnt lassen, daß, nachdem ich mehrere Wochen lang Krankenhaus gekommen war, mit Bewußtsein auch keine sep Stoffe berührt, mich in dieser Zeit auch nicht desinfiziert ha Abschabsel meiner Handoberfläche in die Bauchhöhle mehrerer und eines Meerschweinchens eingespritzt, keinerlei Entzünd scheinungen septischer Art hervorgerufen haben. Die Tiere vollständig munter.

Die Verhältnisse der Praxis nachahmend, nahm ich, nur Abweichung, keine Desinfektion der Hand vorauszuschicke Untersuchung von Frauen vor, die an den verschiedensten kungen litten und einen Ausfluß hatten, dessen Bestandteile m gemein unter die „gefährlichen“ Stoffe rechnet.

In der ersten Serie meiner Versuche strich ich die schmutzten Finger alsbald nach Beendigung der ärztlichen Vo über eine Agarplatte ab, indem ich mit der Volarfläche und d der Nagelseite Figuren, als Striche, Bogen, Kreise, Zahlen Platte zeichnete.

Nun wusch ich die beschmutzte Hand zunächst mit warmem (Wasserleitungswasser) in den Waschbecken meines Untersuc zimmers und mit gewöhnlicher Toilettenseife und fügte da

1) Sämtliche Platten sind, wenn nicht speziell anders angegeben, 48 nach der Beschickung photographiert worden.

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ürstung mit einer gewöhnlichen Wurzelbürste hinzu, die bis kurze
t vorher in Alkohol gelegen hatte, aber etwa 5 Minuten lang in
mendem warmem Wasser vom Alkohol befreit war. Beide Proze-
en zusammen dauerten 3—4 Minuten. Schließlich wurde die Haut
strömenden warmen Wasser von der Seife befreit und mit einem
gewaschenen, bis dahin nicht gebrauchten Handtuche die Hand,
ziell die Finger, abgetrocknet.

Jetzt erfolgte die zweite Aussaat, indem ich in gleicher Weise,
vorher mit den beschmutzten Fingern, nun mit den gereinigten
elben Figuren auf eine Agarplatte zeichnete.

Der Reinigung mit Wasser, Seife und Bürste folgte eine Alkohol-
nfektion nach meiner Methode mit Bürste und Flanell und dann
Entlaugung der Hand in strömendem warmem Wasser, um den
hol soviel als möglich zu beseitigen. Die feuchte Hand wurde
ch mit sterilem Mull gehörig getrocknet und eine dritte Aus-
t vorgenommen, wiederum die gleichen Zeichen, die ich bei der
en und zweiten benutzt hatte.

Von den zahlreichen Versuchen gebe ich die Bilder einiger Platten
füge das Gesamtresultat dieser Serie am Schlusse an.

. Untersuchung einer Frau mit jauchendem Portio-Karzinom. Auf der Platte drei nahezu parallel laufende Striche. 48 Stunden im Brütofen.
   a) Testplatte: Zahllose Kolonien längs der drei Striche, mit Bakterienarten, die nach okularer Besichtigung hauptsächlich den Streptokokken und den Staphylokokken (aureus und albus) angehören. Fig. 4.
   b) Platte nach der Wasser und Seifenwaschung: Zwei Kolonien, keine Streptokokken. Fig. 5.
   c) Platte nach der Alkoholdesinfektion: Eine Kolonie.

. Untersuchung einer Frau, bei der ein jauchender Beckenabszeß sich in die Scheide öffnet.
   a) Testplatte: Drei Striche von drei zur Untersuchung benutzten Fingern. Zahllose Kolonien. Fig. 6.
   b) Platte nach der Wasser- und Seifenwaschung: Vier Kolonien. Fig. 7.
   c) Platte nach der Alkoholdesinfektion: Drei nebeneinanderliegende Kolonien.

. Öffnung einer vereiternden Brustdrüse. Der ausfließende Eiter ergießt sich über die Finger.
   a) Testplatte: Drei Finger, mit Eiter beschmutzt, sind über die Platte weggestrichen. Zahllose Kolonien. Fig. 8.

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b) Platte nach der Wasser- und Seifenwaschung: Drei am gelegene Kolonien. Fig. 9.
c) Platte nach der Alkoholdesinfektion: Zwei Kolonien.

4. Untersuchung einer Wöchnerin mit übelriechendem Ausf
a) Testplatte: Drei Fingerstriche. Zahllose Kolonien. F
b) Platte nach der Wasser- und Seifenwaschung: Drei Ko Fig. 11.
c) Platte nach der Alkoholdesinfektion: Eine eng zusa liegende Gruppe aus mehreren Keimen entwickelt.

5. Untersuchung einer Wöchnerin mit Endometritis puerp hoch fiebernd.
a) Testplatte: Zwei Fingerstriche. Zahllose Kolonien. F
b) Platte nach der Wasser- und Seifenwaschung: Seh Kolonien.
c) Platte nach der Alkoholdesinfektion: Eine Kolonie g Rande. Fig. 13.

6. Untersuchung einer Frau mit jauchendem Beckenexsudat
a) Testplatte: Zwei Fingerstriche mit zahllosen Kolonien seits zwei Luftkeime. Fig. 14.
b) Platte nach Wasser- und Seifenwaschung: Zwei Ko die eine aus einer Gruppe bestehend. Fig. 15.
c) Platte nach der Alkoholdesinfektion: Steril.

Selbstverständlich habe ich den Einwand zu erwarten, d impfungsmethode sei eine viel zu zarte, nicht ausreichende. D wand ist voll berechtigt. Es sollen diese Versuche auch nur z beweisen, wie eine sofortige Reinigung der Hand, allein mit Wasser, Seife, Bürste und Handtuch, die Zahl der aufgenommenen infektiösen Keime in staunenswerter sichtlich vermindern kann.

In einer zweiten Serie der Versuche veränderte ich desh Abimpfungsmethode, indem ich als zweiten Akt nicht das e Bestreichen der Platte mit dem gereinigten Finger vornahm, s ich faßte kleine sterile Mulläppchen mit einer sterilen Pinzet die Finger, besonders die Nagelgegend kräftig ab, breitete da läppchen auf der Agarplatte aus und drückte es mit steriler P sanft dem Agar so auf, daß es ihm überall anlag.

Und, um das weiter anzuschließen, nahm ich in einer d Serie der Versuche die Abimpfung der Hand, des Nagelsau des Nagelfalzes mit sterilen Hölzchen vor, die mit steriler abgeschnitten auf die Agarplatte fallen gelassen wurden; auch ich sie mit zartem Druck gegen die Nährmasse.

Ich kann mich, was das Ergebnis dieser beiden Serien an

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fassen. Es unterschied sich nur sehr wenig von dem der ersten
Der Erfolg der einfachen Waschung mit heißem Wasser,
und Bürste war ein eminenter. Die frisch der Hand auf-
en Keime wurden auf diese Weise fast ganz oder ganz beseitigt.
e bisherigen Versuche bezogen sich aber immer nur auf die
ität der nicht beseitigten Keime und ließen außerdem noch die
chkeit zu, daß ein Teil dieser Keime nicht der Hand angehörte,
rn als zufällige Verunreinigungen aufzufassen war.
benutzte daher zu den weiteren Versuchen Bakterienarten, die
nfolge ihrer subjektiven Färbung aus dem übrigen Bakterien-
ch herauskennen kann und die nur selten als Luftkeime vor-
en, das Bacterium coli und den Prodigiosus.
n jeder dieser Serien sei ein Versuch als Paradigma beschrieben:
schmutzung zweier Finger und des Handrückens mit frischem
kot. — Abstreichen des Schmutzes auf eine Drigalski-Platte in
zweier Bogen mit der Volarfläche der beiden Finger, in Form
Striches mit der Rückenfläche, Nagelgegend (I).
aschen der Hand mit warmem Wasser und gewöhnlicher Seife,
en gröbsten Schmutz zu entfernen. Danach Waschen mit Bürste
Seife zusammen, ungefähr 4 Minuten lang. Abtrocknen mit
n Handtuche. Nun die gleichen Figuren auf eine zweite Dri-
-Platte (II).
rsten der Hand mit Alkohol, 5 Minuten. Entfernen des Alkohols
Auslaugen in heißem Wasser, 5 Minuten. Abreiben der vorher
mutzten Finger mit einem kleinen sterilen Mulläppchen, das mit
r Pinzette gefaßt wurde. Ausbreiten und Gegendrücken dieses
chens auf eine dritte Drigalski-Platte (III).
s Kontrollversuch wurde ein steriles Mulläppchen ohne jede
ge Behandlung auf eine vierte Drigalski-Platte gelegt (IV).
ler 24 Stunden Besichtigung der Platten und Eintragung in das
koll:
Platte I entwickelten sich schon nach 24 Stunden zahllose Keime
r Stelle der Zeichnung, auch zeichneten sich die geimpften Par-
schon nach 24 Stunden durch eine deutliche Rotfärbung aus, die
ung des Bacterium coli, die am nächsten Tage noch intensiver
en.
atte II blieb bis zum zweiten Tage vollständig keimfrei, auch
sich nirgends eine Rötung. Am 3. Tage waren zwei Kolo-
zu bemerken, die sich, nach Bestimmung im hygienischen Institut,
treptokokken erwiesen. Rötung trat nie ein.
latte III blieb zunächst keimfrei. Am 3. Tage waren am Rande,

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außerhalb des Zeichnungsgebiets zwei Luftkeime aufgegangen. trat nicht ein.

Platte IV blieb keimfrei, zeigte niemals Rötung. In ande suchen blieben die Platten von Nebeninfektionen ganz frei einem solchen Versuche gebe ich die Abbildung der Photo Die Zeichnung geschah in Form eines Ringes. Natürlich Photographie der Färbung der Platten halber nicht so deut wie die übrigen. Fig. 16.

Für die Versuche mit dem Prodigiosus ließ ich mir frisc turen aus dem hygienischen Institute geben, die, wie ich mich Zeit hindurch überzeugte, sehr prompt sich weiter entwickelt machte mir eine Aufschwemmung, die leicht rosa gefärbt war, meine Hand in dieselbe ein und ließ die Masse zunächst ant

Wie sonst bei den beschriebenen Versuchen zeichnete ich der Volar- und der Nagelseite Figuren auf eine Agarplatte (I dann die Hand mit Wasser und Seife und bürstete sie ca. 4— ten, wie immer die Nagelgegend ganz besonders berücksichtig zeichnete die gleichen Figuren auf eine Platte (II). Darauf kr mit sterilen spitzen Hölzchen Nagelgegend, Nagelbett und nagelraum aus und ließ die abgeschnittenen Spitzen in ein Schale gleiten, wo ich sie mit steriler Pinzette in den Aga eindrückte.

Die Platten blieben in Zimmerwärme stehen und auf Plat wickelten sich regelmäßig zahllose Kolonien des intensiv rote in Form der gezeichneten Figuren.

Platte II blieb in einigen Versuchen ganz frei von Prodigi anderen entwickelten sich einige wenige Kolonien.

Auf Platte III zeigten die Hölzchen, die ich zum Abkrat Haut benutzt hatte, meist keine Kulturen, hingegen die, mit d Unternagelraum und das Nagelbett ausgekratzt waren, zeigten, n nach mehreren Tagen, an der Spitze des Hölzchens eine si und nach ausbreitende Kultur.

Auch der Frage bin ich näher getreten, ob eine Hand, an die Schmutzteile eine Stunde und darüber hat antrocknen lassen heißem Wasser, Seife und Bürste in gleicher Weise oder we annähernd so wie sofort nach der Beschmutzung sich reinig

Zu dem Zwecke benutzte ich besonders Kotbeschmutzung Prodigiosus. Nachdem ich die Hand mit besagten Stoffen veru hatte, zog ich einen sterilen Gummihandschuh an und behielt i $1^1/_2$ Stunde an der Hand. Danach folgten dieselben Reinigun duren, wie ich sie beschrieben habe.

Das Resultat unterschied sich kaum von dem der übrigen

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n dieser Zeit vermochte die intensive Waschung die Bakterien in ällliger Weise zu beseitigen.

Fasse ich nun die Endergebnisse der verschiedenartigen Versuche mmen, so darf ich mit Sicherheit behaupten:

Bakterien, die erst kurze Zeit vorher auf die Oberfläche Hand gekommen sind, ohne daß man sie intensiv einrieb, en sich durch eine gewissenhafte Reinigung mit warmem sser, Seife und Bürste und Abtrocknen mit einem sauberen dtuche so weit entfernen, daß sie entweder nur in ein- en Exemplaren nachgewiesen werden konnten oder gänz- beseitigt wurden.

Beim intensiven Einreiben, ebenso beim Einreiben auf eine schon etzte Hand, kann das Resultat negativ ausfallen, die Hand, wenn ich um pathogene Mikroorganismen handelt, geschädigt werden, dies u. a. Lauenstein[1]), Engels[2]) und Opitz[3]) begegnete. Wäh- Lauenstein und Engels ein solches Vorkommnis als nicht eiskräftig gegen die Wirksamkeit der Desinfektionsmethode aus- iden, benutzt es Opitz im entgegengesetzten Sinne.

fügt man der eben beschriebenen gewissenhaften mecha- hen Reinigung noch eine genügende Abreibung mit Alko- hinzu, so erreicht man bestimmt Keimfreiheit der Ober- he, in der Mehrzahl der Fälle auch eine solche der tieferen ien.

Die Bestätigung dieses letzteren Satzes konnte ich nach einer puer- len Streptokokken-Autoinfektion an meiner und meines Assistenten d durch bakterielle Untersuchung erbringen und zwar nicht nur rt nach der Reinigung, sondern auch nach einer halben Stunde der Beschmutzung.[4])

Auffallenderweise ist die uns bisher interessierende Frage, inwie- infizierte Hände durch sofortige gründliche Waschung Infektionsstoff befreit werden können, nur sehr vereinzelt rimentell behandelt worden. In verschiedenen Arbeiten wird von Möglichkeit einer sofortigen Beseitigung frisch aufgebrachter Stoffe von einer Tatsache gesprochen, aber nicht angegeben, auf welchen uchen diese Tatsache beruht.

) Münchener med. Wochenschrift 1902, Nr. 30, S. 1251.
) Klinisches Jahrbuch 1905, 13. Bd., S. 610.
) Berliner Klin. Wochenschr. 1898, Nr. 39 und Deutsche med. Wochenschrift Nr. 51, S. 2064.
) *20.* 367.

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In der vorzüglichen Dissertation von Henke[1]) sind einige hi
gehörige Experimente berichtet. Henke wollte feststellen, ob die
infizierte Hand schwerer keimfrei zu machen sei, wie man
annahm, als die Tageshand. Er fand das Gegenteil, also ein
stätigung dessen, was auch meine Versuche ergeben haben.

Zu dem gleichen Resultate gelangte Seitz[2]), der seine Ve
unter v. Esmarchs Leitung im Göttinger hygienischen In
machte. Seine Ergebnisse decken sich mit den meinigen vollst
Selbst nach einer weit kürzeren Waschung (Seife, Wasser, s
Handtuch, eine Minute) gelang ihm bei 12 Versuchen die gänz
Entfernung der Kolibazillen, die vorher als Bestandteile frischer
auf die Hand gebracht waren. Weniger günstig, wie auch in m
Versuchen, fielen die Versuche mit Prodigiosus aus.

Auch Gottstein[3]) benutzte den Prodigiosus. Es gelang il
der Hälfte der Fälle den Prodigiosus schon mit heißem Wasser,
und Bürste zu beseitigen, während die normalen Hautepiphyt
diese Weise in keinem Falle beseitigt werden konnten. Hae
konnte mit Tusche oder Kienruß beschmutzte Hände durch sorg
Waschung wieder gänzlich reinigen. Waren aber die Hände
geworden, gelang das Experiment nicht mehr.

Ziehen wir nun die praktischen Konsequenzen aus diesen gesa
ten experimentellen Ergebnissen, so diktieren sie dem Anstalt
wie dem praktischen Arzte und der Hebamme, in allen Fälle
die Hand oder ein anderer Körperteil mit offenbar
zweifelhaft infektiösen Stoffen in Berührung gekomme
sich nicht mit einer gewöhnlichen Waschung der besch
ten Teile zu begnügen, sondern sofort eine genaue, best
alle Teile der Hand — ev. anderer Körperteile — tref
Reinigung mit warmem Wasser, Seife und Bürste vorzu
men. Ist es möglich, auch an Ort und Stelle gleich
typische Alkoholdesinfektion auszuführen, dann bring
Arzt seine Hand wieder in den Zustand, wie er vor de
fektion war.

Wenn Klemm[5]) schreibt: „Natürlich werden wir ja stets nach
tiven Eingriffen, nach Verbandwechseln, Untersuchungen an Kr

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1) Über die Desinfektion infizierter Hände und die Notwendigkeit der g
hilflichen Abstinenz. Inaug. Diss. Tübingen 1893.

2) Über Händeinfektion und -desinfektion. Zentralbl. für Bakteriologi
1904, Bd. 37, S. 726.

3) Beiträge zur Klin. Chirurgie, Bd. 25, S. 67.

4) Händereinigung, Händedesinfektion und Händeschutz. Basel 1900. S.

5) Deutsche Zeitschrift für Chirurgie Bd. 75, S. 541.

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die Hände genau so reinigen müssen, als ob wir eine aseptische
ation ausführen wollen", so folgen dieser „natürlichen" Verpflich-
nur wenige, nicht einmal sämtliche Anstaltsleiter. Schindler[1])
sogar diese Waschung für nutzlos. Auf die Frage: Kann die
nme sich gegen die venerische Ansteckung im Beruf schützen?
rtet Schindler: „Kaum! Desinfizierende Waschungen nutzen
vorher noch nachher, sonst würden sich eben nicht fortgesetzt
nmen anstecken." Daß sie vorher nichts nützen, ist selbstver-
ich. Wenn sie aber, nachher angewendet, nichts genützt haben,
sind sie entweder ungenügend gemacht, oder es hat eine Impfung
die Haut stattgefunden.

t ein Arzt gewissenhaft in der beschriebenen Weise gehandelt,
st er genötigt, kurze Zeit darauf einen Geburtsfall zu übernehmen
eine Operation auszuführen, so braucht er nur die genau aus-
te verschärfte Desinfektion vorauszuschicken, um mit gleichen
en zu operieren, als wenn er Gummihandschuhe verwendete.
braucht wohl nicht betont zu werden, daß er daneben den
ngsstücken als Infektionsträger die größte Aufmerksamkeit zu-
den hat, die in einem solchen Falle natürlich gewechselt werden
n. Besonders wichtig erscheint es mir, auf die Gefahr hin-
sen, die der gewöhnliche Handschuh mit sich bringt, den man,
man den Kranken verläßt, ohne die Hand, soweit es geht, keim-
emacht zu haben, natürlich innen beschmutzt und sobald man
äter benutzt, die inzwischen gereinigte Hand wieder infiziert.
t diesen meinen Schlußfolgerungen ist die Frage der Abstinenz
nem anderen Sinne beantwortet, als dies bisher die meisten
rgen und Gynäkologen tun. Es wird nicht ausbleiben, wie dies
auch geschehen: man wird Warnrufe vor diesem falschen
eten erschallen lassen.

komme gleich ausführlich darauf zu sprechen, wie wenig be-
et die Anschauungen derer sind, die von einer Abstinenz einen
erwarten. Hier sei das eine vorausgenommen, daß man dem
renden und dem angehenden Arzte gegenüber mit der Anerkennung
Wertes der Abstinenz zugleich eine Diskreditierung der Hände-
ektion vornimmt. „Denn durch diese Prohibitivmaßregel werden
verständlich die Studierenden nicht gelehriger oder geschickter
Kunst der Händedesinfektion gemacht werden, als sie es ohne
be sind und daß die Desinfektion als solche durch das Warten
er gemacht würde — diese Vorstellung muß, wie ich genügend

[1]) Die venerische Ansteckung der Hebamme im Beruf, Berlin 1907, als Manu-
gedruckt; S. 21.

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gezeigt zu haben glaube, gänzlich fallen gelassen werden. M
genaue Vorschriften im Desinfizieren der Hände, möglichst
Überwachung des Desinfektionsverfahrens vor den anzust
Manipulationen an den Kranken, fortgesetzte Bemühungen, das
desinfektionsverfahren immer noch vollkommener und siche
zubilden, als es bisher ist — das sind die rationellen und wi
Prohibitivmaßregeln gegen die Herbeiführung septischer usw. Inf
durch ungenügend gereinigte Hände der Studierenden, nicht
Maßregel der ‚geburtshilflichen Abstinenz', welche nur ande
tige Fächer der Medizin schädigt, ohne der Geburtshilfe zu
So schreibt vor Erfindung der Handschuhe Henke in der
wähnten Dissertation (Seite 38), und ich stimme ihm, die da
Verhältnisse berücksichtigend, vollständig bei.

Um aber nicht mit Laboratoriumsexperimenten und theor
Erwägungen allein meine Anschauungen zu stützen, auch
Praktikus Mut zu machen, unseren Erfahrungen zu trauen,
zunächst kurz daran erinnern, daß auch schon in der vorantise
Zeit man mit einer sofortigen gründlichen Reinigung nach In
der Hand recht gute Erfolge aufzuweisen hatte.

Es ist bekannt, daß die Mehrzahl der Lehrer der Chirurgi
den Operationskursus an der Leiche morgens abhielt. Volk
v. Bergmann, Schönborn u. a. operierten an der Lei
6—8 Uhr und trugen durchaus kein Bedenken, nach vorausges
gründlicher Händedesinfektion alsbald nachher die verschi
Operationen vorzunehmen. Schönborn[2]) spricht sich hierüber b
aus: „Während meiner Tätigkeit in Königsberg hielt ich im Som
chirurgischen Operationskursus an Leichen morgens von 6-
Soweit wie möglich vermied ich es allerdings an solchen Ta
denen ich morgens Operationskursus gab, dann am Vormitt
Laparotomie zu machen; öfters war dies aber nicht zu umge
mußte es doch tun. Der Wundverlauf solcher Fälle wich i
von dem Verlauf derer ab, die ich im Winter, wo ich mit
nur ausnahmsweise in Berührung kam, ausführte."

Es wird wohl kein Leser, nachdem ich dies referiert ha
Schluß machen, auch jetzt sollte der chirurgische Lehrer bein
richt die Gummihandschuhe weglassen. Das wäre töricht.
in den Gummihandschuhen ein Mittel haben, um unsere Hä
Beschmutzung zu schützen, muß der Operateur an der Leich

1) Zentralblatt für Chirurgie 1880, S. 419.

2) Der neue Operations- und Hörsaal der chirurgischen Universitäts
Würzburg. Rede zur Eröffnung. Wiesbaden 1890.

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zen, es sei denn, daß für einzelne Operationen der Handschuh rlich wäre. Denn ich rede nicht der unnützen Selbstbe- utzung das Wort, sondern mache nur auf die Mittel auf- sam, eine nicht zu umgehende Selbstbeschmutzung für re und für den Arzt selbst ungefährlich zu machen.

ch ist es nicht ausgeschlossen, daß genaue Temperaturmessungen auch in den Schönbornschen Fällen Unterschiede ergeben haben n, die zuungunsten der nach Leichenbenutzung ausgeführten tionen sprechen würden.

rartige Einwürfe kann man meinen eigenen Erfahrungen nicht n, da bei uns, wie bekannt, ununterbrochen die exakteste, vom vorgenommene, von mir kontrollierte Temperaturmessung aus- t wurde, immer in der Absicht, diese Zahlen zu wissenschaftlich rtbaren Beweisen zu benutzen. In der mehr denn 25 Jahre nden klinischen Tätigkeit habe ich den Praktikanten gegenüber ls Abstinenz eingeführt, habe ihnen nur zur Bedingung gemacht, ie zu einer Geburt gerufen wurden, — was dem einzelnen vor- ekannt war —, sollten sie einen Rock anziehen, mit dem sie nicht n Krankensälen und bei Sektionen gewesen waren. Wenn aber einer interessanten Geburt schnell eine große Zahl Zuschauer n wurde, konnte auch diese Bestimmung nicht innegehalten n. Natürlich legte jeder, der den Gebärsaal betrat, vorher den ab und bekam einen großen weißleinenen Kittel, der den ganzen r bis weit unterhalb der Knie bedeckte, so daß auch beim Sitzen üßigen Hände nicht mit den Beinkleidern in Berührung kamen. ngegen war die Desinfektion der Herren, die innere Unter- ngen oder Eingriffe vornehmen sollten, eine äußerst genaue und vom Jahre 1895 an ausschließlich mittels der Heißwasser-Alko- thode. Handschuhe wurden nicht benutzt.

d nun unsere Resultate: Trotzdem seit dieser Zeit bei nahezu Geburten im Durchschnitt 7 innere Untersuchungen von Prakti- n und Hebammenschülerinnen ausgeführt wurden, sind an septi- Prozessen nur 4 Wöchnerinnen gestorben, die von einem oder eren Praktikanten untersucht waren:

au G. 1889, Nr. 288. Placenta praevia, zu Haus tamponiert, kam mit Fieber Anstalt. Entbindung durch Wendung und Extraktion; Plazentalösung. Starb Wochen infolge von Thrombose und Pneumonie.

au M. 1891, Nr. 182. Wegen Ileus in der chirurgischen Klinik in Behandlung, ntbindung uns zugeschickt. Spontane Geburt. Tod am 16. Tage infolge bophlebitis, Endometritis und beginnender Peritonitis.

au B. 1896, Nr. 40. Mit Fieber aus der Stadt uns zugeschickt. Deshalb Zange. Infiziert außerhalb durch die Hebamme. Schwere Infektion. Tod am e.

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Frau Sch. 1902, Nr. 325. Zwei Tage zu Hause gekreißt. Mit 39,0° Fi 128—160 Puls eingeliefert. Perforation des absterbenden Kindes. Septis tonitis. Laparotomie am 12. Tage. Tod am 15. Tage.

Nun, von diesen 4 Todesfällen, an denen überhaupt Prakt und die nicht innegehaltene Abstinenz die Schuld ganz oder z tragen könnten, dürfte nur Fall M. 1891, Nr. 182 bei der Schu in Anrechnung kommen, da die betreffende Patientin der chirur Klinik tatsächlich fieberfrei uns zugeführt wurde und bei der die Quelle der Infektion nicht festgestellt werden konnte.

Wenn also bei vielen Tausenden von Untersuchungen Geb durch Praktikanten von etwa 5000 Frauen nur eine als tödl schädigt in Frage kommen kann, so ist das ein Resultat, d kaum von einer andern Klinik erreicht sein wird, ein Resultat, zweideutig dafür spricht, wie man auch ohne Einhalten ei stinenzzeit, allein mit einer strengen Händedesinfektionsmeth günstigsten Resultate erzielt.

Während dies Resultat, soweit septische Todesfälle in kommen, mit dem jeder anderen Entbindungsanstalt, voraus daß regelmäßig von berufener Seite die Kontrolle durch A erfolgt, verglichen werden kann, ist dasselbe mit dem anderen messer, der Morbidität, nicht der Fall, da Differenzen von 2 graden, die für die Statistik ausschlaggebend sind, von der me weniger exakten Vornahme der Temperaturmessung abhängi Ich kann daher nur, um die Wirkung der Untersuchungen von kanten festzustellen, unsere durch Jahre hindurch gleichmäß genommenen Temperaturmessungen und Aufzeichnungen be Ich scheide die Semesterzeit von der Ferienzeit, in der keine kanten gerufen wurden, und zwar 10 Jahre vor Einführung de wasser-Alkohol-Desinfektion und ungefähr ebensolange nac führung der besagten Methode von 1898 bis 1907.

Semester	Ferien
10 Jahre vorher 1737 Geburten mit 61,4% fieberl.[1]) Wochenb.	1263 Geburten mit 62,0% fieberlosen Woc
10 Jahre nachher 2267 Geburten mit 72,5% fieberl. Wochenb.	1566 Geburten mit 67,5% fieberlosen Woch

Danach ist in den letzten 10 Jahren das Resultat währe Zeit, wo Praktikanten und Schülerinnen tätig waren, sogar er

1) Unter fieberlosen Wochenbetten sind solche zu verstehen, die wäh ganzen Zeit des Aufenthalts der Wöchnerin in der Anstalt nicht ein M 38,0° Temperatur aufwiesen. Auf Seite 529 habe ich die Strenge der bei den Temperaturmessungen betont.

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er als in der Zeit, wo nur Schülerinnen untersuchten. Eine ge-
nde Erklärung hierfür kann ich nicht geben. Ich käme nicht über
Vermutungen hinaus. Mir genügt, bewiesen zu haben, daß ohne
inenzeinhaltung das Resultat für die Wöchnerinnen kein schlech-
ist, als nach dem Gesamtdurchschnitt, wie er sich für die letzten
4000 Geburten ergibt und rund 70% fieberlose Wochenbetten
eist.

Diese hohe Zahl für Fiebernde wird denen nicht auffallen, denen
Verhandlungen über das Thema der Temperaturmessungen im
henbette, das ich 1893 anregte[1]), bekannt sind. Damals hatte ich
Genugtuung, daß mit wenigen Ausnahmen anerkannt wurde, wie
bei exakter, streng überwachter Messung zu ganz anderen Resul-
kommt, als wenn das Personal die Thermometer besorgt und
in der Absicht, auf Zehntel genau, zu wissenschaftlichen Zwecken,
Quecksilberhöhestand festzustellen.

Wenn z. B. in neuerer Zeit Bürger berichtet[2]), an der Schauta-
n Klinik sei nach Spontangeburten bei engem Becken ein Morbi-
sprozent von 4,1 beobachtet, und Fehling sagt[3]), „er habe in
ßburg gewöhnlich zwischen 6 und 7% Fieber von 38,1 ab (Axilla)
Fieber gerechnet", so muß dies Resultat unbedingt auf mangel-
r Thermometrie beruhen, vorausgesetzt, daß keine Wochenbetten
elassen sind, es sich um eine größere Reihe handelt und die
achtung bis zum Abgange der Wöchnerin, mindestens 9—10 Tage,
esetzt wurde.

Wie wichtig die genaue Bestimmung der Wochenbettstemperatur
rhellt sehr deutlich aus einem Artikel Winters über Meldepflicht
Hebammen bei Wochenbettfieber[4]). Winter schätzt die Tempera-
eigerungen in der Außenpraxis auf 5—10%! Welcher Irrtum!
um beachtet Winter nicht meinen Aufsatz[5]): „Kreisarzt und
bettfieber", der in derselben Zeitschrift sich befindet, in der er
ffentlicht hat? Dort habe ich festgestellt, daß in der Außenpraxis
30% Fieber bei vortrefflichen Hebammen, und vermutet, daß 40%
len gewöhnlichen Hebammen vorkommen.

uch die Tatsache, daß wir in der Marburger Entbindungsanstalt
septische Station hatten, sondern die gering fiebernden Wöch-
nnen mit den übrigen zusammenliegen ließen, die schwer kranken

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Zeitschr. f. Geb. und Gynäk. Bd. 27, Seite 470 und Bd. 32, Seite 22 und
37.
Die Geburtsleitung bei engem Becken. Wien 1908. S. 122.
Hegars Beiträge Bd. XII, S. 494.
Zeitschr. f. Mediz.-Beamte 1908, Nr. 3, S. 73. 5) ebd. 1906, Nr. 21.

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hingegen in ein Einzelzimmer brachten, daß wir keinen sep
Assistenten hatten, sondern der diensttuende Arzt bald eine g
Wöchnerin zu besuchen, dann auch wieder eine Entbindung
nehmen hatte, — und wir doch eine niedrigere Mortalität, als
Anstalten hatten, beweist, wie eine Abstinenz bei Vorsicht u
wissenhafter Händedesinfektion überflüssig ist.

Da wir in den staatlichen Anstalten die jungen Medizine
für die Krankenhäuser, sondern für die allgemeine Praxis er
so habe ich im Unterricht immer darauf gehalten, Bedingun
schaffen, die denen der Praxis nicht zu grell gegenüberstehen
sonders für die praktische Tätigkeit am Geburtsbett, die ja
nur zum allerkleinsten Teile in Anstalten ausgeübt wird, meis
den wenig günstigen Verhältnissen der Alltäglichkeit, erscheint
geradezu pädagogisch unrichtig, während der Unterrichtszeit
hygienischen Komfort und prophylaktische Maßregeln von eine
dehnung zu benutzen, die nie und nimmer später dem prak
Arzte zu Gebote stehen können.

Ich habe daher auch, wie in der allgemeinen Praxis, die
Leute bald zu einer kranken Wöchnerin, bald zu einer Gebä
geführt.

Neuerdings hat man dieses Verfahren als eine Gewissenlo
gestempelt und es als unerlaubt bezeichnet, daß ein Lehrer d
burtshilfe dem praktischen Arzte mit einem solchen Beispiel
angehe.

Da ich aber der Meinung huldige, der gewissenhafte Arzt
Hinsicht auf Händedesinfektion dasselbe leisten wie ich, wie
Assistenten, meine Hebammen und Schülerinnen, so halte ich
daß dieser mein Rat eher eine Sicherheit bietet, als das Eingest
der Unmöglichkeit der Händedesinfektion, das zweifelsohne im
tischen Leben eine ungenügende Ausführung der Händereinigu
Folge haben muß.

In der vorantiseptischen Zeit sind trotz ganz mangelhafte
nigung der Hände, selbst wenn sie schwer infiziert waren,
Operationen und viele Geburtsfälle ohne jegliche Schädigung
Patienten und Gebärenden verlaufen. Derartige Fälle dürfen w
keineswegs als Zeugnisse für meine oben vertretenen Anschau
anrufen, vielmehr darauf hinweisen, wie zu jener Zeit infolge
Unvollkommenheit viele Menschen geschädigt worden sind.

Wenn man zur Jetztzeit zur Unterstützung der von mir vertr
Ansicht beweisende Fälle bringen will, so muß man nachweise
man sich der Tragweite seiner Handlung voll bewußt war, al
Virulenz des infizierenden Stoffes kannte, die Desinfektion a

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ssenhafteste ausführte und die Operation oder geburtshilfliche ahme eine solche war, die, was Infektionsgefahr anbetrifft, zu gefährlichen gerechnet wird.

erartige Fälle habe ich mir aus meiner Tätigkeit der letzten genauer aufgezeichnet, wo ich selbst oder einer meiner Assi- en unmittelbar nach Beschmutzung unserer Hände mit hochviru- n Stoffen uns genötigt sahen, geburtshilflich einschreiten zu en und dies aus anzugebenden Gründen ohne den Gebrauch Handschuhe getan haben.

wei dieser Fälle habe ich schon früher veröffentlicht[1]) und re- e daher nur ganz kurz:

. Nach einer Streptokokkenverunreinigung bei tödlich endendem Puerperalfieber nahm ich die innere Untersuchung einer Gebärenden vor. Wochenbett glatt verlaufend.

Schwere Sepsis in partu. Kaiserschnitt. Fast fieberloses Wochenbett.

n diesen füge ich folgende neueren Beobachtungen hinzu und etwas genauer, damit der Leser sieht, wie auf jeden einzelnen and geachtet ist.

Vormittags zwischen 10 und 11 Uhr wurde ich veranlaßt, ein jauchendes Scheidenkarzinom zu untersuchen, ob eine Radikaloperation vorgenommen werden sollte oder nicht. Nach der Untersuchung nahm ich sofort die ausgiebigste Waschung und Alkoholdesinfektion vor.

Zwischen 11 und 12 Uhr konsultierte mich eine Dame, von der ich wußte, sie war in der zweiten Hälfte der Schwangerschaft, wegen heftiger Leibschmerzen. Ich nahm vor der inneren Untersuchung die ausgiebigste Händedesinfektion vor, bediente mich aber eines sterilen Gummifingerlings. Die Untersuchung ergab, daß die ahnungslose Dame sich in der Geburt befand, so daß es zweifelhaft blieb, ob ich sie nach Hause zurückreisen lassen konnte oder sie gleich in der Anstalt behalten mußte. Die Entscheidung dieser für die Frau und für mich so wichtigen Frage hing davon ab, ob das Fruchtwasser abgegangen war oder nicht, ob die Blase noch über dem tiefstehenden Kopfe erhalten war oder nicht. Mit dem besten Willen konnte ich das mit dem mit Gummistoff bekleideten Finger nicht fühlen, mußte daher den Fingerling abstreifen und untersuchte nun mit dem freien Finger. Die Blase stand noch. Doch zog die

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20. 367.

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Dame vor, dazubleiben und nach wenigen Stunden trat
burt ein. Wochenbett fieberlos. Höchste Temperatur 3

4. In den Weihnachtsferien 1906 war ich mit einem Assi
allein in der Anstalt. Auf der Station lag eine schwe
peralkranke Wöchnerin, die auch der Krankheit erlag.
der einzige Assistent freie Hand behielt für alle Gebu
übernahm ich persönlich die Behandlung dieser Wöchne
mußte sie am 6. Tage des Wochenbettes bei drohende
tonitis innerlich untersuchen, da ich einen Uterusriß befü
Diese Untersuchung geschah am Vormittag.

Nachmittag mußte eine ältere Erstgebärende mit
engen Genitalien, deren großes Kind in Steißlage sich
entbunden werden. Der Assistent drängte seine Hand mi
durch die Genitalien, um den Versuch zu machen, ein
herabzuholen. Es gelang ihm nicht. So war ich g
gleich selbst einzugreifen, was ich nach einer gründliche
infektion tat. Auch mir gelang es nicht. Ich ließ mir
den stumpfen Haken geben und mit großer Mühe glü
Anlegung und unter kräftiger Anstrengung auch die Ext
nachdem ein tiefer Schuchardtscher Schnitt die äußere
talien weit gespalten hatte.

Das Wochenbett verlief unter geringer Fieberstei
Höchste Temperatur 38,8°. Mutter und Kind verließen
dem die äußere Wunde geheilt war, gesund die Anstalt

5. Am 28. Januar 1907 brachte man uns einen Fall v
schleppter Querlage mit stinkendem Ausfluß und überaus
Gasentwicklung mit pestilenzialischem Gestank. Tem
vor Einlieferung 40,0. Dr. R., mein Assistent, nahm na
Dekapitation vor, die ziemliche Schwierigkeiten bot und
über eine Stunde Hantierens in den Genitalien erforde

Gegen Mittag mußte eine Zange bei einer Erstgebi
angelegt werden, und Dr. R., nachdem er sich sehr gr
desinfiziert hatte, entwickelte mittels mehrerer Traktio
kräftige Kind. Wochenbett absolut fieberlos. Höchst
peratur 37,8.

Man wird fragen, warum denn von der Benutzung der G
handschuhe abgesehen worden sei, da sie doch mindestens ni
schadet haben würden.

Für Fall 1 und 2 erübrigt sich diese Frage, da es zu jen
noch keine Gummihandschuhe gab. Für Fall 3 ist der Grund
Berichte angegeben. In Fall 4 war ich der festen Überzeugu
beim Eindrängen des stumpfen Hakens und beim Manipulie

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selben unzweifelhaft der Handschuh zerfetzt worden und da-
nicht nur überflüssig, sondern hindernd gewesen wäre, und das
he befürchtete ich im Falle 5, wo das Zangenblatt mit Mühe neben
eingekeilten Kopfe vorüber in die Höhe gedrängt werden mußte.
ber auch im allgemeinen sehe ich für meinen Teil gern vom Ge-
che des Handschuhs ab, weil mir das feine Gefühl in den Fingern
die freie Beweglichkeit derselben vermindert wird. Wiederholt
ich, mit dem Fingerling versehen, nicht fühlen können, ob die
e am vorliegenden Kopfe frei oder noch von den Eihäuten be-
t waren. Die Beschaffenheit der Schleimhaut, wie sie z. B. bei
nender Ausbreitung des Karzinoms gefühlt werden muß, kann
ohne Handschuh mit größerer Bestimmtheit feststellen. Bei der
nose beginnender Schwangerschaft ist für mich die Konsistenz
die Cervix füllenden Schleims ein nicht unwichtiges diagnostisches
l. Mit dem Handschuh bin ich nicht in der Lage, die feineren
istenzunterschiede zu erkennen.

h habe mich aber mit diesen subjektiven Empfindungen in der
tischen Tätigkeit nicht zufrieden gegeben, sondern habe mit Hilfe
nstrumente zur Prüfung des Tastsinnes, wie solche dem hiesigen
iologischen Institute zu Gebote stehen, z. T. unter Mitarbeit des
tdozenten Dr. Lohmann, experimentell den Verlust am Gefühl der
feinen Gummihandschuhen versehenen Hand gegenüber der un-
kten Hand festgestellt. Selbstverständlich war nach allen Rich-
n hin eine Verminderung der Wahrnehmungsfähigkeit vorhanden,
geringsten bei der Unterscheidung rauher und weniger rauher
ebe oder Gegenstände, stärker bei der Feststellung näher oder
rnter liegender Tastempfindungen, noch stärker bei Bestimmungen
Konsistenzgraden flüssiger und halbweicher Massen.

on vielen Chirurgen und Gynäkologen wird berichtet, daß sie
alltäglichen Gebrauche der Handschuhe eine gleiche Feinfühlig-
erlangt haben wie zur Zeit, wo sie ohne Handschuhe operierten.
vill es gern glauben. Doch fehlt der Gegenbeweis, der nur zu
n wäre, wenn sie nun wieder längere Zeit ohne Handschuhe
eren würden. Dann erst würden sie sagen können, ob nicht
der Handschuh einen Nachteil abgebe.

ür die zahllosen Ärzte aber, die nicht durch fortdauernde Be-
ng des Handschuhs sich diese Fertigkeit erwerben, bildet sein
auch eine Herabsetzung der Fähigkeiten der Hand.

urz und gut, ich für meine Person begebe mich bei Be-
ung des Handschuhs wesentlicher Vorteile, die ich nur
en dürfte, wenn ohne Gebrauch des Handschuhs sicher
Gefahr für die zu Untersuchende oder zu Operierende

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entstünde. Das ist aber bei meiner Hand nicht der Fall; v
auch glaube, daß die meisten Ärzte diese Gefahr auf Null verm
können.

Bedenkt man nun noch die große Gefahr, die bei nicht ge
gereinigter Hand durch Verletzung der Handschuhe entstehen
so soll man doch ernstlich erwägen, ob die obligatorische Ben
des Handschuhs berechtigt ist.

Ich habe seinerzeit mich einmal so ausgesprochen[1]): „Der G
handschuh ist eine vorübergehende Modesache." Dies Wort
vorgehalten, und ich gebe zu, daß ich damit meine Ansicht nicht
wiedergegeben habe, denn nach einer Seite hin, nämlich zum S
der eigenen Hand und in Fällen, wo eine Händedesinfektion
schlossen ist, besitzt der Gummihandschuh unzweifelhaft seine
Ich habe in jener Publikation dies Urteil dahin begründet: „Die
schuhe werden eine Zeit hindurch von Spezialisten Verwendung
in die allgemeine Praxis werden sie sich nicht einbürgern. Wir
der Geburtshilfe sollen aber unsere Vorschläge, unsere Me
soweit es geht, den Verhältnissen der allgemeinen Praxis anp

Mit dem Ausdrucke „vorübergehende Modesache" wollte ich
Ebenso wie vor Jahren auf die Empfehlung Listers alle Wel
Spray operierte, bis sich ergab, daß dieses Verfahren über
sogar schädlich sei, so gebraucht auch jetzt alle Welt Gumm
schuhe, bis sich herausstellt, daß sie bei vielen Operationen
flüssig sind, insofern sogar schädlich werden können, indem
Gefühl beeinträchtigen, das Fassen von schlüpfrigen Organtei
schweren, ihre Vorbereitungen zum Anziehen die Narkose verl
vor allem aber den Glauben an die Notwendigkeit einer exakten
desinfektion nehmen und damit zu einem Sichgehenlassen
Händepflege und Händereinigung führen, was wieder die weite
Folge haben kann, daß bei Verletzungen der Handschuhe w
der Operation Infektionen von der Handoberfläche aus erfolge

Als Friedrich im Jahre 1898 die dünnen nahtlosen Gumm
schuhe empfahl[2]), fügte er warnend hinzu: „Will man den Ge
der Handschuhe verallgemeinern, wozu mir eine Berechtigun
vorzuliegen scheint, so ist zu bemerken, daß er der Raschh
Operierens auf alle Fälle schadet, den aseptischen Apparat
kompliziert, die allgemein menschliche Seite des eventuelle
trauens auf seinen Schutz die Strenge der sonstigen Händes
tionsmaßnahmen gefährdet."

1) *21.* 847.
2) Zentralblatt für Chirurgie 1898, S. 449.

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)er letztere Punkt, den ich eben und auch früher schon öfter orgehoben habe, scheint mir von größter Bedeutung. Frage sich jeder Arzt, der einen Handschuh gebrauchen will, vor jedem rauche offen, weshalb nimmst du den Handschuh? Ich bin über-, in der Mehrzahl der Fälle muß er sich sagen: Aus Bequem-eit, weil ich mich vorher und nachher nicht zu desinfizieren che.

er hohe Preis der Handschuhe würde nicht in Frage kommen, n wir in den Handschuhen ein absolut sicheres Mittel gegen Tod durch Sepsis und gegen Wundeiterung. „Wir müßten lieber ern, als das Geld auf Kosten der Gesundheit und des Lebens rer Patientinnen sparen!" Diesem Ausspruche Fritschs[1]) fehlt der Nachsatz: Da die Gummihandschuhe aber kein absolut res Mittel gegen Wundfieber und Tod sind, — und ich setze , da wir sie durch eine gründliche Händedesinfektion ersetzen en —, so kommt auch der hohe Preis in Frage und Fritsch eshalb nicht berechtigt, dies Argument gegen den allgemeinen auch „verwerflich" zu nennen.

diesem Sinne spricht sich auch Bezirksarzt Dr. Walther[2]) aus: rden sich die Gummihandschuhe in der Hand der Hebamme hren, so dürfte selbstverständlich der Kostenpunkt, der bei der en Abnutzung von Bedeutung ist, kein Hindernis bilden. Sind Handschuhe aber unnötig oder gar in der Hebammenpraxis schäd- wozu solche unnütze Ausgaben?"

ür die geburtshilfliche Praxis ist es erwiesen, daß der Gebrauch Handschuhe keine Vorteile bringt. Es gibt keine Statistik, die zeugend nachweisen kann, daß bei dem Gebrauche der Hand- he eine nennenswerte Besserung erfolgte gegenüber den Anstalten, trenge Händedesinfektion gefordert und geleistet wird.

Vie es sich in der Praxis der operativen Gynäkologie verhält, ich nicht beurteilen. Nur erscheint mir die Statistik, die Küst-) aufgestellt hat, um das Übergewicht der Handschuh-Operationen eweisen, etwas sehr gedrechselt. Auch läßt Küstner unberück-gt, daß die Besserung proportional den Jahren erfolgt, also sowohl die zunehmende technische Fertigkeit des Operateurs, ch auch der Assistenten, als auch die strengere Auswahl der eine wesentliche Rolle in der Besserung der Resultate mitspielt. bitte in dieser Beziehung auch die Einwürfe Schickeles und

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) Verhandl. der Deutsch. Ges. für Gyn. 1907, XII, S. 482.
) Münchener med. Wochenschrift 1907, Nr. 6, S. 267.
) Verh. d. Deutsch. Ges. für Gyn. 1907, XII, S. 571 f.

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Tauffers gegenüber der Küstnerschen Statistik zu beachten (Ve
der Deutschen Gesellsch. für Gynäkologie 1907, XII, Seite 682
Auch mache ich auf die neueste Publikation von Chrobak[1]) a
sam, dessen Ansicht der meinigen sehr nahe steht.

Ich bin fest überzeugt, würde Küstner, ein Mann, der ei
gezeichnete Technik besitzt und eine gewissenhafte Händedesi
schätzt, ohne Handschuhe operieren, er würde die gleich guten
tate haben und einsehen, wie viel einfacher und angenehm
Operieren mit unbedeckten Händen ginge.

Es sollten sich doch einmal die Chirurgen und Gynäkologe
lassen, die ohne Handschuhe operieren. Von Tavel[2]) las i
er die Doederleinschen Versuche für durchaus nicht maß
halte, ohne Handschuhe operiere und diese nur zum Schut
Infektion gebrauche. Auch Vogel[3]) teilt aus der Bierschen
mit: „Bezüglich der Desinfektion der Hände des Operateu
stets das Hauptgewicht auf die Prophylaxe zu legen sein. Hi
der Hauptwert der Handschuhe, die von uns bei septischen
tionen benutzt werden, während wir uns für aseptische noc
mit ihnen befreunden konnten. Die dicken, vor dem Ze
sicheren, stören das Gefühl zu sehr, die dünnen sind zu leic
letzt und dann gefährlicher als keine Handschuhe."

Auf diese Gefahren, die durch Handschuhverletzungen en
machen Füth[4]) und Gottstein[5]) besonders aufmerksam.
eigenen Erfahrungen habe ich bereits früher veröffentlicht.[6])

Im Anschluß an die Versuche über sofortige Reinigung de
nach eben stattgefundener infektiöser Beschmutzung und info
Betrachtung über den Wert der Abstinenz schien es mir ni
zweckmäßig zu sein, den Vorgang der Selbstreinigung de
etwas genauer zu studieren, zumal ich, wie oben schon aus
nur Ansichten ausgesprochen fand, die auf Vermutungen b
sich aber nicht auf Versuche gründen.

Da ich nach meinem Austritte aus der Frauenklinik und nach
meiner Praxis meine Hand mit keinerlei Krankheitsstoff in Be
zu bringen und deshalb auch keine chemische Händedesinfekti
hatte, so benutzte ich einige Wochen zu diesen Versuchen.

Zu dem Zwecke gebrauchte ich den Prodigiosus und den

1) Zentr. f. Gyn. 1908, Nr. 14, S. 458.
2) Korrespondenzbl. f. Schweizer Ärzte 1907, Nr. 22, S. 711.
3) Deutsche med. Wochenschrift 1905, Nr. 30, S. 1179.
4) Zentralbl. für Gynäkologie 1902, Nr. 39.
5) v. Bruns, Beitr. zur Klin. Chirurgie, Bd. 25, S. 97.
6) *29.* 1957.

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citreus. Aus dem hygienischen Institute erhielt ich von beiden
ien frische Kulturen, die sich, was ich gleich vorausschicken
urch Kontrollversuche als sehr lebensfähig erwiesen.
'angs in schwächeren, später in stärkeren Aufschwemmungen
ich meine eine Hand und ließ die Masse antrocknen. Die
wurde einen halben Tag lang, einmal 19 Stunden lang, nicht
asser in Berührung gebracht, und wenn eine Waschung dann
ihrt wurde, so geschah dies ohne Bürste, nur mit Seifenschaum
asser, so kurz als möglich.
sehe von der Aufführung der einzelnen Versuche ab, sondern
nur die Resultate:
Prodigiosus, mochte die Hand schwächer oder stärker be-
zt sein, war nach zweimal 24 Stunden und später nicht mehr
weisen. Nur bei längeren Nägeln blieb er unter dem Nagel
inige Zeit erhalten.
gelbe Luftpilz, Diplococcus citreus, mäßig reichlich die ganze
beschmutzend, war 7 Tage lang, wenn auch nur in ganz ein-
Kolonien, an der Hand nachweisbar.
ieweit man die Resultate mit derartigen Bakterien, die nicht
Hautparasiten gehören, auf die mit pyogenen Bakterien infi-
Hand übertragen kann, ist freilich eine andere Frage. Gern
ch auch mit diesen Versuche gemacht, doch weiß ich mit dem
Willen keinen Weg zu finden, um den Nachweis zu führen,
e zum Zwecke der Untersuchung auf die Haut gebrachte Spezies
o und so viel Zeit als Reste oder Abkömmlinge der Infektions-
anzusehen seien.
gegen sei hier noch eines Versuchs Erwähnung getan, den ich
e, um zu sehen, ob die Hand durch stundenlange Erweichung
r warmem Wasser ihren Keimgehalt auffallend ändere, wenn
ßen keine neuen Keime hinzukommen können.
ne saubere Tageshand, die wochenlang nichts mit „infektiösen"
zu tun gehabt hatte, auch nicht desinfiziert war, steckte ich in
riles Waschbecken mit $1^1/_2$ l sterilem Wasser und ließ Becken,
und Vorderarm mit einem sterilen Tuche bedecken. Eine halbe
lang bewegte ich zum öfteren Hand und Finger, nahm dann
nd hervor, rieb sie mit einem sterilen Handtuche ab und
e sie sofort in ein zweites und nach wieder halbstündiger Han-
; in ein drittes, viertes, fünftes Becken, jedesmal vor dem
el die Hand abreibend.
s jedem der fünf Becken wurden 10 ccm Wasser mit steriler
e entnommen und in fünf sterile Reagenzgläser gebracht.
n Schlusse dieses $2^1/_2$stündigen Verfahrens kratzte ich mit

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sterilen Hölzchen die Haut der Hand an verschiedenen Stel
kräftig ab und warf die Hölzchen in ein sechstes steriles Rea

Unmittelbar nach Beendigung des Versuchs wurde aus
Reagenzgläsern je 1 ccm Flüssigkeit entnommen, in eine Pe
geschüttet und mit flüssigem Agar übergossen. Auch die
wurden in einer Petrischale in gleicher Weise behandelt.

Nach 4 Tagen hatten sich in der Schale 3, 4 und 5 ungef
pelt soviel Keime entwickelt, als in 1 und 2, während in de
mit den Hölzchen eine mittlere Anzahl zu finden war.

Jedenfalls war ersichtlich, daß mit Aufweichung und Er
der Hand die Zahl der Keime sich vermehrt hatte.

Da ich infolge Aufgabe meiner klinischen Tätigkeit voraus
nicht wieder dazu kommen werde, durch experimentelle
Untersuchungen und Beobachtungen weitere Beweise für d
des Alkohols als Desinfektionsmittel der Haut, vornehm
Hand, zu bringen, so sei es mir gestattet, den Stand der F
zum Schluß des Jahres 1907 zu fixieren.

Dies erscheint mir auch nötig, um der großen Zahl v
kollegen, die sich bei den widersprechenden Publikationen
wissen, was und wem sie nur eigentlich glauben sollen, ein
weiser zu verschaffen.

Hängt doch von der Entscheidung der Frage, ob man, b
auch der praktische Arzt imstande ist, wenn er nur gewissen
fährt, sich derart vorzubereiten, daß er einen chirurgischen,
burtshilflichen Eingriff vornehmen darf, ohne die ausgedehn
phylaktischen Hilfsmittel benutzen zu müssen, die jetzt in den
Operations- und Entbindungssälen eingeführt sind, besonde
ohne sich stets der Gummihandschuhe bedienen zu müssen.

Das Für und Wider den Alkohol ist gerade in de
Jahren in recht auffälliger Weise zutage getreten. Ein Sam
wie das v. Winckelsche, an dem zahlreiche Lehrer der Geb
mitgearbeitet haben, muß ja von vornherein mit Dissonanze
Anschauungen rechnen. Immerhin dürften diese doch für d
in etwas gemildert sein, damit er sich selbst ein Urteil na
oder jener Seite hin schaffen könne. Wenn aber der Inhal
schroff gegenübersteht, wie in dem von Menge bearbeiteten
über Antiseptik und Aseptik, Band I, 2. Teil und in dem
geschriebenen über Händereinigung, Band III, 2. Teil, dann
Vermittlung nicht möglich. Nur der eine kann recht hab

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re lehrt etwas Falsches. Auf dem letzten Gynäkologenkongresse, den 1907, waren beide Referenten, Fritsch und Küstner, Gegner Alkoholdesinfektion und damit auch Anhänger der Gummihand- he. Beide stützten sich auf das von anderen ausgesprochene m von der Unmöglichkeit, die Hand auf die Dauer einer Operation frei zu machen, obwohl ich durch verschiedenartige Experimente gewiesen habe, daß dieses nicht nur mir, sondern vielen meiner tenten und Schülerinnen fast regelmäßig gelungen ist. Wie Fritsch, auch Küstner an die Spitze seines zusammenfassenden Referats bisher noch mit Recht zu bestreitenden Satz: „Es ist erwiesen, daß laut des lebenden Menschen durch kein Mittel und keine Prozedur frei zu machen ist.“ Auch Doederlein führt sich in München er mit der Behauptung ein, alle Versuche, die Hand des Opera- keimfrei zu machen, hätten die Unmöglichkeit ergeben.[1])

elbstverständlich muß sich auf solche Anerkennung der Unvoll- nenheit jeder Desinfektionsmethode ein Gebäude von Schutzmaß- n aufbauen, das jetzt eine Höhe erreicht hat, daß der praktische nicht mehr folgen kann.

uf Paraffin- und Wachsüberzug der Hand folgten Handschuhe von und Leder. Dann Zwirnhandschuhe, Gummihandschuhe. Gummi- schuhe plus Zwirnhandschuhe und Zwirnhandschuhe plus Gummi- schuhe, Firnisüberzug der Hand und Betupfen des ersten Finger- s mit Jodtinktur. Die unbedeckte Hand, als chirurgisches Werkzeug, e geradezu verpönt und das Nonplusultra dieser Händefurcht der oben schon erwähnte Ausspruch Menges, der Arzt dürfe mit r Hand kein Bad für eine Gebärende zurechtmachen.

Vie soll sich nun der Arzt, der selbst keine experimentellen Unter- ungen gemacht hat, Klarheit verschaffen, wenn er die wider- hendsten Urteile hört. Auf der einen Seite: „Der Stern des hols, der so hellstrahlend aufgegangen war, ist wieder im Ver- hen“[2]), und in demselben Handbuche: „Die Aufgabe des Alkohols utet einen Rückschritt in der Desinfektionsfrage“[3]), oder, wie der- Autor sich an anderer Stelle[4]) ausdrückt: „Ohne Alkohol keine ebige Desinfektion.“

Venn einerseits der Referent[5]) einer Arbeit Schumburgs[6]), die ausgezeichneten Erfolgen bei der Alkoholdesinfektion berichtet, zu dem Ausspruch veranlaßt sieht: „Dagegen ergibt sich der Sieg

) Deutsche med. Wochenschrift 1907, Nr. 52, S. 2199.
) Menge, v. Winckels Handbuch, Bd. I, 2. Teil, S. 1215.
) v. Herff, in v. Winckels Handbuch, Bd. III, 3. Teil, S. 781. — 4) Münchener Wochenschr. 1907, Nr. 21. — 5) Zentr. f. Gynäk. 1907, Nr. 30, S. 949. — chiv für Klin. Chir. Bd. 79, Heft 1.

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des Alkohols auf der ganzen Linie des Kampfes gegen die
bakterien", und andererseits Littauer[1]) den Handschuh als
Rettungsmittel empfiehlt, „den Ahlfeld, verblendet vo
Werte seiner Alkoholdesinfektion, eine vorübergehende Mo
nennt".

Ich habe früher Gelegenheit genommen[2]), nachzuweisen, w
die Zahl der Experimentatoren ist, soweit sie nach meiner D
tionsmethode geprüft haben, auf deren Resultate sich das Ax
Unzulänglichkeit der Heißwasser-Alkohol-Desinfektionsmethode
Füge ich noch hinzu, daß von den wenigen, die ausgedehnter
suchungen gemacht haben, die Resultate zum Teil derart war
bei Fortsetzung der Versuche das Ziel der absoluten Keimfrei
Hand wahrscheinlich erreicht worden wäre[3]), Engels[4]), ein
der Methode, sogar, angesichts der Vorzüglichkeit seiner R
zu dem Ausspruch veranlaßt wird: „Das Resultat kann in
Recht den Glauben erwecken, daß nochmal eine Zeit komme
wo wir mit Hilfe bestimmt zusammengesetzter Desinfektionsg
doch eine vollständige Keimfreiheit der Hand erzeugen könn
sinkt die Zahl der gegnerischen Veröffentlichungen in ihrer Be
wesentlich herab.

Überdies sind zu den von mir in einer unvergleichlich g
Zahl ausgeführten Untersuchungen immer neue hinzugekomm
auf noch strengeren Untersuchungsmethoden aufgebaut, zu de
Ergebnissen gelangen, wie die früheren.

Wenn sich trotz des Widerstandes und einer für wissensch
Fragen ungewöhnlichen Agitation gegen die Benutzung des A
als Desinfektionsmittel von seiten einer einflußreichen Parte
den Gynäkologen dennoch von Jahr zu Jahr die Berichte über
reiche bakteriologische Untersuchungen und über ausgezeichnete
tate bei der praktischen Anwendung mehren, so ist dies w
Beweis für die Richtigkeit meiner Vorschläge, wie er selten i
strittigen Frage erbracht wird.

Wenn Fritsch (Seite 187) sagt: „Gerade der Umstand, d
mand erreichte, allgemein gültige Gesetze aufzustellen, sond
überall das gleiche Ziel von getrennt marschierenden Forschern
wird, sichert auch einen ferneren Fortschritt und verhindert
starrung in selbstgefälliger Zufriedenheit", so möchte ich de
gegenhalten, daß man gerade jetzt durch Empfehlung des u

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1) Zentr. f. Gyn. 1907, S. 883. — 2) *20.* 372. — 3) Paul u. Sarwey
med. Wochenschr. 1899, Nr. 51, Tab. Nr. 3, Dr. Meyer. — 4) Klinisches
1905, Bd. 13, S. 606 f.

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kten Gebrauchs der Gummihandschuhe auf dem besten Wege „dieser Erstarrung in selbstgefälliger Zufriedenheit" zu gelangen möchte weiter zu bedenken geben, wie eine solche Sentenz, in lgemeine Praxis übertragen, in das Gegenteil umschlagen würde. raktische Arzt und vor allem die Hebamme und das Warteper- sie bedürfen einer festen Richtschnur in der Frage der Hände- fektion. Es ist daher wünschenswert, im allgemeinen einer edesinfektionsmethode den Vorzug zu geben. Dabei ist es jedem, as Gebiet vollständig überschaut, unbenommen, an deren Ver- rung weiter zu arbeiten oder eine andere, seiner Meinung nach re Methode zu gebrauchen und zu empfehlen, wie auch Ab- ungen von der anerkannten Methode sich von selbst ergeben, ausnahmsweise diese Methode nicht vertragen wird.

Laufe der letzten Jahre haben sich durch eigene und anderer suchungen eine Reihe der strittigen Punkte wesentlich geklärt, rch die Beurteilung der von mir vertretenen Desinfektionsmethode dem Fernerstehenden erleichtert wird.

nehme die Haupteinwürfe nochmals kurz durch, indem ich r Hauptsache nur neuere Beweismittel für und wider anführe, tere, schon zur Genüge besprochene, nur verweise.

r zeitlich erste, von Krönig gemachte und bis auf den heutigen estgehaltene und in den Vordergrund gestellte Einwurf ist der cheindesinfektion.

t dem Worte „Scheindesinfektion" hat Krönig, wie die Öster- r sich ausdrücken, einen großen „Schlager" getan. Für die ählten Autoren, die selbst keine Versuche über Händedesinfektion ht haben, — und da sind unsere bedeutendsten Chirurgen und kologen nicht ausgeschlossen — genügt dieser Mann und dieses um immer und immer wieder den Satz zu wiederholen, der ol härtet nur die oberste Schicht der Haut; aus diesem Grunde ie Hand zeitweilig keine Keime von ihrer Oberfläche ab. Wird berfläche aber wieder weich, wie nach einem längeren Wasser- während einer Operation mit oder ohne Gummihandschuhe, kommen die verheerenden Keime aus der Tiefe hervor und er- n sich über das Operationsobjekt. Der Operateur ist bitter ge- nt worden. Scheindesinfektion.

der Tat würde der Vorgang sich derart abspielen, wenn man Hand, gänzlich unvorbereitet, mit einem konzentrierten Alkohol ndeln wollte. Der würde die oberste Schicht härten, in die Tiefe eindringen und die Bakterien der tieferen Schichten gänzlich ührt und lebensfähig belassen.

nders aber bei unserer Desinfektionsmethode, bei der eine Be-

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seitigung des oberflächlichen Hautfetts und ein Aufweichen der mit heißem Wasser vorausgehen muß, ehe der Alkohol zur Verwe kommt.

Die Scheindesinfektion könnte aber auch dadurch hervorg werden, daß die durch Alkohol erhärtete Oberfläche der Hand b experimentellen Prüfung die Keime nicht abgeben kann, wodurc Abtötung vorgetäuscht würde, ein Fehler des Experiments, der ausgiebige Aufweichung der Oberhaut nach Ausführung der Al waschung vermieden werden kann.

Obwohl ich in keiner Veröffentlichung unserer Desinfektionse mente zu betonen unterlassen habe, wie die Händeprüfung n ohne vorausgegangenes längeres Aufweichen der Hände erfolgt sind doch meine Resultate von den Gegnern, zumeist auf jene Kr sche Behauptung sich stützend, abgetan worden; auch noch, immer überzeugendere Experimente brachte: die Hand erst nac weichung in warmer Fleischbrühe durch eine Stunde lang unter oder nach über eine Stunde dauernden Operationen unter dem G handschuh. Alles half nichts. Es bleibt doch dabei, es war nu Scheindesinfektion.

Auch Fritsch, der früher einmal[1]) die Tiefenwirkung ane hatte, als er schrieb: „Ahlfeld hat Keimfreiheit der Finger Daß er dies erreicht hat, hat er bewiesen", stellt sich neuerdin schieden auf die gegnerische Partei. „Die Idee, durch Alkoh Haut trocken und hart zu machen und dadurch die Abgabe Kokken der Hand zu verhindern, ist doch nicht richtig! Nicht fe wollen wir die Kokken, sondern sie wegschaffen." Von Anschauung der Alkoholwirkung ausgehend, empfiehlt Fritsch, auflockernde Sublimatwaschung dem Alkohol nachfolgen zu l „ein crimen laesae majestatis gegen die Alkoholschwärmer".

Es ist wohl das erste Mal, daß man der kurzen auf den A gebrauch folgenden Sublimatwaschung die Bedeutung einer aufw den Waschung gegeben hat. In dieser Beziehung ist sie wohl g wertlos.

Das Wort „Alkoholschwärmer" kennzeichnet die Situatio erklärt mancherlei. Einen Schwärmer behandelt man natürlich als einen ernsthaften Forscher. Man belächelt ihn, man bem ihn, hat aber nicht nötig, seinen Mitteilungen weitere Aufmerks zu schenken. Er ist eben nicht zu bekehren. Habeat sibi.

„Schwärmer" darf man aber, meine ich, einen Kollegen

1) Lehrbuch der Geburtshilfe. 1900. S. 123.

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n, der unermüdlich einer für die Praxis enorm wichtigen Lehre
Vort redet, sobald sie sich auf kontrollierbare Tatsachen stützt.
ch die Folgerungen, die Fritsch aus der Annahme einer Schein-
ektion macht, fallen fort, wenn die Tiefenwirkung des Alkohols
sen ist. Er schreibt: „Gelänge es aber auch, die Hände für den
nt des Beginns einer Operation keimfrei zu machen, so wissen
och aus unendlich vielen Untersuchungen, daß am Ende einer
tion die Verhältnisse ganz anders liegen als beim Beginn, daß
ände nicht keimfrei bleiben, obwohl sie es vielleicht einmal
."

s ist zweifellos richtig. Nur ist damit nicht gesagt und bewiesen,
Laufe einer Operation auf der Oberfläche der Hand nachweis-
Bakterien seien Handbakterien, die infolge einer bloßen Schein-
ektion im Laufe der Operation an die Oberfläche gekommen
. Es kann sich ebensowohl um Neuinfektion und dann Ober-
infektion handeln, die gleicherweise die behandschuhte Hand,
e vorher absolut keimfreie unbedeckte Hand betreffen kann.
d, um das hier gleich anzufügen, läßt sich diese oberflächliche,
ufe der Operation auftretende Beschmutzung, wie im früheren
nitt bewiesen ist, mit Leichtigkeit durch eine schnell ausgeführte
ung und Abreiben der feuchten Hand mit Alkoholflanell mit
heit beseitigen; eine Hantierung, die schneller auszuführen und
r ist, als das Ausziehen der benutzten und Anziehen von frischen
ihandschuhen, und überdies den Vorteil hat, daß dabei die
berfläche sicherer keimfrei wird, als wenn unter dem ausge-
n Handschuhe sich Keimmassen angesammelt haben, die beim
hen an die Außenwelt treten.

t der Zeit habe ich aber auch in dieser Frage einige Mitstreiter
men, einen oder den anderen freilich wider seinen Willen.
Schäffer[1]), der dem Alkohol früher nur eine mechanische
ng zusprach. Im Gegensatz zu Haegler, Krönig, Paul und
ey, die sämtlich nach der Aufweichung der Hand im Gefolge
vorausgegangenen Desinfektion eine auffallende Vermehrung der
feststellten, berichtet er über 15 Versuche, „in denen ich 15 bis
nuten die Hand nach der Desinfektion in sterilen Flüssigkeiten
ichte (zum Teil im sterilen Kasten) oder 1—2½ Stunden die
mit Gummihandschuhen überzog. In keinem einzigen Falle
sich durch die Aufweichung der Hände eine irgendwie nennens-
Verschlechterung der Resultate ein".

Experimentelle und kritische Beiträge zur Händedesinfektionsfrage. Berlin
. 72.

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Weiter erwähne ich Vogel[1]), der die Kontrolle der desin
Hand im Schwitzkasten vornahm. „Dieses Resultat spricht ge
Annahme von Krönig u. a., daß der Alkohol nur eine Schei
fektion bedinge, indem er die Haut gerbe, dadurch die Poren ver
und die Bakterien in der Tiefe abschließe." „Der Umstand,
Schweiß nur wenig infiziert ist, spricht durchaus besonders
von Ahlfeld vertretene Ansicht, daß dem Alkohol eine nenne
bakterizide Kraft und auch Tiefenwirkung zukommt. Eine ‚G
der Haut mußte doch wohl auch die Sensibilität der Haut abst
Ich betonte aber vorn schon, daß die mit Alkohol gewaschen
gegen die Hitze viel empfindlicher ist."

Reverdin und Massol[2]) kommen ebenfalls zu dem Resul
aus den Schweißdrüsen nur wenige Keime geliefert werden.
Infektion durch den Schweiß der Hände gibt es nicht. W
Untersuchung nach dem Schwitzen eine bemerkenswert größe
von Keimen, als vorher festgestellt war, ergibt, so ist die W
der Hand eine ungenügende gewesen. Reichliche Schweißabso
bedingt keine Gefahr für die Operation."

Die keimtötende Fähigkeit des Alkohols gegenüber d
tativen Formen der Bakterien, also gegenüber der Sepsis, der
bettfieber, dem Wundfieber usw., die anfangs ebenfalls bestritte
hat jetzt von allen Seiten Bestätigung erhalten und zwar auch
zelnen genau entsprechend den Resultaten von Untersuchung
ich zuerst mit meinem Assistenten, Dr. Vahle[3]), zusammen a
und veröffentlicht habe.

Besonders muß ich erwähnen, wie Schäffer seine frühere
zu dieser Frage verändert hat. Während er in seiner größere
aus dem Jahre 1902[4]) dem Alkohol gegenüber den an der H
findlichen Bakterien keine bakterizide Eigenschaft zuerkennt,
den Erfolg als einen rein mechanischen erklärt, berichtet er
„Viele Hunderte von Einzelversuchen aber, die ich seitdem a
haben mich gelehrt, daß der Alkohol allen bekannten Antiseptiz
dem Sublimat in 1 pro mille Lösung — wenigstens feuch
jekten und vegetativen Bakterienformen gegenüber — zum m
gleichkommt."

Ich halte es für wichtig, zum Verständnis von mancher
gängen etwas eingehender auf diese Anschauungsänderung ein

1) Deutsche med. Wochenschrift 1905, Nr. 30, S. 1179.
2) Revue médicale de la Suisse Romande 1905, no. 1, p. 35.
3) 4. (1896). — 4) Experimentelle und kritische Beiträge zur Händedesi
frage. Berlin 1902. S. 66. — 5) Monatsschrift für Geb. u. Gyn. Bd. 19, S

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ffer war durch folgenden Versuch[1]) zu der festen Überzeugung
nmen, der Alkohol besitze keine keimtötende Eigenschaft:

infizierte die Hände mit dem gelben Luftpilz, bürstete in einer Schale mit
Alkohol 6 Minuten lang die Hände ab und goß den Alkohol mit den darin
nmenden Gerinnseln durch ein steriles Filter. Nachdem der Alkohol durch-
en war, wurde mit warmem sterilem Wasser der Filter und sein Inhalt von
l befreit und schließlich die Filterspitze herausgeschnitten und mit steriler
n in Petrischale gebracht.
ch 4 Tagen waren 2—3000 gelbe Kolonien angegangen.

häffer, der diesem Versuche „ganz besondere Beweiskraft"
reibt, notiert als Endresultat: „Aus diesem einfachen Versuch
nit zwingender Notwendigkeit hervor, daß die keimtötende Kraft
lkohols nun und nimmer zur Erklärung seiner sterilisierenden
ng ausreicht."

habe diesen Schäfferschen Versuch wiederholentlich nach-
ht und kann die Richtigkeit des Resultats nur bestätigen, nicht
den verallgemeinernden Schluß Schäffers, der Versuch be-
, daß der Alkohol nicht bakterizid wirke, denn die folgenden
che beweisen das Gegenteil und erklären den Mißerfolg.

benutzte die gleiche Kultur des Diplococcus citreus, vermischte sie mit
Alcohol absol. und schüttelte das Reagenzglas 10 Minuten. Diese Emulsion
in einen sterilen Filter gegossen, um den Bodensatz untersuchen zu können.
em der Alkohol durch steriles Wasser aus dem Filter ausgelaugt war (ca. 6 Min.),
die Filterspitze mit steriler Schere abgeschnitten, in eine Petrischale ge-
mit etwas Bouillon beschüttet und Agar darübergegossen.

sultat: Die Unmasse von Keimen, die in dem Reagenzgläschen
orfanden, wurden durch Alkohol in 10 Minuten bis auf 3 oder
etötet. Beobachtungszeit 9 Tage.

m Schäfferschen Versuche entspricht genau das Sängersche
iment[2]), über das ich früher[3]) schon berichtet habe. Die mit Sta-
coccus aureus vollgesaugten Streichholzstückchen wurden feucht
soluten Alkohol gebracht und dennoch zeigten sämtliche Gläschen
vachstum. Auch Sänger machte denselben Schluß wie Schäffer:
ar also dadurch bewiesen, daß Alcohol absolutus auf Staphylo-
s keinen nennenswerten desinfektorischen Einfluß besitzt." Auch
Versuche erwiesen sich, von mir nachgeprüft, als vollkommen
, aber ebenso stellte ich fest, daß eine Staphylokokkenkultur
ben Stammes, als Emulsion in Alkohol (96% und 64%) ge-
elt, absolut abgetötet wurde.

ide Versuche und ihre Gegenversuche bestätigen eine nun

Zit. unter 4), Seite 65, Versuch 13 (Protokoll Nr. 66).
Verhandl. d. Deutsch. Ges. für Chirurgie, 28. Kongr. Berlin 1899. S. 504.
20. 353.

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ziemlich allseits anerkannte Tatsache: Der Alkohol muß di
terien frei von gerinnbaren und kontraktionsfähigen
finden, wenn er sie abtöten soll, und weiter: Mittels e
starken Konzentration ist er im Beginn seiner Einw
imstande, sich selbst solche Bakterienumhüllung
schaffen.

Infolgedessen wirkt er gegenüber feuchten Bakterien a
trockene bilden um sich eine Hülle, müssen daher erst ange
werden, wenn der Alkohol in sie eindringen und sie abtöten

In diesem Sinne sprechen sich die meisten neueren Arbei
Unter anderen experimentierten mit trockenen und feuchten B
Harrington und Walker.[1]) Sie prüften Bact. coli, B. pyo
B. typhi, B. diphtheriae, anthracis, den Staphylococcus aur
albus und kamen zu folgenden Resultaten:

1. Auf trockene Bakterien ist absoluter bis 70%iger Alko
kungslos.

2. Sporenfreie Bakterien, feucht mit 40%igem Alkohol zus
gebracht, werden in 5 Minuten getötet. Manche Konzent
wirken schon in einer Minute.

3. Alkohol unter 40% wirkt langsam und unsicher.

4. Als geeignetste Konzentration gegen trockene wie feuc
terien erweisen sich 60—70%.

5. Die Hülle der trockenen Bakterien ist für hoch k
trierten Alkohol undurchdringbar. Wasserhaltiger A
gibt sein Wasser erst an die Hüllen ab und wirkt ers

6. Bei feuchten Bakterien hat Alkohol über 70% keinen
gegenüber dem weniger konzentrierten und man kann zur
infektion mit 60—70%igem auskommen.

7. Gelingt es, die tiefer in der Haut liegenden Ba
mit Alkohol in Berührung zu bringen, so werden mei
nuten zu ihrer Abtötung genügen.

Ruß[2]), der die vegetativen Formen der Handbakterien,
genen Bakterien und das Bact. coli prüfte, konnte diese sch
90—70%igem Alkohol abtöten. Von 40% ab wird die
mangelhaft. „Bei vorheriger mechanischer Reinigung mit
Seife und Bürste werden die an der Haut haftenden oberflä
Keime weggeschwemmt, die tiefer liegenden befeuchtet." „B
Vorbereitung nehmen die Keime wohl soviel Wasser auf,

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1) The germicidal action of alcool. Boston med. and surg. Journal 1

2) Zur Frage der Bakterizidie durch Alkohol. Zentralbl. für Bakter. u
Bd. 37, Heft 2, S. 280.

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genügend gequollen ist, um den nun nachfolgenden Alkohol, sei
n absolut oder geringgradig verdünnt (—80%), vollen Eingang
währen."

ch Hansen[1]) bestätigt die Einwirkung des absoluten Alkohols
uchte Bakterien und die der Verdünnungen auf angetrocknete.
nsen, der an ekzemkranker Haut prüfte, bestätigt außerdem
n mir festgestellte Tatsache[2]), daß er „weder unter den Haut-
nter den Ekzembakterien eine einzige sporenbildende Art" ge-
, somit seine Versuche sich nur mit vegetativen Formen befaßt

iter erwähne ich noch, daß auch Füth[3]) neuerdings die bak-
e Eigenschaft des Alkohol anerkennt; wenigstens entnehme ich
einen Worten: „Ahlfeld hat unstreitig recht, wenn er dem
l eine desinfizierende Wirkung zuschreibt."

rgirc[4]) setzt, „infolge des größeren Eindringungsvermögens", die
tende Kraft des konzentrierten Alkohols über die von zwei pro
Sublimat.

igl[5]) verhinderte die Gerinnung der mit Alkohol in Berührung
ngenden Stoffe durch tropfenweises Einträufeln des Alkohols
urch andauerndes Umschütteln und bestätigt ebenfalls die ent-
ene bakterizide Wirkung auf vegetative Formen.

dieser Einmütigkeit der Laboratoriumsversuche bleibt nur die
noch zu erledigen, ob der Alkohol auch die tiefsitzenden Bak-
der Haut erreicht. Experimentelle Versuche, unter meiner
kung von Rieländer[6]) und Fett[7]) ausgeführt, haben dies be-
. Dafür sprechen auch unsere Desinfektionsversuche, voraus-
, daß unsere Verfahren die Kritik bestehen.

einer umfangreichen Arbeit hat Sarwey[8]) unsere experimen-
Verfahren als ungenügend darzustellen versucht. Ich will hier
wiederholen, was ich gegen diese Schrift zu meiner Verteidigung
ben habe[9]), muß hingegen auf Grund neuerer Versuche und
ungen zunächst auf meine, von Sarwey so in den Hintergrund
gte Befürchtung eingehen, ein Teil der Sarweyschen Miß-

Tötende Wirkung des Äthylalkohols auf Bakterien und Hefen. Zentralbl.
t. usw. 1907, Bd. 45, S. 470.
20. 346.
Zentralbl. f. Gynäk. 1906, Nr. 33, S. 925.
Zeitschr. für Hygiene u. Inf. 1904, Bd. 46, S. 617. 5) Arch. f. Hygiene
d. 24, S. 273. 6) *22.* 7) *23.*
Bakteriologische Untersuchungen über Händedesinfektion und ihre Ergeb-
ür die Praxis. Berlin 1905.
29.

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erfolge, die er den nicht zu beseitigenden Handbakterien zus
kommen auf Luft- und andersartige Nebeninfektion hin

Im vergangenen Jahre habe ich, um auch in dieser Frag
eigene Versuche und Beobachtungen mir ein eigenes begründ
teil zu schaffen, hauptsächlich mit Petrischalen und festen
starrenden Nährböden gearbeitet. Diese Experimente haben
Befürchtung voll bestätigt, daß die Gefahr einer Verunrei
bei Benutzung von Petrischalen viel größer ist als b
brauch von Reagenzgläsern.

Die im hygienischen Institute gegossenen Schalen blieben
stets 24 Stunden zur Kontrolle im Brütofen und nachdem sie
geliefert waren und ich sie, wie oben beschrieben, mit eine
tigen Gummiband versehen hatte, damit Deckel und Schal
keinen Umständen sich voneinander entfernen konnten, ließ
vor dem Gebrauche nochmals mindestens 24 Stunden im Br
stehen.

Selbst von diesen unbenutzten, nicht geöffneten Schalen mu
kleine Anzahl ausgeschieden werden. Von den auch nur se
weise geöffneten, zeigten etwa 20% Verunreinigungen, die
sonders deutlich in den Fällen zu erkennen waren, wo ich a
Nährboden Zahlen oder Figuren — wie oben beschrieben — ge
hatte und dennoch, weitab von dieser begrenzten Aussaat,
Keime, also Luftkeime, sich entwickelten.

In Übereinstimmung hiermit beobachteten Stähler und Wi
bei Versuchen an 81 Frauen 50mal Verunreinigung der vo
verwendeten Platten durch Luftkeime, und Schäffer[2]) stellte
mentell fest, daß es in einem beträchtlichen Verhältnis nicht
Luftverunreinigungen von den Platten fernzuhalten. An
Stelle (Seite 88) gibt er dieser Tatsache durch auffallenden F
noch besonderes Gewicht: „Daher ist es unzweifelhaft, daß d
größere Zahl von Kolonien, die nach der Alkoholbürstung
Tageshand angegangen sind, lediglich der ganz unvermei
Luftinfektion anzurechnen ist.“

Bemerkenswert ist, daß auch Engels[3]) zugesteht, die g
einzelten Keime, deren Entwicklung in seinen Versuchen mit
Sublamin-Alkohol die absolute Sterilität verhindert haben,
durch Versuchsfehler entstanden sein, indem beim Gießen de
Eiterkeime aus der Luft übertragen worden sind.

1) Monatsschr. f. Geb. und Gyn. 1899, Juni, S. 737.

2) Experimentelle und kritische Beiträge zur Händedesinfektionsfrag
1902. S. 32.

3) Klinisches Jahrbuch 1905, Bd. 13, S. 592.

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eich im Beginn meiner Desinfektionsversuche, 1895, fiel mir Gefahr bei Benutzung der Schalen auf. Aus diesem Grunde n schnell viele Versuche machen zu können, wie es bei meiner chsanordnung notwendig war, verließ ich den Gebrauch der n und der festen Nährböden und benutzte Bouillon und Reaiser, da die Luftverunreinigung beim Gebrauch letzterer auf ein um herabsinkt.

näffer würde vielleicht zu noch günstigeren Resultaten zun der Heißwasser-Alkoholmethode gekommen sein, wenn er eine unbegründete Furcht vor dem Gebrauche flüssiger Nährgehabt hätte. S. 29 schreibt er: „Manche Versuchsanordnungen erart, daß es fast verwunderlich erscheinen kann, wie überhaupt ur in einem Falle Keimfreiheit erzielt werden konnte! Es gilt nz besonders bei der Verwendung flüssiger Nährböden zur g der Händesterilität. Da ein einziger in die Bouillon gelangter nnerhalb eines Tages sich vermillionenfacht und Trübung des Röhrchens verursacht, da schon ein kurz dauerndes Öffnen attestopfens hierzu bisweilen genügt, da die Partikelchen, welche r Hand abgekratzt und in das Röhrchen versenkt werden, mit gkeit Träger eines Luftkeims sein können, so läßt sich aus der ng des Bouillonröhrchens über die Sterilisierbarkeit der Hand d nimmer ein Schluß gewinnen."

se Folgerung hat doch nur eine Berechtigung, wenn die Bouillon übt, nicht aber, wenn sie klar bleibt, wie es bei meinen Vern tausendfach (wörtlich genommen) der Fall gewesen ist. ist die Behauptung, flüssiges Nährmaterial im Reagenzglas sei der Gefahr der Luftinfektion ungeeignet, auf das glänzendste egt.

ch Sarwey[1]) findet sich mit diesem Resultat nicht auseinander: en da nicht die meisten, wenn nicht alle Bouillonröhrchen lds getrübt sein, da doch bekanntlich bei der Bouillon ein r Luftkeim genügt, um eine diffuse Trübung des ganzen Röhrhervorzurufen? Und doch erzielt Ahlfeld trotz alledem Sterieiner Bouillonröhrchen." Die beiden von Sarwey gemachten ungsversuche sind, was den ersten anbetrifft, es sei so viel ol mit übergeimpft, daß die Luftkeime sich nicht hätten entn können, hinfällig. Den zweiten verstehe ich nicht.

stner, in seinem Referate über diese Antikritik Sarweys,

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[1]) Bakteriologische Untersuchungen über Händedesinfektion usw. Berlin 1905.

[2]) Verhandl. der Deutschen Gesellsch. für Gynäk. 1907, Seite 252.

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gebraucht den Ausdruck, im bezug auf die Abhaltung von Lu
seien meine Untersuchungen „aller Kautelen bar". Schon d
zende Resultat, was ich eben erwähnt habe und das durch Sc
und Sarweys Hervorheben dieser Tatsache noch mehr i
gestellt worden ist, müßte doch Küstner veranlaßt haben,
gelegenheit etwas mehr auf den Grund zu gehen. Er wür
aus meinen Publikationen ersehen haben, daß ich die Vers
einem Raume gemacht habe, der abwaschbare Wände hatte, nu
und Glasgegenstände enthielt, und daß die Schülerinnen bis
Augenblicke, wo die Abnahme erfolgte, die Hand in sterilem
liegen hatten.

Daß in der Tat unser Entbindungsraum, wenn er so z
suche vorbereitet war, arm an Luftkeimen war, beweist fo
alljährlich im Beginn des Hebammenunterrichts wiederholter

Um den Schülerinnen zu zeigen, daß überall in der Lu
vorhanden seien, stellte ich im Anfang der Stunde mehre
schalen offen hin und ebenso eine Anzahl Reagenzgläser,
bald mit sterilem Harn, bald mit Bouillon gefüllt hatte, un
sie nach der Stunde wieder. Während sich, je nach der Sa
des Raumes — im Auditorium ungleich mehr, als im Entbi
raume — auf der Agarbouillon der Schalen Keime entw
blieben die Reagenzgläser des öfteren vollständig klar, u
nach zwei, drei Tagen im Unterricht die Folgen der Lufti
zeigen zu können, brachte ich unveränderte Flüssigkeit mit.

Da nun bei meinen Desinfektionsversuchen die Öffnun
Reagenzgläser vor Abnahme des Wattepfropfens, wie üblich
werden, und der heißere Luftstrom des Inneren in der Richt
kälteren Außentemperatur abströmt, so ist dies, abgesehen
Kleinheit der Öffnung eines Reagenzglases, gegenüber der ein
schale, wieder ein Grund mehr, in der Luft suspendierte Ke
der Öffnung wegzutreiben.

Ein einfacher Versuch, den ich oft wiederholt habe, überz
leichtesten: Stellt man zwei Petrischalen im gleichen Raume,
auf eine erwärmte Unterlage (ich benutzte ein Glas mit heißem
bedeckt mit einem Papier), die andere auf eine kalte, so en
sich auf der kalten Schale annähernd noch einmal so viel Lu
als auf der warmen.

Für den Gebrauch flüssiger Nährmedien zum Nachweis von
kokken tritt auch, auf Grund umfangreicher Versuche, in neue
Zangmeister ein.[1]) „Ich verfüge heute über große Untersu

1) Zeitschr. für Geburtsh. und Gynäk. Bd. 58, S. 427.

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aus denen hervorgeht, daß flüssige Nährmedien zum Nach-
sc. der Streptokokken) absolut nötig sind; in manchen Fällen
das Wachstum auf Agar (im Gegensatz zur Bouillon), selbst
in dem betreffenden Sekret mikroskopisch der Nachweis der
kokken schon erbracht war. Ich muß mich deshalb der be-
en Empfehlung der flüssigen Nährböden durch Bumm[1]) an-
en."

komme nun zum wichtigsten Punkte der Einwürfe gegen meine
hsmethodik, wodurch eine Erklärung gebracht werden soll, auf
Weise meine guten Resultate, gegenüber denen „aller übrigen
mentatoren" zustande kämen. Es handelt sich dabei um den
f der weniger intensiven Abimpfungsmethode.
sen Vorwurf machen mir Sarwey, Engels und Schäffer
i, die überhaupt allein meine Methode eingehender geprüft

beschäftige mich zuerst mit den Arbeiten Paul-Sarweys und
, die, wenigstens in einer Versuchsreihe, mit zusammen 27
hen, ganz gleichmäßig vorgegangen sind.

ihre Versuche in einem sterilen Kasten vorgenommen sind,
weit es sich dabei um Abhaltung von Luftkeimen handelte, gar
Bedeutung für die Aufklärung der Differenz unserer Versuche,
ei meiner Versuchsanordnung sind, wie oben (Seite 550 u. fig.)
auseinandergesetzt, Luftkeime in störender Weise nicht in Frage
nen.

reit es sich um die von Sarwey betonte größere Aufweichung
nd im Schwitzbade des Kastens handelt, durch die tiefersitzende
eher an die Oberfläche gebracht wurden, wird folgende Be-
ng gerechte Zweifel an der Bedeutung dieses Umstandes er-
:

Kolumne 9 der Paul-Sarweyschen Tabelle ist der Keimgehalt
nd nach einem 10 Minuten dauernden Wasserbade, das im
uß an die Alkoholwaschung vorgenommen ist, festgelegt. Bis
sem Stadium gleicht die Ausführung der Desinfektion und die
hsanordnung genau der meinigen, nur daß ich den Keimgehalt
dewassers (Kolumne 8) nicht geprüft habe. Ja, ich habe sogar
fweichung der Hand meist 15—20 Minuten andauern lassen.

hrend nun in den 12 Versuchen Paul-Sarweys nur in einem
Dr. Meyer) danach die Hand keimfrei war, kam ich bei Proben
lbwegs geübten Schülerinnen auf 75% und darüber. Also bei
gleicher Methode solch immense Unterschiede. Auch

[1]) Verhandl. der Deutschen Gesellsch. für Gynäk. X, S. 584.

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gegenüber den Engelsschen Resultaten (Tab. Seite 518) ist
Tatsache zu berichten, aus der also unwiderruflich hervorge
die Ursachen der Differenz nur in der Ausführung d
nigungs- und Desinfektionsmaßregeln der Hand oder
späteren Behandlung der nach dem Wasserbade gewo
Handabschabsel liegen können.

Gewiß ist die auf die eben beschriebene Prozedur folge
reibung der Hand mit heißem sterilem Sand oder Marmorst
Untersuchung desselben auf seinen Keimgehalt eine sehr
Prüfungsmethode. Unmöglich aber konnte deren Resultat befri
ausfallen, wenn die Hände im Stadium vorher (Kolumne 9) ei
friedigendes Resultat ergaben. In dem einzigen Falle (Nr.
Meyer), wo die Hände nach dem Bade steril waren, zeigte si
der Keimgehalt des Sandes und der Handoberfläche gleich N

Gehen wir noch einen Schritt weiter und berücksichtigen
Differenzen der Resultate der einzelnen Untersuchungen untere
bei vollständig gleicher Versuchsanordnung, so werden wir der
der Frage, woher die Differenz, wieder ein Stück näher k
Schon in der Paul-Sarweyschen Tabelle muß es auffallen,
Experimentator, Dr. Meyer, obwohl der Keimgehalt seiner H
Beginn der Reinigung und nach der Heißwasser-Seifenwaschu
bei den übrigen Versuchspersonen, ein überreicher war, d
nahezu Keimfreiheit nach der Alkoholdesinfektion erreichte u
bei einem zweiten Versuche das beste Resultat aufwies.

So auch bei den Engelsschen Versuchen. Schäffer[1]) macht
aufmerksam, daß Engels bei den Kontrollversuchen meiner M
persönlich 100% „gute Resultate" erlangt habe, während d
suche der anderen Herren ein schlechteres Gepräge auf
Engels[2]) erklärt diese Behauptung als nicht zu Recht bes
Doch ist dem so, wie Schäffer angibt, denn Engels hat ni
rücksichtigt, daß Schäffer unter „gute Resultate" alle die zusa
gefaßt hat, die 0—20 Keime aufwiesen.

In der Tat hat dann Engels in den 5 Versuchen, die e
anstellte, 100% „gute Resultate", in 10, die von anderen Unters
gemacht sind, finden sich unter 130 Platten nur 116 „gute Res

Also auch hier ein deutlicher Unterschied abhängig vo
Versuchsperson.

Und nehme ich nun gar erst die Versuche von Engels mit
Alkoholgemischen hinzu und von diesen nur die mit Sublamin-A

1) Monatsschr. für Geb. und Gyn. Bd. 19, S. 693.
2) Monatsschr. für Geb. und Gyn. Bd. 20, S. 249.

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Schäffer[1]), und ich[2]), nachgewiesen haben, daß bei diesen chen der Alkohol das einzig wirksame Desinfizienz bedeutet, ersuche also mit den früheren auf eine Stufe zu stellen sind, ssen die ausgezeichneten Resultate der Versuchsperson Engels geschrieben werden.

ieweit da die Verschiedenheit der Hände, inwieweit die anlete körperliche und geistige Intensität bei der Desinfektionsrung in Frage kommt, läßt sich aus der Ferne nicht feststellen. l auf diesen, obwohl für mich ungemein wichtigen Punkt nicht eingehen, da ich ihn zu wiederholten Malen[3]) schon been habe.

einige, seit meiner letzten größeren Publikation[4]) zustimmende kungen anderer Autoren seien hier noch erwähnt:

nielsohn und Heß[5]) bringen einen sehr überzeugenden Beon der individuellen Verschiedenheit der Hände. Schon an t Wasser und Seife gründlich behandelten Hand des Dr. Heß sich vor der Desinfektion ungleich weniger Keime entnehmen, n der Hand des Dr. Danielsohn. Weit über 100—1000 bei m, meist unter 80, ja wiederholt gar keine bei ersterem. nliches Verhältnis bestand auch nach der Desinfektion.

asmann[6]) kommt bei seinen Händedesinfektionsversuchen ebenr Erklärung auffälliger Unterschiede der Resultate gleichartiger ktionsverfahren durch die Verschiedenheit der Hände der Verersonen.

ich meine guten Resultate zum Teil den Versuchen an Frauengemacht zuspreche, habe ich früher bereits erwähnt.[7])

nso auch, wie körperliche und geistige Anstrengung zu einem Resultate notwendig sind.[8]) Sehr treffend betont Klemm[9]) die erung an Intelligenz von seiten des sich Desinfizierenden: „Wir dabei sehen, daß der Vorgang der Händereinigung ein Prozeß an die Intelligenz des Betreffenden, sei er nun Arzt oder icht unerhebliche Anforderungen stellt. Wer den ganzen komen Akt schablonenhaft behandelt, darf sich nicht wundern, wenn Resultate hinter seinen Erwartungen und den Anforderungen,

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onatsschr. f. Geb. u. Gyn. Bd. 21, S. 193.
9. 1954.
0. 388.
0.
eutsche med. Wochenschr. 1902, Nr. 37, S. 662.
ünchener med. Wochenschr. 1907, Nr. 42 u. 43.
9. 1955.
0. 365.
eutsche Zeitschr. für Chirurgie Bd. 75, S. 514.

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die man zu stellen berechtigt ist, zurückbleiben. Es gehört zu durchgeführten Händedesinfektion ein ganz bedeutender Grad Konzentrationsvermögens; nur wer über jene bakteriologisch fühligkeit verfügt und beständig mit einer scharfen Linse vor geistigen Auge die einzelnen Etappen der Sterilisation verfol dem angestrebten Ziele, der möglichsten Keimfreiheit der H nächsten kommen."

Bei unseren Versuchspersonen, den Hebammenschülerinn ich persönlich diese Aufmerksamkeit übernommen, indem ich der Ausführung ununterbrochen darauf hingewiesen habe, diese, wie nun jene Stelle der Hände bei der Vorbereitung der nachfolgenden Behandlung mit Alkohol zu berücksichti und da die einzelne Versuchsperson, bei den ungemein zah Versuchen, oft an die Reihe kam, so hatten meine Schülerinn wie Ärzte und Praktikanten eine wohlgeleitete Desinfektion gemacht und Erfahrung darin.

Inwieweit in den Versuchen, die Krönig und Blumreich seits, Füth und Meißl[2]) andererseits angestellt haben, di tätigen Praktikanten und Hebammen den Anforderungen an forderliche Genauigkeit bei der Desinfektion entsprachen, k nicht beurteilen. Die Versuchsanordnung ist eine so kom und die Zahl der ausgeführten Versuche eine so geringe, da wenigen negativen Resultate mir nicht beweisend erscheine über meinen Versuchen, die schließlich auch an Genauig Abnahmemethode kaum noch eine Steigerung zulassen.

Was würden Krönig und Blumreich wohl für einen gemacht haben, wenn die Hebamme Lahrsow ihre Hand endeter Alkoholdesinfektion über eine Stunde lang unter B der Finger in ein Standgefäß mit $^1/_2$ Liter Nährbouillon gehalt und es wäre kein Wachstum erfolgt, die Bouillon wäre auf hinaus klar geblieben? Hätten sie da nicht, wenn sie gerecht wollten, sagen müssen: Bei einem derartigen Resultate ist e unwahrscheinlich, daß noch Keime an und in der Haut vo waren? Und so fiel ein unter meiner Leitung vorgenommer such[3]) aus, dem weiter die zahlreichen Versuche entspre denen ich statt des Standgefäßes große Nummern von Gum schuhen benutzte, in denen die desinfizierte Hand in Nähr suspendiert war.

1) Münchener med. Wochenschr. 1900, Nr. 29, S. 1005.
2) Zentralbl. für Gynäk. 1906, Nr. 33, S. 920 und Arch. f. Gyn. Bd.
3) *29*. 1955.

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h meine doch, daß diese Methode an Genauigkeit den eben
nnten mindestens gleichkommt. Und fragt man mich, warum
um die Richtigkeit meiner Behauptung von der Tiefenwirkung
lkohol zu beweisen, nicht auch die Krönigsche oder Füthsche
gemacht habe, so muß ich auf die Schwierigkeiten hinweisen,
in derart komplizierter Weg mit sich bringt. Fehler sind unge-
schwer zu vermeiden und jeder Fehler täuscht einen Mißerfolg
r Desinfektionsmethode vor, Mißerfolge, die erst ausgeglichen
en, wenn man in großer Zahl diese Versuche wiederholen würde.
gegen sind meine Handschuhversuche ungemein einfach auszu-
n und wenn Sarwey ausnahmslos andere, d. h. ungünstige Er-
gehabt hat, als ich, was bleibt dann anderes übrig anzunehmen,
aß doch wahrscheinlich die Händedesinfektion an Genauigkeit
ünschen übriggelassen hat, oder die nachträgliche Behandlung
Handschuhinhalts hat Verunreinigung herbeigeführt.

ie Bewertung der Händedesinfektionsexperimente gestaltet
je nach ihrem positiven oder negativen Ausfalle, ganz verschieden.
nach sorgfältiger Händedesinfektion die bakterielle Untersuchung
stig aus, so ist die Zahl der möglichen Ursachen, die teils bei
usführung der Desinfektion, teils bei der experimentellen Prüfung
Resultat beeinflußt haben können, eine sehr große. Fällt die
ng aber günstig aus, sind keine Keime auf der Platte oder in
Nährflüssigkeit aufgegangen, dann können die Ursachen, auch
es sich um einen Scheinerfolg handelt, nicht in der mangel-
n Händereinigung liegen, sondern nur in Prüfungsfehlern. Die
der Fehlerquellen ist daher eine viel geringere, der einzelne
r leichter erkennbar.

eshalb sind meine Resultate nach dieser Seite hin viel leichter
rüfen, als die der Gegner, die keine oder nur selten eine Keim-
eit erzielten und dies um so mehr, da meine Prüfungsmethoden
ger kompliziert sind, als die der anderen und infolgedessen
in großer Zahl ausgeführt werden konnten.

eim gegeneinander Abwerten der ungünstigen und günstigen Ex-
nente darf ich zuletzt noch an den von mir bereits referierten[1])
pruch v. Behrings erinnern: „Schlechte Resultate zu bringen
icht, gute sehr schwer."

Wenn ich jetzt noch einmal zusammenfassend den Vorgang schil-
in welcher Weise die Heißwasser-Alkohol-Reinigung
nfizierend auf die Hand wirkt, und daran anknüpfend die
elnen Momente der Ausführung der Methode anfüge, so kann

[1]) *20.* 355.

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der mit den Streitfragen weniger bekannte Leser sich am sc
einen Überblick verschaffen und dem Praktiker ist genau die
angegeben, die er einzuschlagen hat, wenn er auf eine erf
Desinfektion seiner Hand rechnen will.

Die Wirkung der Heißwasser-Alkohol-Desinfekti
sich aus mehreren Komponenten zusammen, deren einzelne
genügenden Erfolg erzielen, während richtige Aufeinanderf
Zusammenwirken den Erfolg der Keimabtötung bis in die
Schichten mit sich bringen.

1. Durch die der Alkoholanwendung vorausgehende Wa
der Hand mittels Seife und Bürste findet

a) eine erste Entfernung der oberen Epithelschichten statt
b) erfolgt eine Verseifung des oberflächlichen Hautfettes
c) eine Aufquellung der tieferen Hautschichten.

2. Die Entfernung der obersten Epithellagen wird vervol
durch Abspülen und durch Abreiben mit einem tr
mäßig rauhen Handtuche.

3. Wird nun auf diese so vorbereitete Haut konzent
Alkohol mittels Bürste oder Flanell verrieben,

a) so wirkt der Alkohol fettlösend,
b) dringt infolge Diffusion in die tieferen Schichten der
feuchteten Haut, tötet, wahrscheinlich durch Wasserent
die dort lebenden Bakterien und, indem er nach und
nun obere Hautschicht härtet, ermöglicht
c) ein weiteres mechanisches Entfernen der nun festere
schuppen und mit ihnen einer weiteren Schicht Bakter

Wird die Händedesinfektion unter diesen Gesichtspunkt
geführt, so muß der Erfolg der sein, daß nach einer späte
weichung der obersten Schicht keine, oder unter Umständen n
liche Keime aus der Tiefe nach oben treten.

Der Erfolg muß anders ausfallen, wenn in umgekehrter
folge verfahren wird:

Wendet man erst den Alkohol an, ohne vorherige Aufw
der Haut, so wirkt er gerbend, verlegt sich selbst den Weg
tieferen Schichten, seine fettlösende Eigenschaft kann wenig
sam sein, und der Erfolg würde einfach in einem Abbürste
fernen der oberen Schicht bestehen.

Jede nachfolgende Aufweichung, mag sie experimentell du
Wasserbad erfolgen, mag sie bei Gelegenheit einer Operatio
unter dem Gummihandschuh durch Schweißproduktion erfolge
den in der zweiten und tieferen Schicht der Haut sitzenden B
den Weg auf die Oberfläche.

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dieser Art der Händedesinfektion ist die Oberfläche der Hand
e kurze Zeit keimarm oder gar steril; je mehr aber die Haut
cht, um so zahlreicher werden die Bakterien auf der Hand
. Diese Methode, die wir als Schnell-Alkoholdesinfektion*)
nen wollen, ist nur im Notfall und nur bei einem sehr kurz
den Eingriff zu gebrauchen.

wendet man ohne vorhergegangene Aufweichung der Hand
wasserhaltigen Alkohol, 60—40%igen, so gibt der Alkohol zu-
Wasser zur Aufquellung der Haut ab und führt dann seine ad-
rende Wirkung nicht in dem Maße aus, wie der wasserarme.
bakterizide Wirkung ist dementsprechend geringer, wie auch
Tiefenwirkung. Es erklärt aber diese Deutung die Differenzen
Experimenten, wonach einzelne Untersucher bessere Resultate
ring konzentriertem, andere mit stärker konzentriertem beob-
haben.

der Ausführung der einzelnen Akte der Händedes-
ion sind, unter Berücksichtigung der oben angeführten Punkte,
eiten von Wichtigkeit, soll der Erfolg gesichert werden, und
für mich keine Frage: mancher Mißerfolg im Experiment rührt
her, daß auf diese Einzelheiten nicht geachtet ist.
Man benutze warmes Wasser, 35—45° C.

) neuerer Zeit sind Schumburg[4]) und v. Herff[2]) für diese Schnell-
tion eingetreten. Schumburg empfiehlt die Seifenwaschung wegzulassen
kohol von ca. 50% mit Wattebäuschchen einzureiben. Die Keimver-
ag betrug um 99%. Bei der Versuchsanordnung ist die langdauernde Auf-
g der Hand in heißem Wasser unterlassen. Auch möchte ich nicht, wie
burg es getan hat, bei dieser Versuchsart dem noch an der Hand befind-
lkohol jede Entwicklungshemmung absprechen.

Recht fragt Große[3]): „Warum Schumburg nach diesen so ausgezeichnet
en Prozeduren noch die Anwendung eines Antiseptikums vorschlägt, ob-
diese selbst für überflüssig erklärt, ist nicht einzusehen."

der Tat ist es eine höchst merkwürdige Erscheinung, daß die meisten
en und Gynäkologen auf eine ausgiebige Alkoholdesinfektion noch das
folgen lassen, ein Mittel, das als Händedesinfiziens weit unter dem Al-
ngiert und außerdem viele Nachteile hat.

h die neue von v. Herff empfohlene Alkohol-Azeton-Desinfektion, ohne
e Waschung, halte ich für eine Schnelldesinfektion. Ich bin gerade mit
üfung beschäftigt.

Recht empfiehlt Schumburg seine Methode für Kriegszwecke und Eilfälle.
Herff betont den Vorteil der Schnelligkeit.

rchiv für Klin. Chirurgie, Bd. 79, Heft 1. — Deutsche med. Wochenschr.
. 41, S. 1712. — Deutsche med. Wochenschr. 1908, Nr. 8, S. 330.
Oeri, Korresp. f. Schweizer Ärzte 1908, S. 56. — Hegars Beitr. Bd. 12, S. 493.
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07, Nr. 40.

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2. Ehe man zu bürsten beginnt, schäume man die Hände mit Seife ein, reibe die Handflächen beider Hände gegenei auch die Finger der einen Hand zwischen die der anderen, die Finger der einen Hand krallenartig in die der anderen auch die Fingerkuppen und der Unternagelraum beim Wasch Seife genügend getroffen werde und spüle die Seife zu wiede Malen im Wasser ab, um diese Prozedur mit Benutzung Wassers zu wiederholen.

3. Folgt Nagelkürzung und Reinigung des Unternagelrau des Nagelbetts. — Entgegen der von Haegler vertretenen u Engels akzeptierten Ansicht, der Nagel solle 3 mm lang plädiere ich für eine weitgehende Kürzung, so daß man mit und Flanell den Unternagelraum direkt treffen kann.

4. Nochmalige kurze Waschung mit Seife, Abspülen.

5. Abreiben der Hand und der einzelnen Finger mit einem nen Handtuche.

6. Nun folgt der Hauptakt der präliminaren Reinigung: das mit einer nicht zu kleinen kräftigen Wurzelbürste.

Die saubere Bürste wird in warmem Wasser ein wenig — sie nimmt dann besser die Seife an — und nachdem mehrere Male über die Seifenfläche weggeführt hat, bürstet Hand, genau achtgebend, daß auch jeder Teil derselben, jedes an Zeit und Intensität bei der Bürstung reichlich bedacht wer schwer zu reinigende faltenreiche Haut des Handrückens m durch Faustbildung straffen und auch jeden einzelnen Finge Bürsten bald einschlagen, bald strecken. Man denke dabei be auch an den Daumen, den man für gewöhnlich nicht so beugen pflegt. Ebenso muß man sorgfältig darauf achten, Bürste den Unternagelraum gehörig mit berücksichtigt.

Diese Prozeduren (1—6) können bei Zeitüberfluß auf 10 und darüber ausgedehnt werden, unter wiederholtem Erneu warmen Wassers. Jedenfalls sollen sie nicht unter 5 Minuten

7. Abspülen der Seife und Abreiben der Hand und jedes e Fingers mit einem trockenen Handtuche.

8. Zur nun folgenden Alkoholdesinfektion benutze man ein Gefäß und eine reichliche Menge 80—90%igen Alkohols. Di muß vorher in Alkohol gelegen haben. Wir führen sie in de in einem kleinen Glaszylinder mit, der oben durch einen K schlossen ist. Bei der Bürstung muß die Bürste tüchtig un Alkohol getaucht werden und man muß ebenso, wie bei de wasserwaschung, genau jeden einzelnen Teil der Hand u Fingers vornehmen, die Hand also bald strecken, bald beu

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ballen und die Nagelgegend besonders berücksichtigen. Zeit-
mindestens 3 Minuten.

Nach der Bürstung folgt die Abreibung mit Flanell. Der sterile
lappen, 30 cm im Quadrat, wird in Alkohol getaucht, über die
sinfizierende Hand gestülpt und nun besonders die einzelne
kuppe durch stopfende und drehende Bewegungen mit dem
ol in Berührung gebracht, die beste Methode, um bei kurz ge-
tenem Nagel den Alkohol unter die Nagelkuppe und in das
ett hineinzubringen. Zeitdauer 2—3 Minuten.

mit ist die Desinfektion beendet. Soll aber eine experimentelle
stattfinden, so muß der Alkohol aus der Haut mittels sterilen
rs ausgelaugt werden. Wir haben dann so verfahren:

). Die desinfizierte Hand wird unmittelbar aus dem Alkohol in
terile Waschschüssel, die mit $1^1/_2$—2 l sterilem, 45° C. warmem
r gefüllt ist, gebracht. Die Schüssel stand schon vorher bereit
st mit einem sterilem Leintuch vollständig bedeckt. Das Tuch
an einer Stelle etwas gelüftet und die Hand schlüpft aus dem
ol unmittelbar in die Heißwasserschüssel und bleibt, samt dem
rarm, dauernd vom sterilen Tuche bedeckt.

e 5 Minuten wird eine zweite gleiche Schüssel daneben gestellt
lie Hand schlüpft wiederum unter dem Leinentuche in diese
sel. Wir haben meist drei Schüsseln benutzt, also 15—20 Mi-
Zeit auf die Aufweichung und Auslaugung der Hand verwendet.

ird von den 9 Manipulationen nur eine nicht mit der nötigen
rksamkeit gemacht oder gar unterlassen, so ist damit vielleicht
rund gegeben, daß, falls eine bakterielle Untersuchung vorge-
en wird, das Endresultat nicht nach Wunsch ausfällt.

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Verzeichnis meiner und meiner Schüler Arbeiten über Händedesinfektion.

1895.

1. Ahlfeld, Die Pflege der Hände und ihre Vorbereitung zur geburtsh Untersuchung. Allgem. Deutsche Hebammen-Zeitung Nr. 7.
2. —, Welche Faktoren sind bei der Desinfektion der Hand zu berücksi Monatsschrift für Geburtsh. u. Gynäkol. Bd. 1, S. 262.
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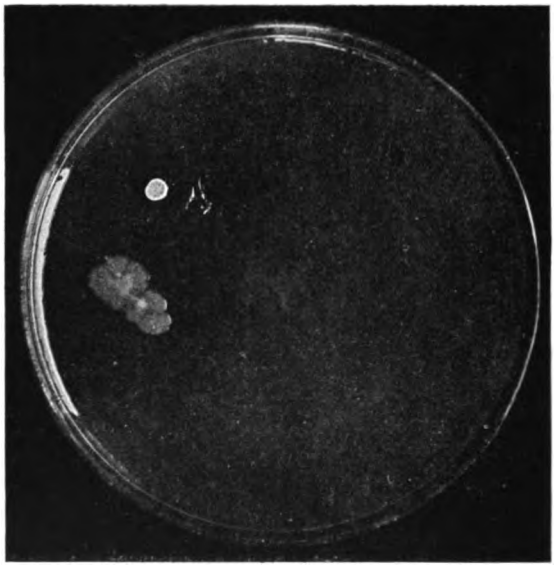

Fig. 15

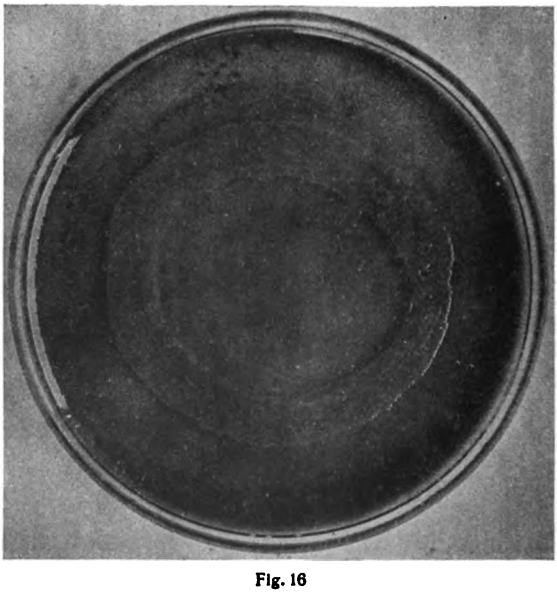

Fig. 16

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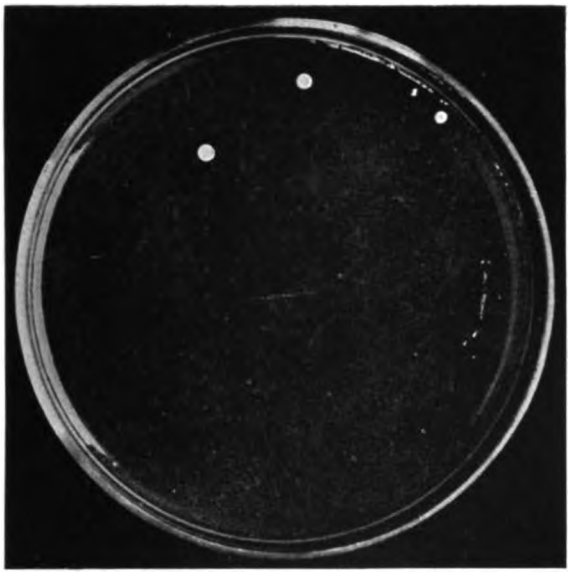

Fig. 9

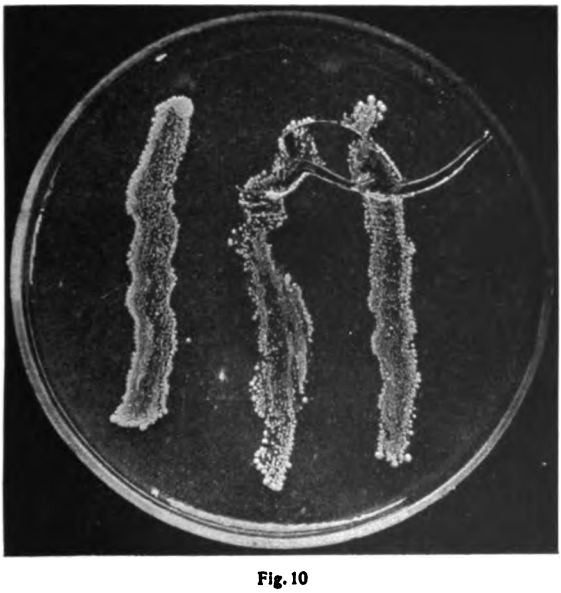

Fig. 10

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die letzten Chancen bei der vorhandenen Allgemeininfektion d
toneums zu bieten.

d) innerhalb der ersten 48 Stunden nach Beginn des Sch
falls = (absolute) Frühoperation.

Ausnahmsweise kann man bereits innerhalb 48 Stunden
ginn des Anfalls vor einer mehr oder weniger diffusen Peria
zitis stehen. Dann würde die Notoperation mit der absolute
operation zu identifizieren sein.

Die Frage der Operation im intermediären Stadium wird
sammenhang mit den Spätoperationen näher erörtert werden,
Fälle nur ausnahmsweise bei dringender Indikation zur O
gelangten und sonst in der Regel erst nach dem 10. Tage oper
gegangen wurden.

Nachfolgende kleine Tabelle gibt eine gedrängte Übersi
die Frequenz und die Mortalität der verschiedenen Kateg
den einzelnen Jahrgängen. Die Zahl der gestorbenen Fälle i
in Klammern beigefügt.

	Sicherheitsop.	Spätop.	Notop.	Frühop.	Nicht op.
1894/95	—	3	—	—	—
1895/96	—	2(1)	2(2)	—	2
1896/97	2	10	—	—	3
1897/98	6	5	—	—	4
1898/99	5	9	1(1)	—	2
1899/00	9	9	3(1)	—	1
1900/01	14	3(1)	—	—	2
1901/02	22	9	3(2)	—	3
1902/03	29	10(2)	3(3)	—	1
1903/04	43	9(1)	2(2)	1	1
1904/05	53	12(1)	2(2)	3	4
1905/06	49(1)	6	3	7	2
1906/07	114(1)	26(3)	8(6)	16	12(1)
Summa	346(2)	113(9)	27(19)	27	37(1)

Man ersieht aus obiger Tabelle ein gleichmäßiges Anste
wohl der Fälle in ihrer Gesamtheit, als auch der chronisch
von Jahr zu Jahr. Es ist dieses wohl als die Folge zu be
der nicht nur bei den Ärzten, sondern auch im Publikum
weiter verbreiteten Kenntnis von dem schnellen und sichere
einer Operation namentlich im chronischen Stadium, gegenü
unsicheren Prognose bezüglich späterer Anfälle bei konservat
handlung.

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uffallend ist es, daß trotzdem die Spät- und Notoperationen sich Zahl nach auf einem ziemlich gleichbleibenden Niveau bewegen. s könnte vielleicht die Folge der immer zahlreicheren Eingriffe hronischen Stadium sein, die bereits nach dem ersten Anfall ch vorgenommen werden, so daß es naturgemäß immer seltner ntstehung der schwereren Krankheitsbilder kommt, welche die und die Notoperation erforderlich machen.

ngegen bewegt sich die Zahl der Frühoperationen, welche erst lem Jahr 1903/04 einsetzt, in schnell aufsteigender Linie und voraussichtlich mit der immer größeren Tendenz zum möglichst eitigen Eingriff auch ferner noch ansteigen.

züglich der Mortalität ergibt naturgemäß die Sicherheits- ation das beste Ergebnis mit 2 Todesfällen an Peritonitis auf Operationen. Das entspricht einem Prozentsatz von 0,58 Todes-, der mit den Ergebnissen anderer Statistiken ziemlich über- mmt.

hat Kümmel auf 695 Sicherheitsoperationen 4 Todesfälle = % zu verzeichnen. Die Todesursachen waren einmal Peritonitis, al Lungenembolie und einmal Pneumonie.

aberer berichtet aus der v. Eiselsbergschen Klinik von 199 Sicher- perationen ohne Todesfall, ebenso hat Mikulicz in 5 Jahren Sicherheitsoperationen ohne Todesfall zu verzeichnen gehabt. hat von 702 Sicherheitsoperationen einen Fall an Ileus und anderen Patienten an Embolie verloren = 0,28% Mortalität.

gegen hat Sprengel auf eine erheblich höhere Zahl von Sicher- perationen 0,86 Todesfälle zu konstatieren.

erzu ist zu bemerken, daß kleinere Statistiken wie die von rer und Mikulicz leichter günstige Resultate liefern können, ie größeren, bei denen gewisse Komplikationen leichter das ntresultat trüben werden. Man wird eben nie an einem größeren ial aus verschiedenen Altersklassen gewisse postoperative Kom- ionen wie Embolien, Bronchopneumonien usw. ganz ausschalten en. Diese Komplikationen dürften auch für die Zukunft die lität der Sicherheitsoperation nicht viel unter 1% herunter- n lassen.

esentlich höher gestaltet sich nach unserer Tabelle die Mortalität Spätoperation im Stadium der umschriebenen Periappendizitis. ommen hier auf 113 Operationen bereits 9 Todesfälle = 7,9% lität.

n fast gleichartiges Verhältnis konnte Haberer konstatieren, der 1 Fälle von Spaltung periappendizitischer Abszesse 3 Todesfälle % zu verzeichnen hatte.

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Auch Silbermark (Mosettig) berechnet bei den einfachen der Periappendizitis die Mortalität ebenfalls auf 6%.

Sprengel hat 5—10% Mortalität gefunden, wobei er all unterscheidet zwischen Früh- und Spätoperation der umgrenzte appendizitis.

Dannehl hat in der Berliner chirurgischen Klinik auf 4 appendizitische Abszesse 5 Todesfälle gefunden = 11,6%, welc noch die Kümmelsche Mortalitätsziffer etwas übersteigt.

Nach diesen wenigen Vergleichszahlen dürfte die Durchs ziffer der Mortalität bei operativer Behandlung der umschr bzw. abgekapselten Entzündung zwischen 5—10% schwanken. ist zu berücksichtigen, daß das Material der einzelnen Statisti hebliche Verschiedenheiten aufweisen muß. Der Begriff d schriebenen Periappendizitis liegt zwischen ziemlich weiten G je nachdem die Umgrenzung der Entzündung früh oder spät ist, und je nachdem ein kleinerer oder größerer Teil des Perit davon ergriffen ist. Es kann sich demnach dabei um Fälle v verschiedener Schwere handeln.

Ferner kann der Zeitpunkt der Operation ein ganz versch sein, je nachdem der Eingriff kurz nach Vorübergehen des i diären, des Zwischenstadiums, also nach dem 7.—8. Krankhe oder erst bei dem deutlichen Nachweis einer vorhandenen A bildung erfolgt. Dementsprechend sind auch die Unterschiede Ergebnissen der einzelnen Zusammenstellungen zu erklären.

Die höchste Mortalitätsziffer finden wir naturgemäß bei de operation im Stadium der allgemeinen Periappendizitis bzw tonitis appendicularis diffusa. Es kommen daselbst nämli 27 Operationen 19 Todesfälle = 70,4% mit tödlichem A Sämtliche geheilten Fälle außer 2 sind in den ersten 48 S nach dem mutmaßlichen Zeitpunkt der Perforation bzw. der Verschlechterung des Leidens, alle übrigen Fälle sind spät Operation gelangt. Um gleichwertige Ergebnisse zu erzielen, man für die Notoperation eigentlich nur die Fälle heranziehe später als 48 Stunden nach erfolgter Perforation operiert si dieser Zeitpunkt bekanntlich die Grenze bildet zwischen gü und ungünstiger Prognose für den weiteren Verlauf der betref Fälle. Nur darf man dabei nicht die Fälle vor 48 Stunden sc weg als Frühoperationen bezeichnen, sondern es empfiehlt sich Fälle unter dem Namen der relativen Frühoperation gehen zu die ja viel später nach Beginn des Schmerzanfalles erst auf di tive Indikation der Perforation hin unternommen wird im Ge zu der absoluten Frühoperation, die allein auf die absolut

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n des Schmerzanfalles hin in jedem Falle innerhalb der ersten unden unternommen wird. Es liegt auf der Hand, daß mit dem nehmen der relativen Frühoperation das Gesamtresultat der peration ein verschiedenes sein wird je nach der Zahl der die in den ersten 48 Stunden nach Eintritt der Perforation ert sind.

ementsprechend erhöht sich in unserer Tabelle nach dem Aus- len der relativen Frühoperationen die Mortalität von 70,4% für älle bis auf 85%, während auf die (relative) Frühoperation der en Periappendizitis bei unserer kleinen Statistik dann das Er- s von 14,3% Mortalität entfällt.

berer berichtet bei 33 Fällen von diffuser Periappendizitis von desfällen. Das ergibt 76% Mortalität. Sprengel findet 1901 ortalität der freien Periappendizitis bei Frühoperation mit 44%, pätoperation mit 70%. Im Jahre 1905 hat dann derselbe Autor relative Frühoperationen nur noch 4 Todesfälle = 4%, womit Ergebnis unserer Aufstellung erheblich übertrifft, während auf pätoperationen nur 40 Todesfälle = 25% Mortalität entfallen. res Ergebnis namentlich muß als ein überaus günstiges bezeichnet n und ist wohl mit auf die frühere Operationszeit und die fort- ittene Technik des Operateurs zurückzuführen.

was schlechter, aber immer noch relativ günstig ist das Ergebnis lbermark, der auf die Frühoperation 10% und auf die Spät- ion der freien Periappendizitis 45,9% Mortalität findet.

esentlich höher ist bereits die Mortalität bei Trendelenburg, f 86 Fälle freier Periappendizitis mit 55 Todesfällen auf 64% ität kommt. Und zwar heilten bei der Operation am ersten 6,6%, am zweiten Tage noch 48%, später dann 0% der ge- Fälle. Damit übertrifft Trendelenburg noch das ungünstige is unserer Statistik der Notoperation nach 48 Stunden.

nnehl an der Berliner chirurg. Klinik unterscheidet bei seiner llung zwischen Appendicitis perforativa, gangraenosa, akut pro- nter und allgemeiner Periappendizitis. Fassen wir diese 4 en unter dem Titel der Periappendicitis destructiva zusammen, eben dieselben auf 57 Operationen 42 Todesfälle = 74,4% Mor-

Mikulicz hatte 1903 auf 24 Fälle von freier Entzündung nur ungen = 83% Mortalität.

t am höchsten kommt Kümmel in seiner Aufstellung, der auf erationen 73 Todesfälle = 89% Mortalität konstatieren konnte. schwanken demnach die Mortalitätsangaben der Notoperation en einzelnen Zusammenstellungen zwischen 25—93%, je nach

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der Zahl der früher oder später operierten Fälle, welche da
treten ist, und nach der Auffassung des weiten Begriffes de
meinen Peritonitis seitens der Autoren. Ein einheitlicheres
wird sich aus eben diesem Grunde gerade bei der Notoperat
kaum jemals erzielen lassen.

Kommen wir schließlich zu der Mortalität der absoluter
operation in den ersten 48 Stunden nach dem Beginn des
Schmerzanfalles überhaupt, so finden wir dieselbe in unserer
Zusammenstellung von 27 Fällen mit dem befriedigenden
von 0% vertreten. Den gleichen günstigen Prozentsatz fin
auch von Nordmann, C. Dowd, Dannehl, Karewski und
Autoren angegeben, während Mahar 2,4% und Israel 2% M
gefunden hat. Das günstige Ergebnis unserer Zusammenstellu
auf 2,9% Mortalität herabgesetzt, wenn wir die 7 Notope
innerhalb 48 Stunden nach der Perforation noch hinzunehm
ja in die Kategorie der absoluten Frühoperationen nicht eige
rechnen sind, da sie zu einem anderen Zeitpunkt und aus
Indikation heraus unternommen sind.

Die Differenz der Ergebnisse bei den verschiedenen Aut
klärt sich hier wohl zum Teil aus der zeitlichen Verschieden
Beginns der Erkrankung mit dem Manifestwerden der erst
ptome derselben. Der letztere Zeitpunkt kann naturgemäß
klinische Zeitberechnung des Operateurs der einzig maßgeben
Da aber der Krankheitsprozeß oft schon eine Zeitlang vo
standen haben kann, so kann der tatsächliche Zeitraum von
der Erkrankung bis zu der angeblichen Frühoperation oft
erheblich längerer sein als 48 Stunden. Daraus erklären si
einmal die auffallend weit vorgeschrittenen pathologischen
rungen, die bisweilen so früh zu konstatieren sind, und and
eben die Mortalitätsdifferenzen, die einzelne Statistiken auf
haben und auch ferner aufzuweisen haben werden.

Die Besprechung der Mortalität im Zwischenstadium,
kanntlich vom 3.—8. Krankheitstage in der Regel gerechn
behalte ich mir für die nähere Erörterung der Spätoperatio

Wenden wir uns zunächst der näheren Besprechung der
heitsoperationen im chronischen Stadium zu, so können
selbe ziemlich summarisch gestalten, da die einzelnen Fälle
nigen Ausnahmen nichts Abweichendes von dem typischen Kr
verlaufe bieten.

Bezüglich des Geschlechts der Patienten finden wir dem
anderer Aufstellungen entsprechend vorwiegend das männli
schlecht betroffen. Es entfallen von 346 Fällen 220 Erkrank

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das männliche, 126 Fälle auf das weibliche Geschlecht. Das entcht analog den anderen Statistiken einem ganz erheblichen Untered der beiden Geschlechter, nämlich 63,6% männlichen und 36,4% olichen Patienten.

Dannehl hat in seiner Aufstellung einen erheblich höheren Untered, nämlich 135 Männer und 52 Frauen gefunden. Diese Zahl pricht einem Verhältnis von 72,2%:27,8%. Fast genau dasselbe zentualverhältnis hat Hoffmann in seiner Sammelstatistik von Fällen mit 1544 Männern und 587 Frauen berechnet. Er erhielt lich danach ein Verhältnis von 72,4%:27,6%.

Auch Sprengel hat in seinen zahlreichen gesammelten Aufstelen der verschiedensten Autoren bis auf wenige Ausnahmen eine rwiegende Beteiligung des männlichen Geschlechts an der Appenrkrankung feststellen können. Die Gründe für diese Erscheinung n wir bereits bei der Ätiologie der Erkrankung des näheren bechen.

Dem Alter nach verteilen sich die Patienten auf die einzelnen ennien folgendermaßen: Von 1—10 Jahren: 16 Fälle, von 11—20: von 21—30: 124, von 31—40: 68, von 41—50: 27, von 51—60: nd von 61—70 Jahren 3 Fälle. Es ergibt sich hieraus ein eriches Ansteigen der Erkrankung vom 10. bis 30. Jahre. Danach gt ein ziemlich steiler Abfall der Frequenz bis auf nur 3 Fälle etzten Dezennium.

Die Zahl der vorausgegangenen Anfälle bewegt sich zwischen 7 Anfällen von wechselnder Schwere und zwar war vorausgeen 1 Anfall in 86 Fällen, 2 in 93, 3 in 76, 4 in 38, 5 in 26, 14, 7 in 4, 8 in 4, 9 in 1, 11 in 3 und 17 Anfälle in 1 Falle. Daentfällt auf die Mehrzahl der Operationen, nämlich 255, eine von 1—3 vorausgegangenen Anfällen. Die darüber hinausnde Zahl der Anfälle verteilt sich auf nur 91 Fälle. Man ersieht us, daß die Mehrzahl der chronischen Fälle verhältnismäßig früh Operation gekommen ist.

Der Zeitpunkt der Operation liegt innerhalb der ersten 9 Wochen dem letzten Anfall in 208 Fällen. 129 Fälle verteilen sich auf 10.—50. Woche nach dem letzten Anfall, während 9 Fälle nach hr und noch später zur Operation gelangten!

Die Behandlungsdauer der chronischen Fälle beträgt 8 bis Tage in 197 Fällen, 21—30 Tage in 106 Fällen, 31—40 Tage 2 Fällen, 41—50 Tage in 12 und 51—60 Tage in 9 Fällen. nach liegt für die meisten Fälle, nämlich 303 Patienten, die ndlungsdauer zwischen 2—4 Wochen. Daß bei den übrigen Fällen ein so langer Zeitraum von 31—60 Tagen zur Heilung

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erforderlich war, erklärt sich daraus, daß die Behandlun vom Zeitpunkt der Aufnahme ab gerechnet wird, und daß b Anzahl von Fällen erst eine gewisse Zeit zum Abklingen des Anfalles in der Klinik notwendig war, bevor zur Operat schritten werden konnte. Dazu kommen noch diejenigen Fälle Heilung sich durch postoperative Komplikationen nachträgli zögerte.

Der Processus vermiformis ist in sämtlichen 346 chro Fällen entfernt worden.

Von anamnestischen Angaben findet man die übliche T Appendizitis fast in allen Fällen vorausgegangen: Schmerz und empfindlichkeit in der Regio ileocoecalis, mehr minder hohes und Obstipation. Seltner Übelkeit und Erbrechen und noch Durchfälle und Urinbeschwerden.

Blasenbeschwerden werden im ganzen in 17 Fällen an Sie sind bedingt durch meist ältere Adhäsionen an der Blase siduen des Übergreifens einer früheren Entzündung auf den mi Peritonealüberzug bedeckten Teil der Blase. Der Prozents 5,0% dafür auf 346 Fälle dürfte als auffallend gering zu bez sein.

Vorausgegangene Durchfälle sind bei 20 Patienten v = 5,8%. Einmal war der betreffende Fall gleichzeitig mit beschwerden verbunden. In 2 Fällen (59 und 194) war frü Durchbruch in den Darm erfolgt, der sich durch Eiterabgä Stuhl äußerlich manifestierte.

Schmerzen in das rechte Bein ausstrahlend werden 108 und 96 angegeben. Im letzten Fall wurde auch gleichzei Schmerzen geklagt, die in die rechte Schulter ausstrahlten einem Anfall von Gallenkolik.

Eine Venenthrombose rechts gelegentlich eines frühe falls hatte Fall 7 vor 4 Jahren durchgemacht. Seit $2^1/_2$ Jah stand in dem Fall noch eine Eierstockentzündung links. D Fall bekam dann während des Heilungsverlaufes nach der Ap tomie eine Thrombophlebitis der Vena femoralis links, die handlungsdauer auf 26 Tage verlängerte. Das Nähere übe Komplikation wird später besprochen werden. Die gleiche kation war in 3 Breslauer Fällen vorausgegangen, so daß 4 Fällen auf einen Prozentsatz von 1,1% kommen.

Gallenkoliken ohne Ikterus sollen in einem Breslauer wie in meinem Fall 46 seit 8 Jahren bestanden haben. In le ist nur ein appendizitischer Anfall 8 Wochen vor der Aufnah hohem Fieber angegeben. Eine Revision der Gallenblase au

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ra operationem nicht erfolgt. Die Gallenkoliken ohne Ikterus
n vielleicht eher früheren katarrhalischen Appendikularkoliken
ochen haben, da der Processus in diesem Falle relativ hoch
ert gefunden wurde. Der Prozentsatz beträgt mithin 0,6%.
s vorausgegangene Erbrechen soll in 2 Fällen (2 und 120)
blutigen Charakter gehabt haben: ob als Folge eines gleich-
vorhandenen Magengeschwüres oder einer Schleimhautblutung
die Anstrengung des vorausgegangenen Brechaktes ist aus der
engeschichte nicht zu ersehen.
ustfellentzündung rechts mit reichlicher Eiterentleerung per
im Fall 171 im Anschluß an einen vor 8 Wochen vorausge-
en Appendizitisanfall aufgetreten und bis zur Aufnahme des
en in die Klinik spontan zur Rückbildung gelangt. Ferner ist
Komplikation in 2 Breslauer Fällen verzeichnet ohne nähere
e der Seite.
Typhus war in 3 Fällen vor 1 Jahr vorausgegangen. Ob
in einem ätiologischen Zusammenhang mit der letzten Erkran-
tehen könnte, ist nicht zu sagen, da nach dem Journal eine
uchung auf Typhusbazillen nicht vorgenommen ist. Möglich
nt mir die Annahme, daß vereinzelt auch der angeblich voraus-
ene Typhusanfall bereits einem verkannten Appendizitisanfall
chen haben kann.
herer erscheint ein solcher Zusammenhang mit einer Infektions-
eit schon im Fall 168, wo der Anfall in direktem Anschluß an
orausgegangene Masernerkrankung mit hohem Fieber als
Anfall überhaupt eingesetzt hatte. Sonst ist anamnestisch nur
n 2 Fällen ein Hinweis auf eine vorausgegangene Infektions-
eit in Gestalt einer Angina zu finden.
ähnt sei schließlich noch, daß in je 2 Breslauer Fällen „Leber-
rzen mit Ikterus", sowie Adnexbeschwerden angegeben
. —
ologisch sind äußere Veranlassungen für den Anfall nur sehr
nämlich in 5 Fällen angegeben. Dieselben bestanden 3mal
m Stoß gegen den Leib. In 1 dieser Fälle wurden die be-
n kleinen punktförmigen Hämorrhagien auf der Schleimhaut
pendix gefunden. Ob dieselben hier möglicherweise auf das
a zurückzuführen sind, möchte ich nicht entscheiden, halte es
icht für wahrscheinlich. In 2 weiteren Fällen war der Anfall
chwerer körperlicher Anstrengung bzw. nach schwerem Heben
agen aufgetreten. In 5 Breslauer Fällen sind Appendizitis in
milie erwähnt; in diesen Fällen war fast immer die Länge des
sus auffallend, die einmal sogar bis 17 cm betrug.

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Inzisionen zirkumskripter periappendizitischer
waren vorausgegangen in 17 Fällen = 5% der Gesamtzahl.

Im Fall 78 hatte dieselbe vor 1 Jahr stattgefunden und
Fistelbildung geheilt.

Im Fall 122 war vor 3 Jahren ein periappendizitische
inzidiert worden. Im Anschluß daran blieb eine Fistel zu
sich nach 9 Monaten spontan schloß. Es bildete sich dann
eine Narbenhernie aus, die bis zur Aufnahme bestand. Die
ergab im Bruchsack der Narbenhernie die Flexura hepatica
Appendix mit grünlich fibrinösen Massen bedeckt. Die
Spin. ant. sup. unter der Haut gelegene Tasche war mit ei
fibrinösen Exsudat gefüllt.

Im Fall 159 war zuerst eine Heilung der Inzisionswunde e
Später brach die Narbe wieder auf und entleerte eine Zeit
um schließlich spontan zu heilen.

Eine Fistel war zurückgeblieben in 5 Fällen. Im Fal
bei einem vor 35 Wochen vorausgegangenen Anfall eine
Inzision wegen multipler Abszeßbildung notwendig gewo
fand sich später eine alte Perforation an der Basis des
vermiformis, die wohl für die Fistel verantwortlich zu m
Im Fall 218 waren früher 2 Abszesse inzidiert worden, ob
zurückblieb ist nicht gesagt. Dagegen stellte sich auch b
Patienten im Laufe der weiteren Zeit eine Narbenherni
sämtlichen Fällen wurde die Fistel bzw. die Narbenhernie
Operation glatt beseitigt.]

Wenden wir uns nun dem intra operationem erhob
fund zu, so finden wir zunächst ältere bzw. schwerere Verw
in 175 Fällen = 50,6% angeführt. Leichtere Verwachsungen
standen in 127 Fällen = 36,7% der gesamten Fälle. Gar
wachsungen sind angegeben in 44 Fällen = 12,7%.

Was die Veränderungen am Appendix selbst anbela
eine Auftreibung bzw. Retention des Inhalts in 79 Fällen =
gegeben.

Ulzerationen ohne Perforation finden sich in 12 Fällen
Eine davon saß an der Spitze und befand sich kurz vo
foration. In 1 Falle fanden sich 2 und in 2 Fällen fand
unter die Serosa reichende Ulzeration. Man sieht auch hi
wie dringend bisweilen die Indikation für einen operativen Ei
kann, ohne besondere äußere Warnungszeichen.

Perforationen am Appendix ließen sich nachweisen in
= 12,7%. Bezüglich der Lokalisation derselben finden wir
Spitze, 5mal die Mitte und 11mal die Basis bzw. das unte

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ppendix angegeben. In 1 Fall zeigte nur noch eine binde-
ige Brücke von 6 cm Länge den Zusammenhang mit dem Frag-
des Processus an. In 3 Fällen war die Spitze mit dem Cöcum
ehsen und in dieses hinein perforiert. In 3 Fällen handelte es
uch um Perforationen des Darmes in der Nähe des Processus.
war bestanden in 1 Fall davon gleichzeitig 2 Perforationen am
arm und 1 am Cöcum, im zweiten Fall eine Perforation an
asis des Processus und eine am Cöcum, und im dritten Fall
den schließlich 2 Perforationen am Dünndarm, deren Charakter
ckperforationen im Krankenjournal in Erwägung gezogen wird.
eiche Entstehungsmöglichkeit dürfte auch für die Darmperfora-
der beiden anderen Fälle in Betracht zu ziehen sein. In den
n Fällen ist eine nähere Angabe nicht vorhanden.

e Verdickung der Wand des Processus wird hervorgehoben
Fällen = 21,4%. Kleine Hämorrhagien der Schleimhaut
rwähnt nur in 4 Fällen = 1,1%.

ine makroskopisch nachweisbare pathologische Ver-
ung am Processus fand sich in 12 Fällen = 3,5%, in denen
abgesehen von dem mikroskopischen Befund, der in den Jour-
nicht verzeichnet ist, die Operation als überflüssig erscheinen
. Immerhin ist dieser Prozentsatz von 3,5%, in dem die Ope-
vielleicht nicht nötig war, doch als ein geringer zu bezeichnen
rhältnis zu den Fällen, welche sämtlich die Operation dringend
rt erscheinen ließen. Interessant sind die von Dr. Capelle
mengestellten anamnestischen Angaben der 7 Breslauer Fälle
makroskopischen Befund, welche ich hier kurz einschieben
e. Der erste derselben hatte angeblich vor 9 Jahren einen
n leichten Anfall nach Trauma (Bauchaufschwung) erlebt und
wegen chronischer Obstipations- und Blasenbeschwerden zur
iven Behandlung. Bei dem zweiten sprachen die von vornherein
Beschwerden ohne eigentlichen Anfall für eine sog. Appen-
larvata. Bei dem dritten Fall war 1 Jahr zuvor 1 schwerer,
hentlicher Anfall vorausgegangen. In 3 anderen Fällen war
die klinische Diagnose unklar geblieben, einmal gegen Hysterie,
weite Mal gegen eine gynäkologische Affektion (Adnexe) und das
Mal gegen Dickdarmtuberkulose. Bei dieser letzten Patientin
seit 1 Jahre Durchfälle vorausgegangen und ziehende Schmerzen
Blinddarmgegend zurückgeblieben. Die Operation präsentierte
klinisch vorher festgestellten, hühnereigroßen, dem Cöcum auf-
den Tumor, der makroskopisch aus schwartigem Gewebe bestand
nit dem Netz verwachsen war. In seiner Nähe lag der Appendix.
r klarer Aszites im Peritoneum.

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Die mikroskopische Untersuchung des schwartigen Gewebes exzision) ergab keinen für Tuberkulose charakteristischen Trotzdem blieb auch nach der Epikrise der Fall unklar und darmtuberkulose suspekt.

Kotsteine fanden sich insgesamt in 37 Fällen = 10,7%. waren in 8 Fällen mehrere Steine gleichzeitig vorhanden u 2 Steine in 4 Fällen, 3 Steine in 1 Fall und 4 Steine in 4 F 1 Fall waren die Steine deutlich facettiert. Die Größe de wird zwischen erbsengroß bis bohnen- und mandelgroß, in haselnußgroß angegeben. In 1 Fall mit 4 Kotsteinen im P hatten dieselben sämtlich Mandelgröße. Es gehört schon ei starke Dilatation des Appendix dazu, um 4 derartige Steine beh zu können. Die Möglichkeit einer Druckwirkung und Ulzer Wand ergibt sich in solchen Fällen wohl von selbst. Über d lisation der Steine ist angegeben, daß in 6 Fällen dieselben a des perforierten Appendix lagen. Darunter war 1 Fall mit 2 die beide außerhalb des Appendix gefunden wurden. Der i des Processus gelegene Stein saß in 3 Fällen über der Basis, in der Mitte und in 10 Fällen im distalen Ende bzw. in d des Processus. In den übrigen Fällen ist die Lokalisation d nicht näher angegeben. Auch hiernach scheint entsprechend sammenstellungen anderer Autoren die Spitze und danach die Prädilektionsstelle für die Lokalisation der Kotsteine im A zu sein.

Der Inhalt des Processus wird, abgesehen von Steine geben als kotig in 11 Fällen, als schleimig-kotig in 10 Fällen mig in 20 Fällen, blutig-schleimig in 5 Fällen, eitrig in 22, s eitrig in 3 Fällen und kotig-eitrig in 1 Fall. In 1 Fall fan lebende Oxyuren im Processus. Man ersieht daraus ohne entsprechend dem chronischen Stadium der Fälle den vo schleimigen Charakter des Inhalts bei 35 Fällen, während 25 Fällen der Inhalt eine mehr eitrige Beimengung zeigte. Ei körper im Appendix fand sich abgesehen von dem Fall mit nur in 1 Fall in Gestalt zweier Haare, daneben war dünner handen. In 2 Fällen wird angegeben, daß kein Inhalt gefu Strikturen des Processus werden angegeben in 72 Fällen lisiert waren dieselben an der Basis 28mal, an der Spitze 2 der Mitte 14mal. In 5 Fällen ist eine Angabe über die La enthalten. In 1 Falle bestanden 2 Strikturen gleichzeitig, v eine an der Basis und die andere an der Spitze saß. Über logie derselben ist nur in 5 Fällen angegeben, daß sie von herrühren. Bei den übrigen muß es unentschieden bleiben,

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narbige Veränderungen von innen heraus, wie viele anderer-
durch adhäsive Abschnürungen und Knickungen von außen her
Ursprung genommen haben.

e Obliteration des Lumens war vorhanden in 50 Fällen
% der Fälle. Entsprechend der Lage der Strikturen saß sie
lls am häufigsten an der Basis und Spitze, nämlich in 21 bzw.
len. In der Mitte war sie am seltensten, nämlich 9mal lokali-
In 5 Fällen war das Lumen des gesamten Appendix in toto
iert. Eine besondere Veranlassung für diese ausgedehnte Ob-
on des Lumen ist aus den Krankengeschichten nicht zu er-

Lage des Appendix ist angegeben in 124 Fällen. Richten wir
ach den in den Krankengeschichten gewählten Bezeichnungen,
der Appendix nach innen oben 22mal, nach innen unten 29mal,
nnen 4mal, nach innen hinten 3mal, nach hinten 13mal, nach
oben 31mal, nach hinten unten 44mal, nach hinten außen 7mal,
orne oben 3mal, nach vorne unten 8mal, nach unten außen
nach oben außen 2mal, nach oben 6mal. In 3 Fällen war der
sus sehr hoch (oberhalb der Nabellinie) gelegen. Danach ist
sere Fälle am häufigsten die Lage des Processus nach hinten
(44), nach hinten oben (31), dann nach innen unten (29), und
oben (22). Die übrigen Richtungen werden nur in ganz ge-
und ziemlich gleichbleibender Zahl (2—13) vom Processus ein-
nen.

Haltung des Processus wird beeinflußt durch seine Länge,
Angaben in unseren Fällen zwischen $2^1/_2$—16 cm schwanken,
die Größe des Mesenteriolums und vornehmlich durch voraus-
ene, ihn fixierende Verwachsungen, als Folge früherer Ent-
gszustände. Diese begünstigen naturgemäß die Entstehung von
ngen und Retentionen und letztere führen dann im Circulus
s wieder zu neuen Entzündungsnachschüben. Genauere An-
über die Haltung des Appendix sind enthalten in 74 Fällen.
er lag eine Knickung an der Spitze in 16 Fällen, in der Mitte
und an der Basis in 5 Fällen vor. In 16 weiteren Fällen ist
nauere Lage der Knickungsstelle nicht näher angegeben. In
en war der Processus zusammengeknäult bzw. aufgerollt, in
en schneckenförmig gekrümmt. In 6 Fällen wies der Pro-
eine S-förmige Krümmung um seine Achse auf, in 7 weiteren
wird er als spiralig gewunden bzw. geschlängelt bezeichnet. In
n war er an der Knickungsstelle in seiner Kontinuität nur noch
eine 6 cm lange bindegewebige Brücke verbunden. In 1 Falle
die Haltung als „posthornförmig" bezeichnet. Eine doppelte

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Knickung lag in 4 Fällen vor, derart daß der Processus in sei der Form eines Dreiecks entsprach. In sämtlichen Fällen Processus in seiner Lage durch mehr weniger zahlreiche Ad fixiert und wies mehrfach deutliche Retentionserscheinungen

Größere Exsudatreste bzw. kleine, ältere Abszes wurden in 57 Fällen gefunden = 16,5%. Die Größe der Absz schwankte zwischen Linsen- bis Pflaumengröße. In einem der Processus in einen faustgroßen Tumor von Exsudatrest bettet. In einem anderen Fall fand sich eine Tuberkulose de Adnexe, sowie reichliche Verwachsungen der verdickten Tube Cöcum. Die Adnexe wurde ebenfalls exstirpiert. Der l selbst wies nur geringfügige Verwachsungen und sonst kein skopisch erkennbaren Veränderungen auf. Nach dem Befun es sich in dem Falle wohl primär um eine rechtsseitige Ad kulose gehandelt haben, die erst sekundär zu einer Affek Processus geführt hat. Die Lage der Exsudatreste befand Becken 4mal, am Cöcum 13mal, darunter 2mal retrocöcal, pendix selbst 24mal, darunter eine zweite Abszeßhöhle an de säule; zwischen herangezogenen verklebten Dünndarmschling am Mesenteriolum 4mal, in dem einen Fall mit kleinen Fet verbunden. Letztere dürften vielleicht als Folge thrombotis fäßverschlüsse aufzufassen sein. In 19 Fällen ist über Lo der vorhandenen Exsudatreste nichts Näheres angegeben. der Breslauer Fälle, bei denen die bakteriologische Untersu Eiterreste regelmäßig stattgefunden hat, bemerkt Dr. Cap diese Reste keineswegs immer steril gefunden wurden. So in einem Falle Kolibazillen, in einem anderen Falle sogar kokken gezüchtet werden, trotzdem dieser letzte Fall erst na 3 Monate langen anfallsfreien Zeitraum zur Operation kam. 12 Fälle der Breslauer Klinik berechnet würden diese 2 Fä Prozentsatz von 16,6% entsprechen, der jedoch bei der Zahl der Fälle einen besonderen Wert wohl kaum bea dürfte.

Der Fall 124 allein verdient es wegen seines eigenartigen näher angeführt zu werden. Von vornherein lag in dem Fal der Verdacht auf einen Tumor vor. Der Patient klagte Schmerzen in der rechten Leistenbeuge beim Gehen. Pa konnte man einen überhühnereigroßen, steinharten, beweglich über dem Lig. Poupartii in den Bauchdecken konstatieren. über demselben war verschieblich. Der Musc. rectus dr Kontraktionen den Tumor nach außen. Die Operation ergab kulatur über dem Tumor sulzig verändert. Der Darm saß

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ansversa und Peritoneum, war der ersteren fest adhärent und
m Peritoneum nicht abzulösen. Er entsprach dem von oben
Tumor ziehenden Appendix. Die Hauptmasse des Tumors
aus außerordentlich derbem Sehnengewebe, an welchem ein
Darmwand fest angewachsen war. Die Darmschleimhaut ließ
einen Kanal verfolgen, der anscheinend dem Appendix ent-
In der Nähe der Spitze bestand im Bindegewebe ein fistulöser
der in eine walnußgroße buchtige Höhle überging. Der Tumor
zusammen mit dem Appendix entfernt. Trotz der Tamponade
n später noch ein Bauchdeckenabszeß auf, dem völlige Heilung
te. Nach diesem Befunde könnte es sich hier um eine pri-
roperitoneale Hernie des Appendix in der Bauchwand gehan-
en, bei der es erst sekundär zur entzündlichen Veränderung
pendix und seiner Umgebung gekommen ist. Wenigsteps
mir diese Annahme wahrscheinlicher, als der Vorgang einer
n Periappendizitis mit Verlötung des Processus am Peritoneum
chwand und allmählicher Perforation des Peritoneum parietale
e der Abszedierung. Wie es dann nachträglich zur properi-
Verlagerung des entzündeten und mit höchster Wahrschein-
seiner Umgebung adhärenten Appendix gekommen sein könnte,
nicht recht erfindlich.

Verlauf der Fälle nach der Operation war in 266 Fällen
% ohne jede Störung und ohne Fieber. Eine Dränage der
war in 23 Fällen notwendig und zwar in 15 Fällen wegen
ebliebener Abszesse und Exsudatreste, in 3 Fällen wegen Ein-
des brüchigen Appendix während der Auslösung desselben
Verwachsungen.

Einnähung der Abszeßwand in die Bauchwunde wurde
em intramural am Cöcum gelegenen Abszeß gemacht.

Eröffnung der alten Narbe mit Tamponade wurde nur 1mal
Tage nach der Operation zur Ableitung eines retrocöcal ge-
Abszesses notwendig.

Sekundärnaht mit Anfrischung der Wundränder konnte
Tagen im Fall 122, in dem es sich um eine Narbenhernie mit
lix im Bruchsack handelte, gemacht werden. Der Fall heilte
er primam.

rungen an der Wunde selbst traten 18mal auf. Es fand sich
deneiterung in 8 Fällen, ein Hämatom der Bauchdecken in
n, ein Bauchdeckenabszeß in 8 Fällen. In dem einen der letzten
Fälle handelte es sich um einen Exsudatrest an der Basis des
dix, in dem anderen Fall um einen Appendix, der mit 4 Steinen
ter gefüllt war. Außerdem ist 1 Fall hervorzuheben, bei dem

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sich post op. ein tiefer Abszeß bildete, der spontan in die B
brach und dann zur glatten Heilung kam.

Störungen seitens der Lungen traten in 16 Fäll
ein. Es fand sich eine Lungenembolie leichteren Grades
Pleuritis exsudativa dextra 2mal (darunter 1mal infolge alt
tuberkulose), leichte Bronchitis purulenta 4mal, eine Pne
rechten Seite 7mal, zum Teil sehr leicht. Wieweit bei d
komplikationen die Narkose, die fast stets mit Äther ausgef
beteiligt sein kann, läßt sich aus den Krankenblättern nich
den. In der Regel handelt es sich dabei um eine von der
aus aufsteigende Infektion durch die Lymphgefäße des Zwer
durch, seltener durch kleine Gefäßembolien, als deren Vo
eine Infektion der Lymphgefäße in der Umgebung des Pr
zunehmen ist. Wo diese jedoch bei dem Fehlen jegliche
reste nicht als wahrscheinlich anzunehmen ist, muß man w
Beteiligung der Narkose dabei durch Infektion der Lung
Aspirationswege denken.

Eine Thrombophlebitis der Vena femoralis fand
samt in 9 Fällen = 2,6%. Dieselbe war entsprechend den
Erfahrungen in der Mehrzahl der Fälle, nämlich 6mal auf
Seite gelegen. Der eine Fall hatte bereits vor 4 Jahren
eines Anfalles eine Thrombose der Femoralis rechts du
Der Fall ist bereits oben bei der Besprechung der Anam
erwähnt worden. Die Behandlungsdauer wurde durch
plikation nur in 1 Falle auf 83 Tage verlängert, die üb
gelangten trotzdem in 24—28 Tagen zur Heilung. Hinsi
Ätiologie bieten sämtliche Fälle nichts Besonderes dar,
der Umstand, daß in sämtlichen Fällen der Schnitt am äu
tusrand angelegt ist. Dieses ist von besonderem Interes
Erklärung der Genese gerade der linksseitig gelegenen Tro
Vena femoralis, die von Witzel angegeben ist. Danach
gleichzeitiger Ligatur der Art. und Vena epigastrica zu einer
in den Wurzeln der Ven. epigastrica kommen, deren Forts
der linken Bauchseite auf dem Wege der Ven. epigastri
zur Thrombose der Ven. femoralis sinistra führen kann.
der Hand, daß der Schnitt am äußeren Rektusrand leich
derartigen Vorgang die Veranlassung abgeben kann, insofe
greifenden Hautnähten zufällig einmal die Ven. und Art.
gleichzeitig mitgefaßt und ligiert werden kann. Von selte
plikationen sind schließlich noch aus der Breslauer Klinik
tive Stenosenerscheinungen in der Cöcalgegend zu nennen
beobachtet wurden und nach 3—4 Tagen spontan verschwan

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mal ein intermittierender Ikterus auf, der wohl auf katarrhali-
Grundlage zustande gekommen ist.

eressant dürfte zum Schluß noch neben einer näheren Erörterung
tlich verlaufenen Fälle die Fieberkurve eines Patienten aus der
uer Klinik sein, der im Anfall zur Aufnahme gelangte. Er hatte
eriappendizitisches Exsudat mit Fiebertemperatur bis 39° und
der Temperatur entsprechend beschleunigten Puls. Plötzlich
Temperatur und Puls bei dem Patienten einen steilen Ab-
e Temperatur blieb einige Tage unter 36°, der Puls unter 35
en pro Minute, ohne daß Anzeichen vorhanden waren, die für
erforation eines Abszesses in die Blase oder Mastdarm ge-
en hätten. Der Fall ist dann komplikationslos in das Sicher-
adium gekommen.

hen wir schließlich noch auf die beiden tödlich verlaufenen
twas näher ein, so handelte es sich bei dem ersten derselben
r Königsberger Klinik um einen 22jährigen Patienten, der bereits
lle durchgemacht hatte und 10 Wochen nach dem letzten Anfall
peration gelangte. Klinisch wurde bei dem Patienten eine
eigroße Resistenz in der Ileocöcalgegend festgestellt. Die Ope-
ergab folgenden Befund: Das Cöcum war in reichlichen, alten
chsungen der Bauchwand adhärent, bei Lösung derselben wurde
trige Lymphdrüse eröffnet. Der Appendix lag nach hinten und
vom Cöcum, seine Spitze war in eine Granulationshöhle der
am Cöcum eingebettet. Nach Entfernung des Appendix und
tzung der Höhle wurde die Operation durch Tamponade der
beendet. Der Patient starb an einer allgemeinen Peritonitis,
hl ihren Ausgang von der eröffneten eitrigen Lymphdrüse ge-
en haben dürfte.

dem zweiten Todesfall, der aus der Breslauer Klinik stammt,
te es sich um einen 22jährigen Arbeiter, der im 6wöchentlichen
ll nach dem ersten schweren Anfall operiert wurde. In ope-
, die durch einen zu klein angelegten pararektalen Schnitt er-
t wurde, lagen Dünndarmschlingen mit starken Adhäsionen
le nach unten und lateral teilweise an das Peritoneum parietale
ogen. Die Lösung dieser Adhäsionen machte große Schwie-
en, ebenso die Entwicklung des Appendix, der 10 cm lang, nach
lateral umbog und mit der Spitze fest fixiert war. Bei der
g der Adhäsionen wurde eine Dünndarmschlinge angerissen,
ision ging bis auf die Mukosa und wurde übernäht. Der Patient
2 Tage nach der Operation an einer Allgemeininfektion des
neums zugrunde. Epikritisch wurde angenommen, daß bei der
g der Verwachsungen höchstwahrscheinlich eine noch infektlöse

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Eiterhöhle eröffnet sei, und daß diese die tödliche veranlaßt habe. Abgesehen davon stellte die Autopsie Unterlappenpneumonie fest.

Wenden wir uns nun den Fällen zu, welche im Stadiu kumskripten Periappendizitis bzw. der abgekapse szeßbildung zur Operation gelangt sind, so verteilen sich auf 69 männliche und 43 weibliche Patienten = 61,6% : 38, 63,2% : 36,4% bei den chronischen Fällen.

Die Fälle verteilen sich auf die einzelnen Altersdezenni dermaßen: 1.—10. Jahre 11 Fälle, 11.—20: 24, 21.—30.: 29 22, 41.—50.: 13, 51.—60.: 9, 61.—70.: 3 und 71.—80. Ja Auch hier halten sich die Zahlen vom 2.—4. Dezenniu gleichmäßig, um dann steil wieder abzufallen.

Die Zahl der vorausgegangenen Anfälle bewegt sic 1—6 Anfällen von verschiedener, meist als sehr heftig ge Intensität der Erscheinungen und zwar ist vorausgegangen fall in 93 Fällen, 2 in 12, 3 in 4, 4 in 1, 5 in 1 und 6 ebenfalls 1 Fall. Es fällt ohne weiteres ins Auge die wiegende Zahl von 93 Fällen, bei denen 1 Anfall von solch vorausgegangen war, daß im Gefolge desselben der ope griff in Gestalt einer Spätoperation notwendig wurde. dieses mit der mehrfach beobachteten Erfahrungstatsach daß gerade die ersten Appendizitisanfälle vielfach die sch sein pflegen.

Der Processus wurde entfernt nur in 22 Fällen = wurde zurückgelassen in 90 Fällen = 80,2%, entsprechend mein üblichen und anerkannten Grundsatz, den Processus zu entfernen, wenn sich dieses ohne längeres Suchen und ol schützender Adhäsionen ermöglichen läßt.

Bezüglich des Zeitpunktes der Operation empfeh hier von vornherein einen Unterschied zu machen zwischen die wir nach der heutigen Auffassung als Intermediäroper zeichnen müssen, und den Spätoperationen im eigentlichen Intermediäroperation bezeichnet man bekanntlich die Op demjenigen Stadium, welches nach Richardson für die Frü zu spät und für die Spätoperation zu früh ist. Dieses „Z stadium", wie wir es nennen wollen, liegt also zeitlich zw Früh- und Spätoperation der Periappendizitis.

Anatomisch läßt sich der Begriff des Zwischenstadiums grenzen als Zeitraum vom Beginn der Miterkrankung des P bis zum Abschluß des Entzündungsvorganges gegen das toneum. Zeitlich läßt sich naturgemäß dieser Zeitraum nich

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nzen, da namentlich für den Endtermin desselben zu viel ver-
ene und wechselnde Faktoren in Betracht kommen, wie Virulenz
fektionserreger bzw. Intensität des Entzündungsvorganges, Wider-
fähigkeit des Peritoneums, Verhalten des Patienten und auch
rztes in diesem Stadium u. a. m. Dementsprechend wird auch
erschiedenen Autoren die Länge des Zwischenstadiums ver-
en berechnet. Der Beginn wird allgemein vom 3. Tage der
kung ab gerechnet. Der Endtermin schwankt zwischen dem
Tage der Erkrankung. Sprengel schlägt auf Grund seiner um-
len Erfahrungen vor, das Zwischenstadium nur bis zum Beginn
Tages zu rechnen. Andere Autoren rechnen bei ihren Aufstel-
mit dem 8.—10. Tage. Dieser Zeitraum vom 6.—10. Tage ist
nit Sicherheit als die Grenze des Zwischenstadiums gegen das
dium zu bezeichnen. An welchem Tage innerhalb dieser Zeit
ufigsten ein vollständiger Abschluß des peritonealen Entzündungs-
ges stattzufinden pflegt, läßt sich nach den bisherigen Angaben
nicht sicher entscheiden. Wir werden daher am besten als
des fraglichen Zeitraumes, den 8. Tag für unsere Betrachtungen
dtermin des Zwischenstadiums zu wählen haben.

präzise Resultate zu erhalten verlangt Sprengel, daß man
lie Gesamtzahl der Fälle, sondern nur die gesonderten Resultate
gen Fälle heranziehen müsse, welche im Zwischenstadium bei
oder umschriebener (aber noch nicht abgeschlossener) Mitbe-
ng des Peritoneums zur Operation gelangt sind. Sprengel hat
diesem Prinzip 102 vom 3.—5. Tag operierte Fälle eingeteilt.
ter befinden sich 38 Fälle mit freier Peritonitis und 18 Todes-
und 64 Fälle mit umschriebener Beteiligung des Peritoneums
Todesfällen. Er hat danach im Gegensatz zu anderen Autoren
ßerst niedrige Mortalitätsziffer von 3% für die Zwischenopera-
erechnen können.

e Gegner dieser Anschauung, wie Körte, Kümmel, Garrè,
elsberg, Sonnenburg u. a. führen gegen dieselbe eine er-
Infektionsgefahr des Peritoneums bei operativem Vorgehen in
n Stadium an und stützen sich dabei auf die von den meisten
chtete erheblich höhere Mortalität 8—16% nach operativem Ein-
Sie schlagen vielmehr ein individualisierendes Vorgehen von
n Fall vor, indem sie sich nur auf mehr weniger dringende
tionen bzw. deutliche Anzeichen einer akuten Verschlimmerung
diesem Stadium zum operativen Eingriff entschließen. In den
n Fällen halten sie es für zweckmäßiger und sicherer, die Pa-
n erst in das Spätstadium überzuführen, da ihnen dieses für den
ndigen operativen Eingriff geeigneter erscheint.

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Hierbei ist jedoch der Umstand in Betracht zu ziehen, dieser Voraussetzung nur die schweren, auf dringende Ind hin operierten Fälle für die Resultate im Zwischenstadium in kommen. Es liegt auf der Hand, daß danach die Resultate sein müssen, als bei denjenigen Autoren, die ohne Auswahl Einschluß der leichteren Fälle im Zwischenstadium operier Beweiskraft für die Entscheidung der prinzipiellen Ope Zwischenstadium kann man somit diesen Zusammenstellun unterlegen. Immerhin wird auch hier eine Gegenüberste Zwischenoperationen und Spätoperationen bzw. der Heilerf allgemeinen Interesses nicht entbehren.

Von den gesamten 112 Fällen unserer Zusammenstell 48 Stunden sind von dem 3.—8. Tage seit Beginn des An Operation gelangt 24 Fälle. Von diesen sind 2 Fälle gestor denen nach der Anamnese der eine am 6. Tage, der a 4. Tage operiert wurde. Im ersten Falle wurde wegen sudatansammlung im Douglas die Inzision und Dränage p gemacht. Später wurde die Spaltung einer Bauchdeckenp und verschiedener metastatischer Abszesse der Haut notwe Eiter fanden sich Staphylokokken und Stäbchen. Der Pa an multiplen Abszessen zugrunde. Inwieweit man durch e zeitiges Eingehen von oben her den Prozeß hätte be können, ist nachträglich kaum zu entscheiden. Der zwei verlaufene Fall zeigte bei der Aufnahme eine deutliche D dominalis, leicht gespanntes Abdomen, sowie eine große in der rechten Beckenseite. Die Temperatur betrug 38°, 128 Schläge in der Minute. Die Operation ergab vorliegen verklebte Netzpartien, sowie von oben herkommenden s Eiter an der Außenseite des Cöcums. Der Abszeßherd wu gefunden. Der Patient starb am 4. Tage an fortschreiten tonitis. Ein Obduktionsbefund ist nicht vorhanden. Hierna auf unsere 24 Zwischenoperationen eine Mortalität von 8 fallen.

Ausgenommen bzw. nicht berechnet sind hierbei die Fäl mit einer entzündlichen Beteiligung des gesamten Periton Operation gelangten, und die auch nach der Auffassung von für die Beurteilung der Zwischenoperation fortfallen müsse diese die Prognose nach 48 Stunden heute kaum noch als z gelten kann. Dieselben sind später unter einer besonderen zusammengefaßt. Es kommen hier auf 17 Fälle, die vom 3 nach der Perforation operiert sind, tatsächlich auch nur 2 fälle, was einer Mortalität von 82,2% entspricht.

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n den übrigen 89 Fällen nach 48 Stunden wurden operiert in Woche 21 Fälle, in der 3. Woche 24, 4. Woche 13, 5. Woche 12, che 5, 8. Woche 3, 9. Woche 1, 10. Woche 3, 11. Woche und 7 Fälle.

dieselben entfallen im ganzen 7 Todesfälle, was einer Mortali- 7,9% entsprechen würde. Wir finden also bei dieser kleinen von Fällen ein Fallen der Mortalität um nur 0,4% gegenüber erativen Eingriffen im Zwischenstadium, ein Umstand, der zum ten die Entscheidung der Frage nicht gegen die Zwischen- on beeinflussen dürfte.

Behandlungsdauer der gesamten 113 Fälle schwankt zwi- 6—116 Tagen. Dieselben verteilen sich folgendermaßen: Die der Behandlung betrug 1—10 Tage in 4 Fällen, 11—20 Tage 12—30 in 25, 31—40 in 26, 41—50 in 16, 51—60 in 9, 61—70 71—80 in 2, 81—90 in 3, 91—100 in 2, 101—110 in 2 und 20 Tage in 3 Fällen.

ach liegt für die Mehrzahl der Fälle die Behandlungsdauer en 11—70 Tagen, und nur bei 12 Fällen erstreckt sich die Be- ng über 70 Tage hinaus. Bei 2 von den Fällen mit einer Be- ngsdauer unter 10 Tagen handelte es sich in dem einen um ktale Punktion, die einige Tropfen Eiter lieferte, während die gende Inzision ein negatives Resultat ergab. Nach afebrilem Ver- t Heilung ein. Bei den anderen Fällen trat innerhalb der ersten der Exitus an sekundärer, diffuser, fibrinös-eitriger Peritonitis ein. den übrigen Fällen wurde vielfach erst längere Zeit nach der me operiert, bis ein Abklingen der akuten Erscheinungen zu ieren war. Daraus erklärt sich die lange Behandlungsdauer reffenden Fälle.

üglich der Ätiologie sind Angaben nur in 9 Fällen vorhanden. illen wird ein Diätfehler bzw. starkes Trinken, im 3. und 4. nwere körperliche Arbeit als Ursache des Anfalls beschuldigt. all hatte der Patient bei einem Fall mit dem Revolver, den er bei ug, einen Stoß gegen den Leib erhalten. 10 Tage später stellten e ersten Beschwerden ein. Im 6. Falle hatte Patient vor 4 Wochen ereiterte Verletzung an der Sohle des rechten Fußes gehabt. Wochen, also 8 Tage später, hatten die ersten Symptome der kung eingesetzt. Inwieweit im letzten Fall ein tatsächlicher Zu- nhang beider Erkrankungen besteht, ist mit Sicherheit nicht tellen. Immerhin scheint hier eine solche Annahme nach dem post hoc ergo propter hoc nicht unberechtigt. Ferner werden Abführmittel und 1mal eine vorausgegangene doppelseitige Par- ls Ursache bzw. als verschlimmerndes Moment erwähnt.

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Von den anamnestischen Angaben der einzelnen Fäl folgendes von Interesse sein.

Spontanperforationen waren dem operativen Eingriff gegangen insgesamt in 8 Fällen. Davon waren per rectum forationen erfolgt: im Fall 6 vor 3 Jahren und im Fall 73 8 der Aufnahme ohne wesentliche Besserung der Erscheinung forationen in die Blase hatten in 2 Fällen stattgefunden. Fa vor 3 Jahren eine Rektumperforation durchgemacht hatte, 5 Tage vor der Operation plötzlich reichliche Eitermengen Urin. Dieselbe Erscheinung zeigte auch Fall 44 am 2. Tage Aufnahme. Im letzteren Fall war die vordere Vaginalwand stark geschwollen und empfindlich. Im zystoskopischen Bilde an der vorderen Blasenwand rechts von der Mittellinie ein ru pfennigstückgroßes Geschwür von bläulich-roter Farbe, ans geschlossen. Merkwürdigerweise wird gerade in diesem Fa Anamnese ausdrücklich angegeben, daß keine Blasenbes vorher bestanden.

Schließlich war noch eine Perforation in die Vagina 1ma die Bauchwand in 2 Fällen vorausgegangen. Im Fall 20 h solche am Nabel vor 3 Tagen stattgefunden, nachdem be 3 Monaten Schmerzen um den Nabel herum bestanden hatt der Perforationsstelle in der Haut entleerte sich ziemlich reichl riechender Eiter, welcher seine Herkunft leicht erraten li Operation ergab in der Abszeßhöhle einen freien Kotstein. ausgegangene Perforationsstellen in der Bauchwand, deren sich anamnestisch nicht bestimmen ließ, wurden in Fall Inzision rechts von der Linea alba freigelegt. Dieselben führt in eine hühnereigroße zwischen den Därmen gelegene Abs Unter fieberhaftem Verlauf wurde dann später noch eine z zision in der Mittellinie notwendig, worauf die weitere He Falles ohne Störung verlief.

Das prozentuale Verhältnis der vorausgegangenen Perf auf die Gesamtzahl der 113 Fälle berechnet sich danach auf

Urinbeschwerden bestanden in 11 Fällen = 9,7 %. I waren in 4 Fällen durch einen vorhandenen Douglasabszeß Im Fall 46 reichte die große Abszeßhöhle bis zur Symph Fall 48 lag ein Abszeß vor der Blase dicht über der Symph Fall 54 reichte die sehr große Eiterhöhle hinter der Blas linken Inguinalgegend hinüber und nach unten bis an das heran. Im Fall 57 und 59 ist nur von einem großen Ab rechten Beckenschaufel die Rede, dessen Grenzen nicht näh geben werden. Im Fall 66 war auch nach der Eröffnung de

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ein Infiltrat überhaupt nicht zu finden. Der Fall gelangte im 897/98 zur Operation. Man suchte damals, um eine weitere onsmöglichkeit des Peritoneums auszuschließen, nicht weiter lem Appendix, und es trat dann nach einem Abfall der vorher aften Temperatur eine glatte Heilung ein. Ob es sich in diesem berhaupt um eine Periappendizitis gehandelt hat, dürfte danach veifelhaft erscheinen, wenigstens bietet der angegebene Operafund keine sicheren Anhaltspunkte für diese Annahme dar. ndlichen Fällen erklären sich auch hier die Beschwerden durch bergreifen des Entzündungsprozesses auf die Blase, soweit ein r überhaupt nachzuweisen war.

hmerzen in der rechten Brustseite mit Atmungsstörung hem Fieber, also eine rechtsseitige pleuritische Affektion war ll 8 eingetreten, nachdem bereits 4 Wochen vorher in der gegend und im Leibe rechts wechselnde Schmerzen bestanden Die Operation ergab in dem Fall gleichzeitig einen subschen Abszeß rechts, von dem augenscheinlich die Lungenn ihren Ausgang genommen hatte. Der Fall entspricht 0,9% esamtzahl.

rchfälle abwechselnd mit starker Obstipation waren in 22 = 19,5% vorausgegangen. In 1 Falle bestand seit Beginn der kung ein Ausfluß aus der Harnröhre, über dessen mikroskon Charakter nichts Näheres angegeben ist. Es handelt sich in all um einen Douglasabszeß, der durch eine Mitbeteiligung der ta vielleicht die Veranlassung dazu gegeben hat. Urinbeschwerie an eine gleichzeitige Gonorrhöe denken lassen könnten, bein in dem Falle nicht.

3 Fällen soll das vorausgegangene Erbrechen schließlich einen enten Charakter angenommen haben, wohl als Folge einer ung des Darmlumens durch den Druck der Exsudatmenge.

beweglichkeit bzw. Flexionskontraktur des rechten Beines d in 7 Fällen. Dieselben werden im Zusammenhang mit den erativen Komplikationen näher besprochen werden.

enden wir uns nun der klinischen und anatomischen Betung unserer Fälle zu, so empfiehlt es sich wohl, zunächst die orbenen Fälle im Zusammenhang zu besprechen.

n diesen können wir zunächst Fall 9 ausscheiden, der an einer ntellen Todesursache zugrunde ging. Es handelte sich dabei ine ziemlich frische Exsudatbildung in der Höhe der rechten ant. sup. mit geringem Fieber, die ungefähr die Größe eines hatte. Der adhärente entzündlich veränderte Appendix war in terhöhle eingebettet. Der Heilungsverlauf war völlig reaktionslos.

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Der betreffende Patient war jedoch in seinem Beruf als
starker Potator gewesen und ging infolgedessen am 6. Tage
an einer akuten Herzschwäche elend zugrunde; hinzufügen m
obwohl die Narkose nicht mit reinem Chloroform, sondern mi
scher Mischung gemacht worden war und nicht übermäßig
dauert hatte. Der Fall kommt somit für die Mortalität
Statistik eigentlich in Fortfall und ist eher unter die Kate
Spätchloroformtodesfälle zu rechnen.

Gleichzeitig ausscheiden müßte auch der Fall 84, bei
fraglich erscheint, ob eine Periappendizitis überhaupt mit
gewesen ist. Der Fall bot klinisch durchaus das Bild eine
Erkrankung dar mit einem gänseeigroßen fluktuierenden Ex
der rechten Beckenschaufel, fieberhafter Temperatur und ein
zeitigen Flexionskontraktur des rechten Oberschenkels. Da
war bis unterhalb des Lig. Poupartii nachweisbar, weshalb
zunächst die Inzision vorgenommen wurde. Dieselbe entlee
lich dicken, kotigen Eiter aus einer von dicken Schwarten u
Höhle oberhalb des Lig. Poupartii. Von dieser aus hatte ein
längs des rechten Schenkelkanals stattgefunden, so daß die
gefäße freigelegt waren. Infolge weiterer Eitersenkung
Schenkelgefäße wurde dann noch eine Inzision am Obersch
wendig. Der Fall ging am 10. Tage post. op. an zunehmen
schwäche zugrunde. Die Sektion ergab ein Karzinom des
Da auch hierbei der Appendix nicht gefunden wurde, läßt s
für noch gegen das gleichzeitige Vorhandensein einer Ap
eine bestimmte Entscheidung treffen. Als primäre Ursache d
heitsvorgänge ist hier jedoch in jedem Fall wohl das b
Cöcumkarzinom anzusehen, das ja möglicherweise sekundär
Affektion des Appendix geführt haben könnte.

War in diesem Falle das bestehende Karzinom übersehe
so ging umgekehrt der Fall 49 an den Folgen einer irrtü
Karzinom gestellten Diagnose zugrunde. Die klinische Unt
ergab in der Lumbalgegend eine bewegliche Dämpfung
deutlich fühlbare Resistenz. Per rectum fühlte man einen
kleine Becken einnehmenden, teigigen Tumor, der nicht
deutet werden konnte. Die Laparotomie in der Lin. alba
freie, blutigseröse Flüssigkeit. In der Ileocöcalgegend fand
von fest verklebten Darmschlingen umgebener Tumor, de
Karzinom angesprochen wurde. Im Mesenterium mehrere hart
die scheinbar diese Diagnose bestätigen mußten. Da ein
anastomose unmöglich war, wurde ein Anus praeternaturalis
10 Tage später trat unter fieberfreiem Verlauf der Exi

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inungen von Herzschwäche ein. Die Sektion ergab einen alten
ppendizitischen Abszeß ohne Perforation des Appendix und
kundäre, diffuse fibrinös-eitrige Peritonitis, die ihren Ausgang
m alten Abszeß genommen hatte. Eine richtige Erkennung des
ionsstatus bzw. eine rechtzeitige Entleerung des Abszesses hätte
ohl den bedauerlichen Ausgang verhindern können.
den übrigen 6 Todesfällen bieten 4 das typische Bild mul-
metastatischer Abszesse dar, während 2 an fortschreiten-
ritonitis zugrunde gingen.
Fall 5 war bei dem 32jährigen Pat. zunächst ein Douglas-
per rectum inzidiert und dräniert worden. 10 Tage später
die Spaltung eines Abszesses per abdomen notwendig. Im
n Verlauf wurde noch die Spaltung einer Bauchdeckenphlegmone
rschiedener Hautabszesse gemacht. Im Eiter wurden Staphylo-
und Stäbchen nachgewiesen. Der Pat. erlag schließlich nach
er mühe- und qualvoller Behandlung der fortschreitenden In-
Eine Sektion wurde nicht gestattet.
Fall 22 wurden zunächst per abdomen 3 Abszesse eröffnet.
ppendix war dicht unter der Spitze perforiert. Er wurde am-
und enthielt in seinem Innern einen Kotstein. Trotz des bis
inge Temperaturen fieberfreien Verlaufs wurde weiter die In-
eines periumbilicalen, dann eines retroperitonealen und schließ-
ch zweier intraperitonealer Abszesse notwendig. Der Pat.
ach 116tägiger Behandlung. Die Sektion ergab einen weiteren
links oberhalb des Zwerchfells und multiple Leberabszesse
diffuser Bronchitis.
Fall 41 wurde durch Schrägschnitt eine mit gashaltigem, stin-
Eiter gefüllte Höhle auf der rechten Beckenschaufel eröffnet.
ppendix ragte in die Höhle hinein und wurde amputiert. Von
ten erfolgte die Eröffnung einer zweiten nach links hinüber-
den Höhle, die ca. 1 L. gleichartigen Eiters enthielt. Gegen-
, Dränage und Tamponade. Nach 3 Wochen Verschlechterung
d. Die Sektion ergab auch hier multiple, eitrige intraperitoneale
Daneben eine Thrombose beider Ven. iliacae, die bis in die
henkel reichte.
letzten 3 Fälle stammen aus der Breslauer Klinik. Der eine
nen war am 13. Tage nach Beginn der Erkrankung der Eröff-
ines großen Douglasabszesses per rectum unterzogen worden.
od erfolgte 4 Tage nach der Operation unter den klinischen
nungen der Peritonitis. Die Autopsie ergab neben einem
Abszeß, der die ganze rechte Bauchhälfte vom Subphrenium
Fossa iliaca ausfüllte und gegen die übrige Bauchhöhle durch

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Adhäsionen geschlossen war, multiple kleinere Abszesse zwis
verklebten Dünndarmschlingen. Der Appendix war mit eine
schlinge verklebt und zeigte eine erbsengroße Perforation in d

Die 2 anderen Fälle gingen beide an fortschreitender P
zugrunde. Der eine war am 4. Tage nach Beginn der Er
zur Abszeßinzision gelangt und ist bei den Zwischenoperation
beschrieben worden. Der andere Fall war 5 Tage nach Be
Erkrankung mit erhöhter Temperatur und Puls, sowie eine
großen Resistenz in der rechten Unterbauchgegend zur Aufn
kommen. Unter konservativer Behandlung trat am 12. und
eine plötzliche Exazerbation der Erscheinungen mit Schüttelf
peritonitischen Symptomen ein, denen eine vorübergehende B
folgte. Am 19. Tage war eine deutliche Vergrößerung des E
nachweisbar, die Inzision rechts ergab einen kleinen von re
Adhäsionen umgebenen Abszeß auf der rechten Beckenschauf
median im Becken eine Resistenz, die auf Punktion Eiter
worauf daselbst inzidiert und eine Abszeßhöhle entleert wur
Patient starb am 7. Tage nach der Operation unter dem k
Bilde der fortschreitenden Peritonitis. Die Autopsie erg
Pelveoperitonitis purulenta mit allgemeiner fibrinöser Pe
Außerdem fanden sich Netzadhäsionen, eine partielle Obliter
Appendix, Hepatitis, vereinzelte Abszesse in den beiderseitige
Lungenlappen und doppelseitige Pneumonie. Man darf de
Ausgang des Falles demnach wohl mit Recht auf die s
Spontanperforation eines zunächst abgekapselten Abszesses
freie Bauchhöhle zurückführen, und die Annahme ist nicht
Hand zu weisen, daß dieser unglückliche Zufall bei einer
konservativen Behandlung sich vielleicht hätte vermeiden
Der Fall spricht somit für die radikalere Anschauung bezügli
operativen Vorgehens im Zwischenstadium, welche dadurch
wie möglich die eigentliche Causa nocens zur Ausschaltung
und den Patienten vor derartigen traurigen Zufällen bewah

Für die Einteilung der gesamten Fälle nach anatom
Prinzip werden wir am besten nach dem ältesten Vorschlag
bergers unterscheiden zwischen den intra- und extraperit
legenen Abszessen.

Für die ersteren scheint mir von den zahlreichen Einteilu
Sprengel anführt, diejenige von Rotter und von Ger
besten und einfachsten zu sein. Der letzteren gibt auch S
den Vorzug. Rotter unterscheidet: 1. Douglasabszesse, 2.
der rechten Beckenschaufel, 3. Abszesse der Lumbalgegend
szesse der linken Beckenschaufel, 5. subphrenische Abszesse

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ist die Einteilung von Gerster, der im übrigen ebenfalls 5 Ab-
en unterscheidet: 1. den ileoinguinalen Typus als den häufigsten,
lumbalen oder posteroparietalen Typus, 3. den anteroparietalen
4. den rektalen oder pelvikalen Typus und 5. den mesocölia-
Typus. Den ileoinguinalen Typus könnte man a priori wohl
ide Seiten gleichzeitig beziehen. Daß Gerster darunter den
der rechten Beckenschaufel verstanden wissen will, ergibt
raus, daß er ihn für den häufigsten Typus erklärt im Gegen-
dem seltenen Abszeß der linken Beckenschaufel, den er in
Einteilung gar nicht weiter berücksichtigt hat.

len Einteilungen gemeinsam ist 1. der Douglasabszeß = pelvi-
ypus, 2. der Abszeß der rechten Beckenschaufel = ileoinguinaler
3. Abszeß der Lumbalgegend = lumbaler oder posteroparietaler
Rotter hat dazu noch 4. den Abszeß der linken Becken-
l und 5. die subphrenischen Abszesse angeführt. Gerster
noch hinzu 6. den anteroparietalen Typus und 7. den meso-
en Typus. In den letzten beiden Punkten ergänzen sich beide
ngen sehr glücklich und wir werden daher im folgenden ver-
unsere intraperitonealen Abszesse nach den obigen 7 Abtei-
zu rubrizieren, zu denen ich noch als 8. Abteilung die mul-
Abszesse hinzufügen möchte.

der Verteilung der Fälle auf die einzelnen Kategorien ist jedoch
enken, daß im Laufe der Entwickelung die einzelnen Abszeß-
leicht ineinander übergehen können, wenn auch nach Sprengel
Vorgang für die beiden häufigsten Formen, den ileoinguinalen
nbalen Typus nur äußerst selten zutreffen soll. Ist es doch
r großen Abszessen selbstverständlich, daß sie sich von der
Beckenschaufel nach irgendeiner Richtung, sei es unter die
sei es bis in die linke Inguinalgegend oder nach dem Douglas
breiten müssen. Durch spätere Teilung bzw. Verkleinerung
zesses von einer Seite her, falls eben eine Resorption eintritt,
ann wohl der eine oder andere Typus der vorherrschende
Solange man aber Abszesse in derartiger Größe vor sich
d man sie immer zu den Mischformen zählen müssen und im
sein, zu welcher der obigen Abteilungen man sie zu rechnen
h habe mir da in der Weise geholfen, daß ich derartige Ab-
ohne weiteres zu dem ileoinguinalen Typus rechnete, da sie
age der Dinge von hier aus entstanden und nur durch ihr
sen die Grenzen nach der einen oder anderen Richtung ver-
ten.

ferneren habe ich bei dem Vorhandensein mehrerer größerer
e dieselben auch unter den einzelnen Formen angeführt, so

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daß die betreffenden Fälle mehrfach angeführt sind. Es ersc
dieses wichtig für die Häufigkeit der einzelnen Abszeßforme
wären sonst einzelne Kategorien vollständig unter derjenigen
tiplen Fälle verloren gegangen, wie wir später genauer sehe

Am zahlreichsten vertreten finden wir naturgemäß den
inguinalen Typus.

Derselbe ist in 72 Fällen = 63,8% vorhanden. In 4 Fälle
der große Abszeß bis in den Douglas hinab. Sämtliche 4
auf 1 wurden inguinal eröffnet und bis in den Douglas drän
2 von ihnen wurden per rectum entleert, worauf glatte Hei
trat. In 3 anderen Fällen reichte der Abszeß bis zum Rip
hinauf. In 1 Fall wurde der alte Abszeß als Karzinom ange
und erst durch die Sektion die Klärung der Sachlage herb
Die übrigen Fälle bieten keine Besonderheit dar und brauc
näher besprochen zu werden.

2. Der Abszeß der linken Inguinalgegend fand sic
7 Fällen = 6,2% vor. Im Fall 17 bestand neben einem gäns
Abszeß der rechten Beckenschaufel ein zweiter faustgroße
der linken Leistenbeuge, mit gashaltigem stinkendem Eite
Nach einer Inzision beiderseits glatter Heilungsverlauf.

Im Fall 19 fand sich schon bei der Untersuchung nur ei
empfindlichkeit und Resistenz über dem linken Lig. Poup
die Inzision und Dränage nur der linken Inguinalgegend
machte. Es zeigte sich nun, daß die große dickwandige Ab
von vorne oben links sich nach hinten rechts und in den
hinein erstreckte und mit fäkulentem Eiter gefüllt war. Der
wurde nicht gefunden. Nach 6tägigem Fieber glatter Heilun

Im Fall 22 handelte es sich um eine walnußgroße Ab
links seitlich an der Bauchwand neben verschiedenen and
szessen. Der Fall wird bei den multiplen Abszessen näher b
werden.

Im Fall 26 reichte die sehr große Abszeßhöhle von rec
nach oben bis zum Rippenbogen und nach links bis zum li
Poupartii hinüber. Der Fall ist also richtiger zur ersten
zu rechnen und ist dort auch mitgezählt worden.

Im Fall 54 ließ sich sowohl rechts, wie links eine deutli
stenz nachweisen. Die beiderseitige Inzision über der Leis
ergab rechts eine mit ca. $^1/_2$ l Eiter gefüllte Höhle, die n
hinter der Blase herum bis auf die linke Seite derselben rei
zweite Höhle links enthielt ebenfalls 1 l Eiter und erstre
nach unten bis in die Gegend des Rektums hin. Eine Ve
zwischen den beiden Höhlen ließ sich nicht nachweisen.

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e linke von der rechten ihren Ausgang genommen und erst lich ist es durch spätere Verklebungen zu einer Trennung en Höhlen gekommen.

en lumbalen oder posteroparietalen Abszeß finden wir 0 von unseren Fällen = 8,8% vertreten.

dem 1. Fall (3) handelte es sich um eine intraperitoneale, ritische Abszeßhöhle neben einer gleichzeitigen extraperito- Höhle. Trotzdem der Fall durch Diabetes kompliziert war, Inzision und Gegeninzision glatte Heilung ein.

. Fall bestand ein sehr hoch, direkt hinter der Leber ge- Abszeß, der dem ebenfalls sehr hoch gelegenen und nach oben agenen Appendix entsprach. Auch hier trat trotz gleichfalls nem Diabetes nach breiter Inzision und Dränage glatte Hei-

. Fall (15) war die Höhle von einem retrocöcal gelegenen k ausgegangen und bot sonst nichts Bemerkenswertes dar.

4. Fall (31) zeigte bei normaler Temperatur in der rechten gend eine handtellergroße Geschwulst ohne entzündliche Er- gen, von der ein Drittel oberhalb und zwei Drittel unterhalb Rippe gelegen waren. Auf Druck ließ sich daselbst ein plät- s Geräusch hören. Der Inhalt lag dicht unter der Haut vor Rippe. Über der ganzen Resistenz war tympanitischer Darm- rhanden. Nach Anlegung einer 20 cm langen Inzision vom Rande der 12. Rippe nach abwärts zeigten sich unter der en Muskulatur und Peritoneum harte Schwielen, darunter glasige Granulationen mit einem unter der 12. Rippe ge- freien Kotstein. Von hier aus führte ein fingerlanger Gang hen Granulationen bis hinter das Cöcum. Später erfolgte von Winden und einer weißlichen Flüssigkeit aus der Drä- e und es blieb eine kleine Kotfistel zurück. 2 Monate später dann die 2. Operation mit Naht einer Perforationsstelle am nd Tamponade. Der Appendix wurde auch jetzt nicht ge- Glatte Heilung ohne Fistelbildung.

5. Fall (36) entsprach einem walnußgroßen Abszeß zwischen und lateraler Bauchwand und bietet sonst nichts Besonderes.

Fall (67) handelte es sich um eine wurstförmige, hinter dem gelegene Abszeßhöhle mit stinkendem Eiter, die sich bis in en- und linke Beckengegend hin erstreckte. Später wurde ch die Eröffnung einer zweiten vor dem Rektum gelegenen on der alten Wunde aus notwendig.

Fall enthielt die große retrocöcale Höhle neben stinkendem e alte Blutung infolge einer vorausgegangenen Gefäßarrosion.

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Nach Inzision auf retroperitonealem Wege und lumbale
inzision trat Heilung mit Zurückbleiben einer kleinen Fist
Dränagestelle ein. Die übrigen 3 Fälle der Breslauer Kli
mir in einem näheren Bericht nicht vor.

5. Dem anteroparietalen Typus dürften 5 Fälle = 4,4
Zusammenstellung entsprechen, in denen der Abszeß nach
hin gelegen war.

Im 1. Fall (8) handelte es sich um eine große, mit grü
haltigem und stinkendem Eiter gefüllte Höhle, die an der
Bauchwand unterhalb des Zwerchfells gelegen war. Der
in seinem Verlauf durch eine Pleuritis serosa über der L
pliziert, welche wegen Gefahr der Vereiterung die Resekt
bis 10. Rippe notwendig machte. Darauf glatte Heilung.

Im 2. Fall (13) bestand eine sehr große Höhle, die
nach oben bis zum Rippenbogen und nach links über die
hinausreichte. Nach Inzision und Drainage derselben wu
noch die sekundäre Eröffnung und Drainage eines Senkung
vom Douglas aus notwendig.

Im 3. Fall (20) lag eine teils intra-, teils extraperitonea
höhle vor. Die Patientin hatte seit 3 Monaten an inner
Schmerzen um den Nabel herum gelitten. Auf Umschläg
3 Tagen eine spontane Perforation und Entleerung von übelr
Eiter am Nabel eingetreten, so daß nur noch eine Disz
Fistel mit Tamponade nötig war. Danach zeigte sich vom
links gelegen eine größere Höhle in der Muskulatur (Rektus
nach rechts ein langer kommunizierender Gang bis in die
gegend führte, in welchem ein freier Kotstein lag. Der Ab
sich also von der Ileocöcalgegend nach vorne oben bis z
wand ausgebreitet, hatte allmählich Peritoneum und Fasc.
perforiert und sich nun sekundär in der Muskulatur au
während intraperitoneal bereits eine Resorption und Schru
der Ausgangsstelle des Exsudats erfolgte. Der Fall bietet
gute Illustration zu den hinsichtlich der Einteilung der Ab
machten Ausführungen.

Im 4. Fall bestand neben einer intraperitonealen Abszeß
sich von der Ileocöcalgegend bis zum Nabel erstreckte und
Blase hin abgeschlossen war, eine zweite kleinere Abszeß
vor der Blase dicht über der Symphyse, also vermutlich
toneal gelegen war. Von dieser führte ein breiter Gang
rechts hin, der jedoch eine weitere Kommunikation nicht
ließ. Es mußte also auch hier erst eine Perforation des P
und der Fasc. transv. stattgefunden haben, ehe es zu der

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tevesikal gelegenen Abszesses kommen konnte. Der 5. Fall ebenfalls von oben eröffnet und bot keine Besonderheit dar. hst dem ileoinguinalen Abszeß ist am häufigsten der 5. pelvider rektale Abszeß in unserer Zusammenstellung mit 24 Fällen % vertreten. Von diesen verdienen nur wenige Fälle eine Erwähnung. So war im Fall 13 nach Inzision eines anteroen Abszesses eine sekundäre Eitersenkung nach dem Douglas eten, welche die Eröffnung und Dränage per rectum notwendig

gekehrt wurde im Fall 5 nach Spaltung und Dränage eines sabszesses per rectum die weitere Spaltung eines zweiten Ab- per abdomen notwendig. Es war der Fall, in dem später eine eckenphlegmone und zahlreiche metastatische Abszesse der inzukamen, an denen der Patient schließlich zugrunde ging.
e sehr große Höhle wurde im Fall 29 per rectum eröffnet. tleerte aus derselben nicht weniger als 2 l Eiter. Der Heilungswar glatt.
Fall 40 wurde die im kleinen Becken gelegene Höhle von einer n per abdomen inzidierten Höhle aus eröffnet. Es wurde von er, aber auch gleichzeitig durch das Rektum dräniert. Unter n Verlauf trat Heilung ein.
42 enthielt neben dem Douglasabszeß weitere multiple Abund wird unter jener Kategorie näher besprochen werden.
Der mesocöliacale Abszeß ist nur in 4 Fällen = 3,5% der fälle vertreten. Alle 4 Fälle gehören zu der Kategorie der en Abszesse. Einen Fall von reiner mesocöliacaler Abszeß- allein habe ich unter unseren 113 Fällen nicht finden können. ge Fälle müssen naturgemäß äußerst selten sein, da bei der n Bewegung der Därme das Exsudat immer die Tendenz haben nach dem Punkte der Ruhe auszuweichen. Dieser entspricht er vorderen und seitlichen Bauchwand und dort wird es naturauch am ehesten zu Abkapselungen und Verklebungen der kommen können.
Der subphrenische Abszeß ließ sich nur in 2 Fällen = 1,8% ieren. Im Fall 8 trat später eine Pleuritis serosa über der ein. Nach Resektion der 7.—10. Rippe glatte Heilung. Im war ein subphrenischer Abszeß rechts und links gleichzeitig deren multiplen Abszessen vorhanden.
hst dem ileoinguinalen und pelvikalen Typus sind am häufigsten multiplen Abszesse mit 17 Fällen = 15% vertreten. Diese naturgemäß auch die höchste Mortalitätsziffer von allen Kate, indem auf sie weit über die Hälfte sämtlicher Todesfälle

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entfällt. Ich habe zu dieser Kategorie alle Fälle mit mehr Abszeßhöhle gerechnet, da man sonst nach oben hin kei Grenze für diese Fälle ziehen kann.

Bei den meisten Fällen handelte es sich um eine Ko von 2 oder 3 der bereits oben beschriebenen Abszeßformen. zelnen Abszesse sind dort schon näher beschrieben worde wir uns hier damit begnügen können, nur kurz die vers Kombinationen anzuführen. So finden wir im Fall 17 ein und einen linksseitigen Abszeß der Leistengegend, im Fall 1 56 einen ileoinguinalen Abszeß mit einem pelvikalen Abs biniert, während im Fall 77 zunächst ein pelvikaler Abszeß war, dem später ein zweiter Abszeß in der linken Leistenb folgte. Ferner ist der rektale Typus im Fall 13 mit de parietalen und im Fall 67 mit dem lumbalen oder poster Typus kombiniert. Eine Vereinigung von ileoinguinalem anteroparietalen Typus findet sich im Fall 13 und mit d cöliacalen Typus bzw. weiteren multiplen Abszessen im Fal Vereinigung von extraperitonealem mit einem intraperiton szeß bot Fall 3 in Gestalt zweier neben der rechten Niere Abszesse, die nicht miteinander in Zusammenhang stande diesen mehrfachen größeren Abszessen finden sich multipl im eigentlichen Sinne des Wortes nur in 4 Fällen, die kleine und kleinste verstreute Abszesse aufweisen. Zu hören auch die 3 Todesfälle im Fall 5, 22 und 41, die be gelegentlich der Erörterung der gesamten 6 Todesfälle so besprochen sind, daß hier darauf verwiesen werden kann.

Eine nähere Erwähnung verdient jedoch der einzige ge Bei demselben (Fall 42) wurde zunächst am 4. Oktober e bis in das kleine Becken reichende Höhle rechts per ab öffnet. Am 9. Oktober wurde eine lumbale Gegenöffnun Am 17. Oktober erfolgte die transpleurale Eröffnung de wähnten subphrenischen Abszesses links. Am 22. Oktober Resektion der 11. Rippe und transpleurale Eröffnung des zw phrenischen Abszesses rechts gemacht. Am 7. Novembe schließlich die Inzision eines Abszesses links unter dem N Patient hatte also während der 64tägigen, mehr weniger fi Behandlungsdauer 6 größere operative Eingriffe durchmache Wenn er schließlich als geheilt entlassen werden konnte, s das jugendliche Alter des Patienten von 10 Jahren ein gut dem glücklichen Ausgang beigetragen.

Wenden wir uns nun den extraperitonealen Abszess teilen wir dieselben am besten in retro- und präperitonea

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sse ein und fügen den ersteren noch als besondere Unterabteilung
trofaszialen Abszesse hinzu.

r die retroperitoneale Lokalisation können wir im ganzen 11
= 9,7% anführen. Im Fall 3 handelte es sich um den oben er-
en paranephritischen Abszeß mit gleichzeitigem Diabetes. Im
2 bestanden gleichzeitig zahlreiche andere Abszesse. Im Fall 43
sich eine faustgroße retroperitoneale Höhle auf der rechten
nschaufel, die ohne Eröffnung des Peritoneums auf retroperi-
em Wege entleert werden konnte.

Fall 47 erstreckte sich der Eiter bis unter das Lig. Poupartii
er, was allein schon als pathognostisches Merkmal für die retro-
neale Lokalisation des Abszesses anzusehen ist, da sich intra-
neale Abszesse stets oberhalb des Poupartschen Bandes nach
hin bemerkbar zu machen pflegen. Auch hier wurde die Ent-
g auf retroperitonealem Wege vorgenommen.

Fall 48 handelte es sich um eine große Höhle zwischen Peri-
n und Beckenschaufel. In der Tiefe war der Musc. psoas frei-
Die Höhle stieg seitlich an der Wirbelsäule nach oben empor
enkte sich nach unten in das Becken hinein bis zur Vagina.
bestand eine Eitersenkung längs der Gefäße nach dem Ober-
el zu. Hiernach ist nicht mit Sicherheit zu entscheiden, ob
in retrofaszialer Abszeß vorgelegen haben könnte. Nach der
s Befundes will mir das letztere wahrscheinlicher vorkommen,
leich in dem Krankenblatt nur von einem retroperitonealen
die Rede ist. Nach retroperitonealer Inzision fieberhafter
f und glatte Heilung.

Fall 68 reichte die große retroperitoneale Abszeßhöhle von der
einschaufel bis zur rechten Niere an der Wirbelsäule aufwärts.
nzision trat unter fieberfreiem Verlauf glatte Heilung ein.

retrofaszialen Abszeß dürfen wir mit Sicherheit den Fall 30
chen, bei dem es sich um eine reichliche Eiterentleerung direkt
em Musc. ileopsoas handelte neben einem großen intraperi-
n Abszeß, der von vorne her eröffnet wurde. Der ringförmig
mte Appendix war retrocöcal gelegen und konnte entfernt
. Glatter Heilungsverlauf ohne Fistelbildung.

präperitoneale Abszesse werden in den Journalen die
1 und 52 = 1,8% bezeichnet. Danach bestanden in beiden Fällen
über der Leistenbeuge vor dem Peritoneum gelegene Höhlen,
eine in Faustgröße mit fäkulentem, gashaltigem Eiter gefüllt
während die andere in Hühnereigröße geruchlosen Eiter ent-
Ob diese Höhlen von einem intra- oder retroperitonealen
ihren Ausgang genommen haben, ist aus den Journalen nicht

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zu ersehen. Im zweiten Fall scheint mir die gleichzeitig vorl
Flexionsstellung des rechten Beines für die letztere Annal
sprechen.

Als dritten Fall könnte man noch hinzurechnen den Fall
dem es von einer hühnereigroßen intraperitonealen Höhle
folge zweier Perforationen des Peritoneum parietale und d
transvers. zu einer großen Eiterhöhle innerhalb der Bauchwa
kommen war.

Von Erscheinungen am Processus vermiformis im besonder
vornehmlich die Häufigkeit von Perforationen und das Vorhan
von Kotsteinen von Interesse sein.

Diejenigen Fälle, in denen eine Perforation des Append
drücklich angegeben ist, sind in verhältnismäßig geringer Anz
treten. Das erklärt sich ohne weiteres aus der Tatsache, daß
23 Fällen = 20,3% der Appendix aufgefunden bzw. entfernt
konnte. Von diesem Gesichtspunkt aus hat auch das pro
Zahlenverhältnis der Perforationen keinen Wert, da in de
wiegenden Mehrzahl der übrigen Fälle mit größter Wahrschein
eine vorausgegangene Perforation anzunehmen ist. Mit Si
kann sie jedoch nur da angenommen werden, wo das Vorhan
eines freien Kotsteins in der Abszeßhöhle dafür spricht.

Unter den 23 Fällen von Amputation des Processus ist in
drücklich eine Perforation desselben angeführt. Und zwar lag
in der Mitte des Processus 4mal, an der Spitze 7mal und an d
in 2 Fällen. In einem Fall bestanden außer an der Spitze no
Perforationen an der Basis und in einem anderen Fall eine s
der Wand des Cöcum. Ferner fanden sich gleichzeitig be
Fall zwei Perforationen am Dünndarm und eine am Cöcu
Processus war nekrotisch zerfallen, so daß man dieselben v
Reperforationen mit Recht auffassen darf. Ebenso fanden
einem anderen Fall zwei alte Perforationen am Cöcum, für d
die gleiche Ätiologie zutreffen dürfte.

Das Vorhandensein eines Kotsteins wird ausdrücklich erwä
in 14 Fällen. Auch hier geht man nicht fehl in der Annah
bei der namentlich in früheren Jahren geübten einfachen Inzi
Abszeßhöhle ohne genauere Revision derselben bisweilen ein
häsionen eingebetteter Kotstein zugleich mit dem Processus de
nehmung entgangen sein kann, ein Vorgang, der ja auch heute
lich nicht immer mit absoluter Sicherheit auszuschließen
ausnahmsweise in anderen Fällen eine Verflüssigung des Kot
dem Exsudat stattgefunden haben kann, ist eine Frage, über
die Akten noch nicht geschlossen sind.

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edem Fall erscheint die Annahme berechtigt, daß die tatsäch-
ahl der vorhandenen Kotsteine etwas höher zu veranschlagen
die in den Journalen angegebene.
en angegebenen 14 Fällen lag 10mal der Kotstein außerhalb
ocessus in der freien Abszeßhöhle, darunter einmal in der
les Cöcum. In 1 Fall fanden sich zwei freie Kotsteine gleich-
or. Nur in 2 Fällen lag der Stein innerhalb des Processus.
deren Fällen ist eine genauere Lokalisation des Steines nicht
ngegeben. In 6 von den Fällen wurde der Processus nicht
n, bei den anderen wird ausdrücklich eine Perforation des
us in den Journalen vermerkt. Die 6 Fälle sind trotzdem
herheit als Perforationsfälle aufzufassen, so daß dadurch die
r sicheren Perforationen auf 23 Fälle steigt, eine Zahl, welche
noch als zu gering erscheinen muß für die Gesamtzahl von
en.
die sonstigen pathologisch-anatomischen Veränderungen am
ix näher einzugehen verlohnt sich einerseits nicht im Hinblick
geringe Anzahl von 23 Fällen, in denen er überhaupt gefunden
dererseits sind auch in diesen Fällen nur ganz vereinzelt
Angaben darüber in den Journalen vorhanden, was sich viel-
daraus erklären dürfte, daß auch hier meist das Bild durch
rtgeschrittene Nekrosen und Zerfallserscheinungen am Appen-
eits ein stark verwischtes war.
Therapie bestand in sämtlichen Fällen in Inzision und Drä-
it Jodoform- bzw. Isoformgaze, die in der Regel durch Ein-
ines dicken, durchlöcherten Gummi- oder Glasdräns unter-
urde. In einzelnen Fällen wurde eine Gegeninzision in der
gegend oder im Rektum notwendig, durch welche dann eben-
äniert wurde. Die reinen Douglasabszesse wurden sämtlich
sion und Dränage vom Rektum aus behandelt, nachdem die
Sphinkterdehnung vorausgegangen war. Die Inzision per ab-
wurde stets in ausgiebiger Länge gemacht, so daß sie einerseits
uten Überblick über die Wundhöhle und das Operationsfeld
dererseits eine ausgiebige Entleerung der Höhle durch Tupfer
ülungen ermöglichte. Stets wurde nach Möglichkeit die Er-
des freien Peritoneums zu vermeiden gesucht. Wo dieses
öglich war, erfolgte der Schutz der freien Bauchhöhle sofort
visorisch eingelegten Kompressen von steriler Gaze, die ihren
vollständig erfüllten. Am Schluß wurden die Inzisionen in der
von den Wundwinkeln her bis auf die für die Dränage not-
e Öffnung verkleinert. In fast sämtlichen Fällen trat in mehr
kurzer Zeit nach dem operativen Eingriff die völlige Ent-

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fieberung ein und hielt bis zur Heilung per granulat. an. Ei
nahme davon bildeten die Fälle, welche durch das Auftreten
Abszesse oder sonstiger Komplikationen von dem gewöhnliche
heitsverlauf abwichen. Hervorheben möchte ich noch, daß in
nach 9 Tagen die Sekundärnaht gemacht wurde, und die gla
lung derselben p. p. eintrat. Nur bei 4 Radikaloperationen d
lauer Klinik wurde primär geschlossen und zwar 2mal mit u
ohne Tampon.

Von Komplikationen der Erkrankung finden wir D
in 2 Fällen erwähnt. Im Fall 3 lag neben der intraperi
eine extraperitoneale paranephritische Abszeßhöhle vor. In
handelte es sich um einen unter der Leber gelegenen Abs
beiden Fällen läßt die Lage der Abszesse an die Möglichke
ätiologischen Zusammenhanges der Erkrankung mit dem
denken, so daß dann der letztere als sekundär aufzufassen wä
wiesen wäre derselbe nur mit dem Aufhören des Diabetes n
lung der Periappendizitis. Leider ist eine Angabe darüber in
Krankengeschichten nicht zu finden. In beiden Fällen wurd
falls der Heilungsvorgang durch den Diabetes nicht beeinflu
eher für als gegen die Annahme eines derartigen Zusamme
sprechen dürfte.

Erysipel trat bei einem präperitonealen Abszeß von der
Inzisionswunde aus auf. Der Fall verlief mit entsprechende
peratursteigerungen, kam aber auch zur Heilung. Wie zu erwar
wurden in dem Abszeßeiter mikroskopisch Streptokokken nachg

Seröse Pleuritis rechts trat nach Spaltung eines subphr
rechtsseitigen Abszesses auf und machte die Resektion de
10. Rippe notwendig. Die Infektion hat hier wohl in einer
Durchwanderung der Erreger durch das Zwerchfell auf dem
wege bestanden. Eine embolische Verschleppung auf dem
dürfte für den Fall kaum zutreffend sein. Ein direkter Dur
durch das Zwerchfell war abgesehen von der späteren O
schon durch den serösen Charakter des Exsudates mit Si
auszuschließen. Auch hier trat später glatte Heilung ein.
fand ich in je einem Fall eine trockene und eine exsudative
die beide spontan zurückgingen.

Ein Abszeß links oberhalb des Zwerchfells wurde
Sektion des Falles 22 gefunden, nachdem vorher bereits 5mal A
darunter ein periumbilikaler, ein retroperitonealer und die
intraperitoneal gelegen, inzidiert waren. Auch hier ist na
Sektionsbefund der obige Infektionsmodus als der wahrsche
anzunehmen.

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e Flexionskontraktur des Oberschenkels, die in der ache auf eine Psoitis zurückzuführen sein dürfte, fand sich in n.

selbe war im Fall 52 mit einem präperitonealen Abszeß rechts den. Das Vorhandensein der Flexionskontraktur scheint mir Annahme zu sprechen, daß aus einem ursprünglich retroperi- n Abszeß durch Senkung bis zur Leistengegend und aufstei- nfektion der Lymphgefäße oder Venen die präperitoneale Ab- ung erst sekundär entstanden sein könnte. Zum mindesten der präperitoneale Abszeß allein kaum für die Ätiologie der skontraktur in Betracht zu ziehen sein.

Flexion des rechten Beins bestand ferner im Fall 22 seit en vor der Aufnahme. Es handelte sich um eine sehr große trocöcal, teils ileoinguinal gelegene Abszeßhöhle, in welcher pendix nicht gefunden wurde.

nso bestand im Fall 23 bereits seit 4 Wochen vor der Auf- eine Unbeweglichkeit des rechten Hüftgelenks, die ihre Er- durch eine faustgroße retroperitoneale Abszeßhöhle mit einer g längs der Oberschenkelgefäße fand. Ganz ähnliche Verhält- ot der Fall 58. Auch hier bestand die Flexionskontraktur als ines auf dem Musc. psoas gelegenen Abszesses, der sich an fäßen entlang nach dem Oberschenkel zu gesenkt hatte. Auch r der Abszeß mit Sicherheit als retroperitoneal anzusprechen. ter Parallelfall dazu dient Fall 84, in dem bereits seit 10 Wochen ch eine Lähmung des rechten Beins bestanden hatte, so daß der enkel in Beugestellung gehalten wurde. Auch hier bietet die g der großen Abszeßhöhle bis unter das Leistenband die Gewähr, daß man es mit einem retroperitonealen Abszeß zu te. Es war derjenige Fall, bei dem 10 Tage post op. die die Diagnose eines Cöcumkarzinoms ergab, ohne daß der us selbst gefunden wurde.

Flexion beider Beine bestand im Fall 61 neben einer sehr intraperitonealen Abszeßhöhle, die bis zur Blase und von nach links über die Mittellinie hinüber reichte. Der Processus nicht gefunden und erst später nach dem 12. Anfall im Sicher- dium entfernt.

ämtlichen 6 Fällen finden wir also 3, bei denen wir es un- haft mit einem retroperitonealen Abszeß zu tun haben, und bei dem es wahrscheinlich aber nicht bewiesen ist. In den übrigen Fällen wird der Abszeß als ein sehr großer intraperi- gelegener angegeben. Das Auftreten der Flexionskontraktur retroperitonealen, und ganz besonders bei retrofaszialen

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Abszessen ein so häufiges, daß man sie heute mit Recht als ei
gnostisches Merkmal für dieselben bezeichnet. Aber auch b
peritonealen Entzündungsprozessen kann es leicht zu einer
kommen, selbst wenn diese zunächst nur in einer rein entzü
Affektion des retrocöcalen Peritoneum parietale ohne Absz
bestehen.

So hat Sprengel einen solchen Fall beobachtet, in dem
bei der wegen Verdachts auf Appendizitis vorgenommenen O
weiter nichts nachweisbar war, als eine vermehrte Peritone
keit und nur wenig entzündlich veränderte rechtsseitige Adn
führt in seinem Fall die Erscheinung auf eine entzündliche
des peritonealen Überzuges des Musc. ileopsoas zurück,
Schmerzhaftigkeit bei jeder Dehnung des Muskels zur Folg
muß. Um diese zu vermeiden, wird dann das Bein von den
in willkürlich flektierter Stellung gehalten. Auch bei anderen
findet man häufig Fälle, bei denen auf eine ausgesprochene
haftigkeit bei Bewegungen und namentlich bei Streckung des
Beins im Hüftgelenk hingewiesen wird. Man hat darin
Recht ein Frühsymptom einer entzündlichen Reizung des ret
Peritoneum parietale zu erblicken, welche in der Regel ihren
von einer Periappendizitis, bei weiblichen Patienten selte
von einer rechtsseitigen Adnexitis genommen haben wird.

Bei vorgeschrittener intraperitonealer Abszeßbildung kön
weiter myogene oder auch neurogene Ursachen für die
kontraktur in Frage kommen, indem es zu einer infektiö
toxischen Myositis des Musc. ileopsoas oder einer Neuritis
dem Muskel hinter der Faszie verlaufenden Nerv. cruralis
kann. Ebenso ist die Möglichkeit einer rein mechanische
wirkung der sich ausdehnenden Abszeßwandung auf den N
ralis nicht von der Hand zu weisen, deren Folgen sich eb
Schmerzhaftigkeit bei Bewegungen und dadurch bedingter will
Ruhestellung des rechten Beins in flektierter Haltung äußern
Letztere Erklärung scheint mir auch für unsere beiden Fälle
peritonealer Abszeßbildung zutreffend zu sein. Wenigsten
einerseits die Größe der Abszesse für die Möglichkeit eine
wirkung auf den Nerv. cruralis, während andrerseits für ent
Veränderungen am Musc. ileopsoas durch die Operation e
weis nicht zu erbringen war. Alles in allem dürfte die
haftigkeit bei Bewegungen und spätere Kontrakturstellung de
Beins als Frühsymptom beginnender Periappendizitis ein
sein, dessen Bedeutung der Beachtung nicht dringend genug e
werden kann.

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ne Thrombose der linken Femoralvene fand sich im ganzen
darunter 1mal nach der Operation einer großen intraperitonealen
ßhöhle rechts, die mit braungelbem, stinkendem Eiter angefüllt
Über die Möglichkeit einer vorausgegangenen Unterbindung der
und Vena epigastrica ist aus dem Journal nichts zu ersehen.
ieberfreie Verlauf scheint mir dafür zu sprechen, daß die Throm-
auf den Zusammenhang mit einer solchen zurückzuführen ist,
en ja oben bereits hingewiesen ist. Anderenfalls müßte man auf
ltere Anschauung zurückgreifen, welche die Ursache der links-
en Thrombose in einer Erschwerung des Abflusses aus der linken
femoralis in die rechts gelegenen Vena cava erblickt, indem der
ündungswinkel der linken Femoralvene erheblich stumpfer zu der
cava verläuft, als derjenige der rechten Femoralvene.
hrombose der rechten Femoralvene fand ich nur in 1 Falle.
ne Thrombose beider Ven. iliac., die bis in die Unterschenkel
reichte, ergab die Sektion des Falles 41. Schon 8 Tage nach
inlieferung trat in dem Fall eine klinisch nachweisbare Throm-
beider Unterschenkel unter hohem Fieber ein. Die Operation
zwei große intraperitoneale Eiterhöhlen, deren rechts gelegene
cm Eiter enthielt, während die größere links gelegene mit
l Eiweiß gefüllt war. In diesem Fall ist die rein infektiöse
der ausgedehnten Thrombophlebitis durch das ganze Krank-
ild und den hohen Temperaturanstieg bei ihrem Beginn ein-
sfrei bewiesen.
eritonitische Erscheinungen post op. traten nur in einem
auf, gingen aber spontan zurück. Als ihre Veranlassung nahm
eine intraabdominale rezidivierende Abszeßbildung auf, die sich
an zurückbildete.
ne Fistelbildung bei der Entlassung der Patienten bestand
Fällen = 5,3% der Gesamtfälle. Im ersten Fall fand sich an der
des Processus eine Perforation neben einer Obliteration im
eren Drittel desselben. Nach Amputation des Processus war mit
ormgaze dräniert worden.
n 2. Fall bestand eine Perforation in der Mitte des Processus
n einem bohnengroßen Kotstein, der die Basis des Processus
loß. Auch hier wurde der Processus amputiert und mit Jodo-
gaze dräniert. Der Fall verlief subfebril. In beiden Fällen werden
isteln als Kotfisteln bezeichnet.
it „eiternder Fistel" entlassen wurde der Patient im Fall 36. Es
elte sich um einen walnußgroßen zwischen Cöcum und lateraler
hwand gelegenen Abszeß mit stinkendem Eiter gefüllt. Der Pro-
us wurde nicht gefunden. Auch hier dürfte aller Wahr-

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scheinlichkeit nach eine Perforation des Processus vorausge
sein.

Mit „kleiner Fistel“ entlassen wurde Fall 30, bei dem ein
faszialer und ein intraperitonealer Abszeß durch lateralen I
schnitt entleert war. Der Processus wurde gefunden und am
Es ist der einzige Fall mit Fistelbildung, bei dem ausdrückli
geführt wird, daß am Processus keine Perforation vorhande
Trotzdem bestand bei fast afebrilem Verlauf eine Eiterung fo
schließlich eine Fistel bestehen bleiben ließ.

Im Fall 74 blieben bei der Entlassung zwei Fisteln gleichzei
stehen. Der Processus wurde in dem ileoinguinalen Abszeß
gefunden. Ob eine Perforation stattgefunden hatte, ist ungewi

Ebensowenig ließ sich dieses im Fall 83 feststellen, da auc
nach Entleerung des ileoinguinalen Abszesses der Processus
gefunden wurde. Über das spätere Schicksal der Fisteln find
nur im Fall 33 die Angabe, daß nach einem Monat eine Sp
heilung eingetreten ist.

In 3 von den 6 Fällen mit Fistelbildung hat also eine Amp
des Appendix stattgefunden, während derselbe in den anderen 3
zurückgelassen werden mußte. In 3 Fällen hat vorher mit Sic
eine Perforation stattgefunden, in 2 Fällen ist sie als wahrsch
anzunehmen und in 1 Fall ist dieselbe mit Sicherheit auszusch

Ob in den obigen Fällen das Lösen der Jodoformgazedrän
der Stelle der Darmnaht zu einem Aufgehen derselben und
haltung der Fistelbildung geführt haben kann, läßt sich nacht
nicht entscheiden. Immerhin ist vielleicht für einzelne Fälle
Möglichkeit nicht von der Hand zu weisen.

Eine Periappendicitis diffusa ist in 27 Fällen zur Op
gelangt. Wir haben den Eingriff aus den oben angeführten G
als Notoperation bezeichnet. Auf diese Notoperation entfall
ganzen 19 Fälle mit tödlichem Ausgang = 70,4% Mortalität.

Auf die beiden Geschlechter sind die Fälle ziemlich gleich
verteilt, indem 16 Männer und 11 Frauen davon betroffen sin

Bezüglich des Alters verteilen sich die einzelnen Fälle folg
maßen: Von 1—10 Jahren 4 Fälle, von 11—20 7, von 21—30
31—40 6, von 51—60 3 und von 61—70 Jahren ebenfalls 3 Fä

Was die Anzahl der vorausgegangenen Anfälle anbelangt, so
wir in 20 Fällen = 74,1% nur 1 Anfall, in 6 Fällen = 22,2%
ausgegangene Anfälle und in 1 Fall = 3,7% 3 vorausgegange
fälle angegeben. Es entspricht dieses der schon bei den Spät
tionen hervorgehobenen und bestätigten Erfahrungstatsache vo
vielfach besonders schweren Verlauf gerade der ersten Appen

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so daß man diesen gegenüber bezüglich der Prognose ganz
ers mißtrauisch sein muß.

Zeitpunkt der Operation ist in diesen Fällen berechnet von
eitpunkt der Perforation bzw. des Beginns der Allgemein-
tis ab, soweit derselbe aus den in der Anamnese angegebenen
renden Symptomen zu ersehen war. In der Regel ließ sich
der Zeitpunkt mit ziemlicher Sicherheit festlegen.

diesem Gesichtspunkt sind innerhalb der ersten 48 Stunden
r Perforation zur Operation gelangt 7 Fälle. Sämtliche Fälle,
, sind zur Heilung gelangt. Von den übrigen 20 Fällen sind
'age 8 Fälle, am 4. Tage 3, am 5. Tage 4, am 6. Tage 1 und
päter 4 Fälle operiert worden. Die beiden einzigen geheilten
von sind am 3. und 5. Tage zur Operation gelangt.

rend wir hier also in den ersten 48 Stunden das relativ
Ergebnis von 14,3% Todesfällen haben, steigt die Mortalitäts-
ch 48 Stunden steil auf 90% an.

Processus konnte gefunden und entfernt werden in 20 Fällen
, er wurde nicht gefunden und mußte zurückgelassen werden
len = 22,2%, in 1 Fall ist es nach dem Journal unentschieden.
teren Fälle sind sämtlich Todesfälle, womit natürlich nicht
ein soll, daß dieses der alleinige Grund für den tödlichen
war. Ein Grund mehr dazu wird das Zurückbleiben eines
ll begriffenen Processus jedenfalls sein.

den anamnestischen Angaben ist von besonderem Inter-
Häufigkeit bzw. Konstanz der für die Perforation als patho-
h zu bezeichnenden Symptome. Von diesen kann wohl oft das
r andere fehlen, immerhin wird das gleichzeitige Vorhanden-
r übrigen alarmierenden Symptome die eingetretene Ver-
erung des Krankheitsbildes nur selten verkennen lassen.

mehr weniger heftige und plötzlich einsetzende Charakter des
anfalls wird fast in sämtlichen Fällen hervorgehoben. In der
ar derselbe mit Übelkeit verbunden und machte sich auch
enten selbst durch seine schnell zunehmende Intensität gegen
hergehenden Krankheitszustand deutlich bemerkbar. Meistens
Schmerz als sehr heftiger Leibschmerz angegeben, seltener
rselbe in der Ileocöcalgegend lokalisiert.

Erbrechen setzte fast immer gleichzeitig mit dem Schmerz-
in und ist in sämtlichen Fällen vorhanden gewesen außer im
In diesem Fall ist es trotz einer bereits 4 Tage lang be-
en Verhaltung von Stuhl und Winden nicht zum Erbrechen
en. Über den Charakter des Erbrochenen ist im Fall 1 an-
, daß derselbe gallig war. Im Fall 7 war das Erbrochene

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kothaltig, es bestand seit 2 Tagen Verhaltung von Stuhl und
Sonst sind nähere Angaben darüber nicht vorhanden.

Verhaltung von Stuhl und Winden bestand in 15 Fällen
im Durchschnitt seit 2—3 Tagen, nur im Fall 13 allein wird
raum mit 7 Tagen angegeben. Trotzdem war das Erbroch
kothaltig, jedoch bestand gleichzeitig ein starker Meteorism
ein Ödem beider Oberschenkel. In den übrigen Fällen bes
näckige Obstipation, außer Fall 15, der allein mit Durchf
Erbrechen einsetzte, und Fall 5, bei dem 5 Tage vorher 2
Durchfälle bestanden hatten, und dann erst die übliche O
einsetzte.

Urinbeschwerden sind nur in 2 Fällen angegeben. Im 1.
sie 1 Tag vor der Perforation, im anderen Fall bereits 14 Ta
eingesetzt.

Die Temperatur war in 22 Fällen mehr oder weniger
jedoch wird nur in 1 Fall ausdrücklich ein Schüttelfrost an

Eine vorausgegangene Verabfolgung von Abführmitteln
4 Fällen erwähnt, von denen 3 zum Exitus kamen. Es ist
weiteres anzunehmen, daß Abführmittel in einer weitaus
Mehrzahl von Fällen vorher genommen sind, da ja das Publ
leider auch noch ein Teil der Ärzte bei einem Anfall von Lei
und sei er auch als Blinddarmanfall angesprochen, den Pati
Abführmitteln zu mißhandeln pflegt. Daß ein derartiges
in allen schweren Fällen, die ja klinisch zunächst gar
zustellen sind, die Perforation beschleunigen und die Pro
nachfolgenden Peritonitis durch die künstlich angeregte
staltik ganz erheblich verschlechtern muß, liegt auf der H
solche Fälle dann durch diejenigen aufgewogen werden,
der Anfall durch Abführmittel, insbesondere das beliebte
kupiert worden ist, erscheint doch recht fraglich. Zum
müßte einmal bewiesen werden, daß die letzteren Fälle ohn
mittel per os, allein durch Einläufe und entsprechende Reg
Diät, nicht auch zur Heilung gekommen wären, und zun
müßte man in der Lage sein, vor einer Ordination von Abf
untrügliche klinische Kennzeichen für die Schwere oder Ge
keit der am Processus tatsächlich vorhandenen pathologis
änderungen anführen zu können. Solange das nicht der
wird jeder Arzt, der nach Ordination von Purgantien ge
eine sehr virulente und foudroyante Peritonitis auftreten s
der schweren Verantwortung nicht entziehen können, du
Medikation ein gut Teil zu dem bedauerlichen Ausgang
beigetragen zu haben.

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us dem klinischen Aufnahmebefund möchte ich nur kurz hervorn, daß eine verstärkte bis brettharte Spannung der Bauchdecken 5 Fällen angeführt ist. Daneben bestand gleichzeitig im Fall 3 tark eingezogenes, kahnförmiges Abdomen, aus dem Magen wurde dem Schlauch fäkulenter Mageninhalt entleert, trotzdem ist anatisch das vorher Erbrochene nicht als fäkulent angegeben.
leteorismus bestand in mehr oder weniger ausgeprägtem Grade Fällen. Ein Ödem an der Haut des Abdomens rechts seitlich sich im Fall 6, Ödem an beiden Fußgelenken bestand im Fall 13. Flexionsstellung beider Beine fand sich allein im Fall 7. Bech der Ätiologie derselben verweise ich auf das oben Gesagte. iegend kostaler Atemtypus wird hervorgehoben in 4 Fällen allein, utlich dürfte derselbe sich jedoch auch in einem Teil der anderen gefunden haben, da er ja bei allen stärkeren Affektionen des n Peritoneums nur selten zu fehlen pflegt. Bezüglich des Urins im Fall 10 angegeben, daß derselbe Albumen und Indikan entim Blute wurden bei dem Fall 56000 Leukozyten gezählt. Ebenndikan im Urin wurde auch im Fall 13 gefunden.
enden wir uns nun dem pathologisch-anatomischen Befund er Fälle zu, so empfiehlt es sich wohl zunächst, die innerhalb ersten 48 Stunden operierten und geheilten Fälle gesondert zu echen.
n 1. Fall zeigte sich der Processus sehr verdickt, aufgebläht, mit kleinen Perforation an der Basis. In der Umgebung dünnges, leicht getrübtes Exsudat, ebenso eine reichliche Ansammlung lben im Douglas. Das Netz zeigte in der Umgebung des Appenreichliche Adhäsionen mit dicken, fibrinösen Beschlägen, ebene fanden sich am Cöcum.
n 2. Fall war der Processus in der Richtung nach hinten oben ckt und in der Psoasgegend fixiert. In der Mitte war er vollig durchgebrochen. Im freien Peritoneum reichlich seröses, eitrig btes Exsudat, ebenso im Douglas. Das Cöcum war infiltriert, eicht rauher Serosa.
n 3. Fall war der Processus ebenfalls nach oben umgeknickt, sehr gelegen und in frische und ältere Verwachsungen eingebettet. unterhalb der Spitze eine große Perforation, daneben zwei kleine eine gelegen. Unter der Leber, sowie nach dem Becken zu eine e Ansammlung eines serös-eitrig getrübten Exsudats, das sich eringerer Menge auch zwischen den freien Darmschlingen vort.
n 4. Fall freies trüb-gelbliches Exsudat im Peritonealraum. Serosa Därme in der rechten Beckengegend injiziert, daselbst reichlich

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Verwachsungen mit dicken grau-gelblichen Belägen. Proc
frische Verklebungen eingebettet, bleistiftdick, in der Mitte ei
Perforation.

Im 5. Fall war der Processus nach unten vollständig al
in Verwachsungen eingebettet und zeigte in der Mitte ei
Perforation. Der Inhalt des Processus bestand aus Eiter.
Umgebung des Processus fand sich wenig dünnflüssiger Eiter
den vereinzelt verklebten Darmschlingen, der mit der offene
nealhöhle in breiter Verbindung stand.

Im 6. Fall fand sich ein trübseröses Exsudat in der link
Bauchhöhle. In der Ileocöcalgegend war das parietale Pe
mit Darmschlingen verklebt, nach deren Lösung sich aus d
Peritoneum serös-eitrige Flüssigkeit entleerte, nach unten zu
lich verfärbtes Gewebe mit 100 ccm stinkenden Eiters. In di
szeß lag der perforierte gangränöse Appendix mit einem
Aus dem kleinen Becken entleerte sich ebenfalls stinken
Dieser Fall kam als der einzige von sämtlichen relativen F
tionen zum Exitus an fortschreitender Peritonitis, die auch
Sektionsbefund bestätigt wurde.

Im 7. Fall endlich fand sich ebenfalls ein trübes Exsud
freien Bauchhöhle, sowie mehrere Abszesse zwischen leicht v
Darmschlingen. Der Appendix war ohne frische Erscheinu
härent und lag in einer anscheinend alten Abszeßhöhle
Cöcum und Dünndarmschlingen. Der Fall entsprach einer
perforation eines alten latenten perityphlitischen Abszesses.

Allen Fällen gemeinsam ist also außer im 7. Fall eine P
des Processus, ferner ein seröses, mehr oder weniger in
zur Eiterung begriffenes Exsudat der freien Peritonealhöhle,
dieser in breiter Verbindung stehend, und schließlich di
Injektion und Trübung der Darmserosa, stellenweise mit
Beschlägen. In sämtlichen Fällen wurde der Processus ent
die Bauchhöhle unter Verkleinerung der Inzisionswunde mit J
gaze dräniert. Alle Patienten gelangten in einem Zeitraum v
26 Tagen zur Heilung ohne Fistel, mit Ausnahme des Fall
dem nach 20 Tagen noch die Inzision eines Douglasabsz
rectum notwendig wurde. Kotsteine wurden allein im Fall
gefunden. In ersterem Fall wurde wegen seines reaktionslo
laufes 4 Tage post op. die Sekundärnaht gemacht, die dann
Heilung gelangte.

Besprechen wir in gleicher Weise nach dem Zeitpunkt
ration geordnet die weiteren 20 Fälle, so finden wir bei
3. Tage zur Operation gelangten Fällen folgenden Status:

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Fall 6. Freies jauchig-eitriges Exsudat mit abgegrenzten Eiter- im Peritoneum und reichlichen Verklebungen. Processus nicht en. Die Sektion am 3. Tage post op. ergab eine Perforation Spitze des Processus, daneben einen erbsengroßen Kotstein em abgekapselten Abszeß.

Fall 12. Im freien Peritoneum seröses, gallig gefärbtes Exsudat. inen Becken grünlich-gelber, mißfarben-jauchiger Eiter. Sämtärme gebläht und intensiv gerötet. Processus nach dem kleinen zu abgeknickt, an der Basis eine bohnengroße und an der zwei erbsengroße Perforationen.

Fall 14. Freies fäkulent-trübes Exsudat im Abdomen, diffuse se Darmverklebungen. Im kleinen Becken stinkender Eiter. sus in Verwachsungen eingebettet, fingerlang, mit der Tube t, entzündlich verdickt. Breite Perforation in der Mitte, ein großer Kotstein im Processus. Ein zweiter Stein frei im liegend. Einige Dünndarmschlingen schwarzbraun, gangränös. ocessus wurde amputiert. Bereits einige Stunden post op. trat itus ein.

Fall 15. Eitrig-dünnes Sekret in reicher Menge im Peritoneum, uflagerungen am Cöcum und Dünndarm. Kein Abschluß gegen ie Bauchhöhle. Im Becken eine große, jauchig-stinkende Eiterhlung. Prozessus nicht gefunden. Nach 3 Tagen Exitus.

Fall 17. Trübes Exsudat im freien Abdomen, Darmserosa mit , in deren Wand der Processus mit einer Dünndarmschlinge hsen ist, die um 120° gedreht ist. Processus amputiert, Exitus. reies seröses Exsudat, leichte Injektion der freien Dünndarmen und des Cöcum. Appendix ohne Perforation ins kleine geschlagen, mit leicht injizierter Wand. Bei der Lösung reißt tze ab, worauf sich serös-eitrige Flüssigkeit aus seinem Lumen t. Nach 24 Stunden Exitus. In dem makroskopisch klaren t Bacterium coli: Exitus durch Endotoxinwirkung.

n der freien Bauchhöhle fäkulente Exsudatmassen, Appendix efunden. Exitus 2 Stunden post op. Die Sektion ergab eine nperforation des gangränösen Appendix mit mangelhafter Adbildung, davon ausgehend diffuse, fäkulente Peritonitis.

Fäkulent-eitrige Peritonitis der rechten Bauchseite, ausgehend m tief im Becken perforierten Appendix, serös-eitrige Periton übrigen Abdomen. Es ist dies einer der beiden noch nach nden geheilten Fälle, der Patient konnte nach 5 Wochen entwerden.

ch hier finden wir in sämtlichen Fällen, außer im Fall 6, ein Exsudat im freien Peritoneum, jedoch bereits mit mehr eitrig-

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jauchigem Charakter, sowie größere Ansammlungen von fä
Eiter im Douglas. Die Darmschlingen zeigen in ausged
Maße starke, fibrinöse Beschläge und mehr oder weniger aus
Verwachsungen, so daß es bereits zur Abkapselung von
und größeren Höhlen gekommen ist, die aber in allen Fälle
freien Bauchhöhle kommunizieren. In 1 Fall (14) ware
gangränöse Veränderungen einiger Darmschlingen vorhan
vielleicht auf Ernährungsstörungen durch eitrige Gefäßve
zurückzuführen sind. Wir sehen hier deutlich die Kennzei
weiter fortgeschrittenen Peritonitis.

Der Processus wurde in 3 Fällen nicht bei der Oper
funden infolge bereits vorhandener Verklebungen, die ma
der Infektionsgefahr nicht lösen wollte. In 2 Fällen davon
nachträgliche Sektion eine Perforation an der Spitze und
einen freien Kotstein.

In den 5 anderen Fällen wurde der Processus gefunden
fernt. In 3 Fällen davon war eine Perforation an dem Appe
handen, neben der sich in 1 Fall 2 Kotsteine fanden, von de
innerhalb, der andere außerhalb des Processus gelegen war

Am 4. Tage wurden 3 Fälle operiert.

1. Fall 11. Fäkulenter Eiter im freien Peritoneum, Ap
der rechten Fossa iliaca fixiert, zeigt eine bohnengroße, g
Stelle mit Perforation, im Douglas reichlich Eiter. Stellenwe
Membranen auf der injizierten Darmserosa. Die Sektion er
freiem Eiter zahlreiche, kleine Abszesse und vergrößerte Me
drüsen.

2. Fall 9. Stark geblähte und injizierte Dünndarmschl
freiem getrübtem Exsudat. Ein Appendix epiploic. der Flexur
zieht nach hinten oben und ist an der Radix mesenter. fixie
dahin zieht der in Verwachsungen eingehüllte Appendix.
Basis in den Adhäsionen ein kleiner Eiterherd. Dicht unte
Klappe eine Schnürfurche. Die Sektion ergab neben alten
tischen Verwachsungen und einem vernarbten Geschwür de
klappe ein hämorrhagisches Infiltrat der Dünndarmwand und
Peritonitis.

Der letzte Fall bietet kein klares Bild infolge der frühe
auf der Basis der Pylorusaffektion entstandenen Peritonit
hat dann wohl durch Strangulationen und Knickungen zu eine
Affektion des Appendix geführt, von dem die zweite tödliche
ihren Ausgang genommen hat. Für die letztere Annahme sc
wenigstens der an der Basis des Processus gelegene kleine
zu sprechen.

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reies trübseröses Exsudat mit diffus injizierten Dünndarm-
n. Stärkere grünlich-gallertige Exsudatmassen führen von
rch das Becken nach rechts in die Ileocöcalgegend. Appendix
l, im Bogen nach medial ziehend, perforiert, in einem Ab-
egen. Die Dünndarmschlingen zum Teil stark gebläht, zum
abiert. Zwischen beiden Abschnitten zieht ein $1^1/_2$ cm breiter
g hinweg, der nach rechts unten fixiert ist.
s 15 Tage post op. Autopsie verweigert. Der Fall weist
vorhergehenden insofern eine gewisse Ähnlichkeit auf, als
Kombination von Perforationsperitonitis mit Strangileus dar-
l der Netzstrang wohl als Residuum einer vorausgegangenen
ndizitis aufzufassen ist.
en 3 Fällen also ebenfalls freies eitriges Exsudat, nur im 1.
all eine Perforation, dagegen im 2. und 3. Fall die Residuen
heren Peritonitis als Ursache der letzten Erkrankung.
. Tage nach der Perforation gelangten nur 4 Fälle zur Ope-

l 5. Dünner fäkulenter Eiter im freien Peritoneum, fibrinös-
eläge der Darmschlingen mit reichlichen Verklebungen. Pro-
it der hinteren Wand des Cöcum verwachsen, an der Spitze
engroße Perforation. Im Douglas eine Menge eitrig-fäkulenten
. Vor dem zentralen Ende im Processus ein Kotstein.
ll 16. Im Abdomen ein freies, trübes Exsudat, rechtsseitlich
ße in Abkapselung begriffene Eiterhöhle, Därme mit dünnen
Processus gangränös und perforiert.
eies eitriges Exsudat mit Belägen und Verklebungen der
lingen. Processus an der Spitze kolbig aufgetrieben, per-
nd gangränös.
rös-eitriges Exsudat im freien Peritoneum, Appendix nicht
. Exitus nach 24 Stunden. Sektion verweigert.
en 4 Fällen bestand also ebenfalls freies eitriges Exsudat neben
rforation des Processus in 3 Fällen, einmal mit einem Kot-
Inneren des Processus. Nur der 2. Fall kam zur Heilung,
noch eine zweite Laparotomie oberhalb des Nabels vor-
en war zur Lösung einer Darmschlinge, welche durch perito-
Verwachsungen Ileus verursachte.
lich dünnflüssiger Eiter mit Fibrinflocken vermischt fand sich
dem am 6. Tage operierten Fall 7. Der Processus wurde
funden. Die Sektion ergab diffuse Verklebungen der Darm-
n, eine schmierige, gelb-grünliche Verfärbung des Peritoneums,
e, multiple Abszesse mit eitrig-kotigem Inhalt. Processus am
gelegen, fast völlig zerstört. Zahlreiche Perforationen am

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Dünndarm, Cöcum und Kolon, die wohl als Rückperforatio
zufassen sind. Das in diesem Fall vorausgegangene Koterbr
nach dem obigen Befunde erklärlich.

Besonderes Interesse bietet der am 7. Tage zur Operation
Fall aus der Breslauer Klinik, insofern man zunächst ei
kommenen, akuten Obturationsileus mit Sitz der Obturation a
darm angenommen hatte, ohne Peritonitis. Die Operation e
wenig trübes freies Exsudat im Peritoneum mit blaurot
Darmschlingen. In der Ileocöcalgegend eitrig belegte Darmp
Form eines Darmwandbruches, Dünndarmschlingen im kleine
kollabiert. Spontane Eröffnung eines rechtsseitigen stinkenden
abszesses, in welchem der an seiner Spitze gangränöse u
Kotstein aufweisende Appendix fixiert war am Mesenteriu
Dünndarmschlinge. Es handelte sich demnach um einen
Kombination eines rein mechanischen Ileus mit Appendiz
welchem der Darmverschluß eventuell in der Weise zust
kommen war, daß durch den an seiner Spitze fixierten Appe
Dünndarmschlinge abgeschnürt sein konnte. Wahrscheinli
früher einmal bereits eine Appendizitis bestanden (Kotstein),
die Einklemmung der Dünndarmschlinge zu neuem Auffla
bracht wurde. Der Fall starb wenige Stunden nach der O

Die restierenden 3 Fälle sind erst am 9., 14. und 35.
Operation gelangt und nehmen daher eine besondere Stellu
die obigen bis zum 6. Tage operierten Fälle ein. Sie bie
eigentlich das Bild der akuten allgemeinen Peritonitis dar,
mehr das Bild einer Peritonitis fibrinopurulenta progredie
Mikulicz, zum Teil übergehend in das Bild der akuten, all
Peritonitis.

So ergab im Fall 13 die Operation am 9. Tage an der S
Processus eine Perforation, sowie einen bis in den Douglas re
Abszeß, der mit einem großen Abszeß der linken Lumbalgeg
munizierte. Der Processus war nach unten umgeschlagen.
nachfolgende Sektion des kurz nach der Operation vers
Patienten ergab neben der zirkumskripten die gleichzeitig be
allgemeine Peritonitis.

Ein ähnliches Bild ergab auch die Operation am 14.
Fall 10. Es bestand eine große, von trocknen, roten, fibrinös
und verklebten Darmschlingen gebildete Höhle mit dickem, fä
Eiter. Ein Gang führte von hier aus nach hinten oben in die
gegend, ein anderer nach vorne und unten zwischen Nabel u
physe. Daselbst zwei Gegeninzisionen angelegt. Sonst übe
klebungen der Därme, die auch ein Auffinden des Processus

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chten. Die Sektion nach 24 Stunden ergab eine Periappendicitis
tiva, Pelveoperitonitis chron., Peritonitis diffusa acuta purulenta
lpingitis sinistra.
chronische, eitrige Peritonitis mit abgeschlossenen intraperi-
multiplen Abszessen ergab schließlich die Operation am
e nach Beginn der Beschwerden im Fall 8. Die Spitze des
ix war in die Bauchhöhle und nach Verwachsung mit dem
in das letztere perforiert. Ferner bestand eine Rückperforation
n und Magen. Der Fall wurde noch dadurch kompliziert, daß
rativer Pneumothorax links mit Atelektase der linken Lunge
sektion der linken 11. Rippe notwendig machte.
tliche drei letzten Fälle könnten somit nach einem Teil des
s ebensogut unter den zirkumskripten Periappendiziten ange-
erden, wenn eben nicht die gleichzeitig vorhandene allgemeine
tis ihnen diesen gegenüber eine besondere Stellung zuweisen
In allen drei Fällen lag ebenfalls eine Perforation des Appen-

eigentliche Zeitpunkt der Perforation läßt sich hier natur-
bei der langen Dauer der Erkrankung aus den Journalen nicht
herheit ersehen, so daß der angegebene Zeitraum sich mehr
Beginn der Beschwerden als auf den Zeitpunkt der Per-
beziehen dürfte. Im übrigen stimmen aber die anatomisch-
gischen Veränderungen der einzelnen Fälle mit dem angegebe-
traum auch bezüglich der Möglichkeit einer so lange bestehen-
foration so ziemlich überein.
zusammengefaßt finden wir also in 21 Fällen eine Perforation
klich angegeben, dagegen wurde ein Kotstein nur in 7 Fällen
n. Im freien Peritoneum finden wir bis zum 3. Tage in allen
ein mehr seröses, leicht getrübtes Exsudat, das vom 3. bis
eine deutlich eitrig-jauchige Beschaffenheit annimmt und zu
lungen im Douglas Neigung hat. Gleichzeitig eine fort-
nde Verklebung der Darmschlingen auf der Grundlage einer
en Ausscheidung des Exsudates. Vom 9. Tage ab entweder
l einer langsam fortschreitenden fibrinösen Peritonitis oder
rkumskripten Periappendizitis mit sekundärer Perforation in
e Bauchhöhle und nachfolgender akuter Allgemeinperitonitis.
Therapie bestand bei sämtlichen Fällen in breiter Eröffnung
itonealhöhle durch Medianschnitt, Schrägschnitt oder Schnitt
ralen Rektusrande, zu dem nach Bedarf eine Gegeninzision in
nbalgegend oder auch im Douglas hinzugefügt wurde. Die
erfolgte stets in ausgiebiger Weise mit Jodoform- bzw. Iso-
e und bisweilen gleichzeitig mit einem dicken, bis in den

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Douglas eingelegten Glasdrän. In Fällen mit dickem eitrig sudat wurde eine ausgiebige Spülung der Abdominalhöhle mit logischer Kochsalzlösung vorgenommen. Stark kollabierte P mit darniederliegender Darmtätigkeit erhielten zur Anregung de reichlich Kochsalzeinläufe per rectum. Die Narkose wurde du mit Äther gemacht und nur auf eine besondere Indikation seit Lungen wurde ausnahmsweise einmal Billrothsche Lösung ve

Kommen wir zu den absoluten Frühoperationen, d. jenigen Fällen, die bereits 48 Stunden nach Beginn der Erk überhaupt ohne Rücksicht auf das Fehlen schwerer Sympto Operation gelangt sind, so ist deren Zahl verhältnismäßig ge einerseits erst im Jahre 1903/04 mit der Frühoperation be wurde, und andererseits die Zahl der Fälle, welche so früh in die Hand des Chirurgen gelangt, an sich schon eine s schränkte ist. Diese letzte Tatsache wird sich vermutlich sobald auch die ausschlaggebenden Kreise der praktischen Ärz und mehr von dem Nutzen der Frühoperation durchdrunge wie es z. B. von Sprengel aus der Umgebung von Braun und von Riedel aus der von Jena heute schon hervorgehobe

Wir haben es daher im Laufe der Jahre 1903—1907 nur Fällen von absoluter Frühoperation zu tun, die sämtliche g Heilung gelangt sind.

Es handelte sich dabei um 16 = 59,2 % männliche und 11 = weibliche Patienten im Alter bis zu 40 Jahren. Davon entfa das erste Dezennium 1 Fall, auf das zweite Dezennium 8 Fä das dritte 11 und auf das vierte 7 Fälle.

In 18 Fällen davon wird der Anfall als der erste angege 6 Fällen war bereits ein anderer Anfall vorausgegangen und 2 Fällen handelte es sich um den dritten, sowie in einem F den vierten Anfall.

In sämtlichen Fällen wurde der Processus leicht gefund entfernt.

Die Behandlungsdauer lag zwischen 12—20 Tagen in 7 zwischen 21—30 Tagen in 2, und zwischen 31—42 Tagen e in 2 Fällen.

Ein operativer Eingriff war vorausgegangen allein im Fall vor 2 Monaten bei dem ersten Anfall eine Inzision und Drän Douglas durchgemacht hatte. Seitdem hatte der betr. Patien an Blasenbeschwerden gelitten, wohl als Folge der entzü Adhäsionen vom ersten Anfall her.

Von sonstigen Angaben aus der Anamnese sei hier einiges gehoben, was allgemeineres Interesse bietet.

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Temperatur wird als fieberhaft vor und bei der Aufnahme
en in 20 Fällen. In 3 Fällen wird ein Schüttelfrost angeführt.
len bestand kein Fieber und in 3 anderen Fällen ist weder
Anamnese, noch im Status eine Angabe über die Temperatur
en.

chen ging voraus in verschiedener Intensität und Häufigkeit
illen, während in den anderen 11 Fällen nur von Übelkeit
des Anfalles berichtet wird.

chmerzen begannen mehr weniger heftig in der Magengegend
ganzen Leibe und lokalisierten sich dann schnell in der Ileo-
end. Vereinzelt werden sie gleich bei Beginn in der Gegend
endix empfunden.

ll 5 hatte Patient noch an dem Tage nach Beginn des An-
längere Wagenfahrt unternommen, worauf denn auch prompt
nelle Verschlechterung seines Zustandes eintrat. Im Fall 8
ient nach Beginn des Anfalls noch die Schule besucht, worauf
sten Tage eine rapide Verschlimmerung der Schmerzen und
Erbrechen bei jeder Nahrungsaufnahme auf die Schwere des
hinwies.

ler Aufnahme bestand deutlicher Meteorismus und Span-
r Bauchdecken in 20 Fällen, in einem Fall wird die letztere
art bezeichnet, Blasenkrampf war nur einmal vorhanden.
Druckempfindlichkeit wird als allgemein angegeben in
in 24 Fällen war dieselbe besonders in der Ileocöcalgegend
t, in einem Fall war eine stärkere Druckempfindlichkeit über-
cht vorhanden.

deutliche Resistenz in der Ileocöcalgegend ließ sich in
nachweisen, dieselbe schwankte zwischen Hühnerei- bis
stgröße, und nur in einem Fall wird dieselbe als bleistift-
rang angegeben. In 2 Fällen ließ sich die Resistenz gleich-
m Rektum aus nachweisen. In 2 anderen Fällen fehlte jede
nachweisbare Resistenz.

an wurde im Urin nur im Fall 10 gefunden. In einem Fall
gleichzeitig eine urethrale Gonorrhöe vorhanden.

der Durchsicht der anatomisch-pathologischen Operations-
dieser Kategorie werden wir öfters finden, daß die patho-
Veränderungen weit mehr fortgeschritten sind, als man nach
en Zeit seit dem angeblichen Beginn des Anfalls erwarten
Die Veränderungen entsprechen mit anderen Worten einem
Stadium der Erkrankung, als dem, welches wir nach den
en Angaben des Patienten vor uns haben. Dieses erklärt
e weiteres aus der schon oben hervorgehobenen Erfahrungs-

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tatsache, daß eben die klinischen Erscheinungen bisweilen i
Gegensatz zu den pathologischen Veränderungen stehen kön

Die subjektiven Beschwerden können an sich schon ge
und werden außerdem noch je nach Sensibilität des Patie
schieden stark empfunden werden. Sie können daher name
Beginn der Erkrankung der Beobachtung völlig entgehen und
dem Patienten erst bei stärkerem Auftreten zur Wahrnehm
von diesem Augenblick naturgemäß den Beginn der E
rechnen wird.

Andererseits kann auch eine virulentere Infektion inner
zerer Zeit zu schweren anatomischen Veränderungen führen,
bei einer weniger virulenten Infektion der Fall sein wird.

Kurz zusammengefaßt stellen sich nun die Operationsbef
den Krankengeschichten folgendermaßen dar:

1. Processus in alte und frische Verwachsungen eingebe
dickt. Er ist nach außen und unten umgeschlagen, die Wan
infiltriert und brüchig. Im Douglas trübseröses Exsudat. Die
haut des Processus zeigt grauweißliche, diphtherische Beläg

2. Retention an der Spitze des Processus, keine Verwa
dagegen Hämorrhagien der Schleimhaut. Der Processus
lang, nach hinten und oben umgeschlagen mit starker Schwe
Wand. An der Basis eine Striktur infolge einer alten Na
Peritonealhöhle ist frei von Exsudat.

3. In der rechten Darmbeingrube wenig seröses Exsuda
Reinkultur Streptokokken enthält. In einem zusammengeball
klumpen der 10 cm lange, dicke und hochrote Processus. Pe
parietale der hinteren Beckenwand stark injiziert, Mese
ödematös verdickt.

4. Processus frei nach dem kleinen Becken ziehend, ver
rötet und geschwollen. Nahe der Spitze eine fünfpfennigst
gangränöse Stelle. Eiteransammlung im kleinen Becken. Ei
Abszeß unterhalb des Nabels nach links zur Mittellinie zieh
klebung der Därme am Peritoneum parietale.

5. Ziemlich reichliches, leicht getrübtes Exsudat im fr
domen, besonders im kleinen Becken angesammelt. Der en
veränderte Processus wird von dem zu einem Klumpen ver
Netz in der rechten Beckenschaufel umschlossen. Darin ei
mit braunem, stinkendem Eiter. Am Processus eine m
große Gangränstelle, dahinter ein Kotstein in Kirschkerngrö

6. Processus stark gerötet an der Spitze im kleinen Beck
Die Serosa ist mit kleinen Knötchen besetzt, Mesenteriol
ödematös geschwollen, mit einem großen Fibrinfetzen. In

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eine geringe Menge nicht eitrigen Exsudates. Im Lumen des
us Eiter und Stuhl.
rocessus nach oben geschlagen, in Netz eingebettet. An der
ine erbsengroße Perforation, die ganze Basis brüchig mit
nder Gangrän. Processus stark aufgetrieben, in der Wand
Eiterherde. Blutig-schleimiger Inhalt, ausgedehnte Nekrosen.
s, seröses, freies Exsudat. Neben dem Processus in dem um-
n Netzklumpen ca. ein Teelöffel voll Eiter. Obliteration des

eritoneum ödematös, verbackene Darmschlingen, ein Netz-
bedeckt das kleine Becken, unter demselben reichlich fäku-
iter. Processus in der Abszeßhöhle gelegen, ringförmig ge-
an der Spitze eine linsengroße Gangränstelle, dahinter ein
großer Kotstein.
itze des geschwollenen Appendix nach oben geschlagen, gegen
er zu verwachsen. Basis des Appendix bis auf Pflaumen-
ufgetrieben, Wandung stark injiziert. Kein Exsudat, keine
schläge, Cöcalwand sehr brüchig, so daß die Nähte durch-
n. Das Mesenteriolum wird auf den Stumpf genäht. Nahe
e eine Striktur. Schleimhaut an der dilatierten Stelle gangränös.
n freien Peritoneum reichlich getrübtes, nicht fäkulentes Ex-
as sich besonders reichlich im kleinen Becken vorfindet und
en deutlich fäkulenten Geruch annimmt. Processus in der
es Nabels zwischen Netz und Dünndarmschlingen frisch ad-
Bei der Auslösung Entleerung von fäkulentem Eiter aus
n den Appendix gelegenen Abszeß. Kolonwand ist infiltriert.
iefere Muskelschichten, Faszie und Peritoneum infiltriert,
s. Netz zu einem Klumpen verbacken, vielfach mit Abszessen
zt. Resektion des Netzlappens. Zwischen Cöcum und Netz-
eine Abszeßhöhle, die durch das Netz in die Bauchhöhle
rochen ist. In der Bauchhöhle und im Douglas freier Eiter.
osa spiegelnd. Processus 6 cm lang, an der Basis stenosiert,
ein Empyem mit 15(!) flachen, linsengroßen Kotsteinen. Die
g des Processus ist trotzdem nirgends perforiert, nicht einmal
Stelle ulzeriert.
Venig freies seröses Exsudat, vorliegend injiziertes Netz, dar-
jiziertes Cöcum. Appendix nach hinten unten gelegen, in
ien eingebettet, mit geschrumpftem Mesenteriolum.
orliegend infiltriertes Netz, nach rechts unten fixiert. Appen-
eknickt nach hinten oben, stark verändert durch alte und
Entzündung, basaler Teil verdickt. Unter dem infiltrierten
nflüssiger Eiter.

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14. Vorliegend Adhäsionen. Appendix nach oben auße
giert, injiziert, 13 cm lang, mit beginnender Perforation in
15. Cöcum vorliegend, Appendix frei, 6 cm lang, ins kl
geschlagen, im ersten Stadium der Entzündung. An eine
gräuliches Infiltrat der Wandung des Appendix, im basale
ein Kotstein.
16. Vorliegend Netz und Dünndarm, im Becken se
nicht stinkendes Exsudat. Appendix leicht verklebt, nach
schlagen, purpurrot, kleinfingerdick, mit weißen Fibrinfet
Perforation. Inhalt des Appendix eitriger Kot. Mesent
filtriert.
17. Cöcum vorliegend, nicht injiziert. Processus ret
Hinterwand des Cöcum breit aufliegend und hier fest
stark geschwollen und gerötet. Im Lumen ein langer,
roter Thrombus, zu einer Stelle zwischen mittlerem u
Drittel führend, wo die Wand gangränös und minimal p
Im Becken serofibrinöses, nicht stinkendes Exsudat.
18. Leicht Exsudat im Becken. Appendix in alten,
häsionen, in der Tiefe fixiert, auf dem Cöcum festgewac
lang, enorm gebläht, injiziert, mit stinkendem Eiter und K
als Inhalt, eines davon Dekubitalgangrän verursachend, d
der Perforation steht.
19. Vorliegend Netz. Dem Cöcum anliegend injizierte
schlingen. Appendix zwischen Cöcum und Dünndarm
über die Iliacalgefäße ins kleine Becken ziehend, in Adhä
gebettet, mit eitrig infiltrierter Wand. Bei Lösung der
serös-eitriges Exsudat zwischen Dünndarmschlingen abge
freien Peritoneum wenig seröse Flüssigkeit.
20. Appendix geschwollen, 6 cm lang, leicht gewunde
knickt, Mesenteriolum infiltriert. Appendixserosa injiziert,
fibrinösen Auflagerungen. Netzverklebungen an der Vor
Appendix, sein distales Ende frei in der Bauchhöhle.
21. Cöcum vorliegend, mobil, injiziert. Leichte Adhä
schen Netz, Ileum und hinterem parietalem Peritoneum.
Lösung einige Tropfen stinkenden Eiters. Daneben App
cöcal, hochgradig infiltriert, in breiten Adhäsionen mit Pe
22. Keine Verwachsungen, Appendix lang, frei, nicht
der Spitze derb. Freies Peritoneum ohne Befund.
23. Eigroßes in verdicktes Netz eingebettetes Exsudat
dem Lig. Poupartii. Appendix im Exsudat, mit drohender
in der Mitte. Zwei Kotsteine. In den Adhäsionen zw
serös-eitriger Flüssigkeit.

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Appendix 7 cm lang, nach außen oben hinten am Cöcum liegend, , prall injiziert, in fibrinöses Exsudat eingebettet, an der Spitze ion. Im freien Peritoneum wenig seröse Flüssigkeit.

Appendix nach oben geschlagen, außen am Cöcum, fettes rium, fibrinös-eitriger Belag, wenig Serum im Peritoneum. us hochrot, geschwollen, ohne Inhalt mit nekrotischer Schleim- Mesenteriolum brüchig.

Appendix durch alte Adhäsionen retrocöcal fixiert, Wand infolge beginnender Gangrän, Peritoneum frei von Eiter, Gestank nach Bacter. coli.

Sofort nach Eröffnen des Peritoneums quillt stinkender, grün- iter heraus, ca. 2—4 Teelöffel voll. Vorliegend stark ge- Cöcum, injiziert im unteren Teil. Appendix nach hinten oben en, fest adhärent, stark gerötet. Auf der Grenze zwischen n und unterem Drittel eine große Perforation, aus der ein Kotstein herauskommt. Der Perforation entspricht ein tiefes In der Cöcalgegend stinkender Eiter.

obigen Befunden finden wir weder ein seröses Exsudat, ine Eiterung nur in 5 Fällen. In diesen Fällen waren die rungen vorwiegend auf den Processus beschränkt. Im Fall 2 nur eine Retention an der Spitze mit zahlreichen Hämor- der Schleimhaut und starker Schwellung der Wand. Trotz- tte der Fall mit Schüttelfrost eingesetzt und war bis zur n mit geringem Fieber verlaufen. Der Fall dürfte dem ent- , was Riedel unter einer Appendicitis granular. haemor- verstanden wissen will.

ll 9 waren die Veränderungen am Processus erheblich weiter ritten. Es bestand eine frische Verwachsung und eine Striktur Spitze, sowie eine Retention und Gangrän der Wand an der Bemerkenswert ist, daß, wie im Journal ausdrücklich hervor- wird, trotz dieser vorgeschrittenen Veränderungen kein Ex- d keine fibrinösen Beläge der Serosa in der Umgebung be-

Ob der erste Grad des Exsudats in Gestalt einer ödema- hwellung der Darmserosa vorhanden war, wie sie namentlich rengel hervorgehoben wird, ist aus dem Journal nicht zu

Die Ursache für die fehlenden Veränderungen in der Um- könnte man hier vielleicht in der Annahme erblicken, daß ine Thrombose bzw. Verlegung der Lymphgefäße am Pro- eine weitere Verbreitung der in ihm enthaltenen Toxine ver- wurde. Derartige Fälle pflegen dann im Fall einer Perforation ste Prognose abzugeben, da dem Peritoneum nicht durch das le Vorauseilen der Toxine eine Gelegenheit gegeben ist zur

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Vorbereitung von Schutzmaßregeln gegen die plötzliche In
Infektionserreger.

Einen fast normalen Befund bietet allein Fall 22 dar, be
es sich um einen langen, freien, nicht injizierten Appendix
der sich nur durch eine gewisse Derbheit an der Spitze au
Das Peritoneum war ohne Befund.

Erheblich fortgeschrittene Veränderungen am Appendix
Fall 9, fanden sich auch im Fall 20 und 26. Im ersteren
der Appendix geschwollen, seine Serosa injiziert, mit leic
nösen Auflagerungen, sowie Netzverklebungen an seiner V
Das Mesenteriolum war ebenfalls infiltriert. Peritoneum
Befund. Im letzteren Falle handelte es sich um einen durc
häsionen retrocöcal fixierten Appendix, dessen Wand infolge
der Gangrän bereits eine schwärzliche Verfärbung zeigte.
toneum war frei, zeigte aber einen auffallenden Geruch nach F

Sämtliche 5 Fälle repräsentieren bei dem Mangel ein
logischen Peritonealbefundes gewissermaßen das frühzeitigs
der gesamten 27 Frühfälle. Sie zeigen aber auch anderer
lich, in wie weiten Grenzen die Veränderungen am Appe
fehlender Peritonealreaktion sich bewegen können.

Ihnen am nächsten stehen die Fälle, bei denen wir ein
trübtes, seröses Exsudat im freien Bauchraum finden, ohne e
abgekapselte Abszeßbildung. Dazu gehören im ganzen 10

Im Fall 1 bestand ein trübseröses, nicht abgekapseltes E
Douglas. Der Processus war in reichlich alte und frische
sungen eingebettet, seine Wand stark infiltriert und brü
eben entstandene Exsudat hatte sich naturgemäß zunächst i
angesammelt.

Fall 3 ergab wenig seröses Exsudat auf der rechten
schaufel, ebenfalls mit reichlich schützenden Verwachsung
entzündlich veränderten Processus. Trotzdem das Exsud
kokken enthielt, heilte der Fall nach primärer Naht der Ba
völlig reaktionslos. Es bestand nur 2 Tage nach der
eine geringe Steigerung der Temperatur.

Ein geringes, freies seröses Exsudat im Douglas bestan
lich im Fall 6. Daneben fand sich ein leichtes Empyem
cessus und wenig frische Verwachsungen. Sanat. p. p.

Im Fall 12 war neben dem geringen Exsudat ein nur
änderter, in Verwachsungen eingebetteter Appendix mit gesc
Mesenteriolum vorhanden. Ebenso im Fall 16 leichte Ve
an dem geschwollenen und geröteten, mit Fibrinfetzen
Appendix. Das serofibrinöse Exsudat lag im Becken.

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im Fall 17 und 18 war das leicht getrübte Exsudat im Becken
nelt. Im ersteren Fall zeigte der stark geschwollene und
Appendix, welcher mit der hinteren Wand des Cöcum breit
en war, in seinem Lumen einen festsitzenden, roten Throm-
auf eine gangränöse und perforierte Stelle der Wand zwischen
und unterem Drittel hinführte. Im letzteren Fall war der
gleichfalls auf dem Cöcum adhärent, enorm gebläht infolge
seines Inhalts, der aus stinkendem Eiter und Konkrementen
Das eine derselben hatte eine Dekubitalgangrän verursacht,
vor der Perforation stand.
ll 19 zeigte der in Adhäsionen gelegene Appendix eine eitrig
e Wand und es fand sich neben dem freien auch abgekap-
sudat zwischen verklebten Dünndarmschlingen vor.
beginnende Perforation zeigte an seiner Spitze auch im Fall 24
verdickte und prall injizierte Appendix, während es sich im
n einen hochroten, geschwollenen Processus handelte, dessen
aut bereits nekrotisch war.
harakteristikum dieser Gruppe bilden also neben dem leicht
, serofibrinösen Exsudat, das sich mit Vorliebe im kleinen
nzusammeln pflegt, entsprechend dem fibrinösen Charakter
sudates beginnende Verklebungen und Verwachsungen in der
g des Entzündungsherdes, aus denen bereits deutlich das
des Körpers hervortritt, den Entzündungsherd von dem
Peritoneum auszuschalten.
en sind die pathologischen Veränderungen am Appendix
erheblich vorgeschrittener als bei der ersten Gruppe, in
von häufiger Retention des Inhaltes, Neigung zu Ulzeration
ose der Schleimhaut als Vorbereitung für die drohende bzw.
nde Perforation seiner Wandung.
ritte Stufe nach dem pathologischen Befund würden die Fälle
en, bei denen es neben einem freien Exsudat bereits zu einer
benen Abszeßbildung gekommen ist, bzw. bei denen es um-
nach älteren Entzündungsvorgängen durch eine neue Infektion
aus zu einem neuen freien Exsudat gekommen ist. Welche
beiden Möglichkeiten vorliegt, läßt sich aus den patologi-
funden nicht immer mit Sicherheit entscheiden.
pisches Beispiel für den letzteren Vorgang bietet der Fall 11.
em war der primäre Abszeß zwischen Cöcum und einem
en gelegen. Durch eine Perforation in die freie Bauchhöhle
zu einem sekundären, freien, eitrigen Exsudat gekommen.
appen selbst bestanden multiple Abszesse. Der Processus
den Abszeß hinein und zeigte an der Basis eine Stenose,

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hinter der ein Empyem mit 15 flachen, linsengroßen S
legen war.

Zu dem ersteren Vorgang dürften die Fälle 5, 7 und 10 zu z

Im Fall 5 bestand neben einem um den Processus vor
gekapselten, fäkulenten Abszeß ein freies, trübes Exsudat d
höhle mit einer Ansammlung desselben im Douglas. Am
selbst fand sich eine markstückgroße Gangrän mit einer
dahinter.

Im Fall 7 bestand ein geringes, seröses, freies Exsudat ne
kleinen Abszeß am Processus, sowie mehrere ganz kleine
in der Wand des Processus selbst. Ferner befand sich an
des Appendix eine Perforation und umschriebene Gangrän

Ebenso bestand auch im Fall 10 ein Abszeß mit fris
wachsungen um den Processus herum, sowie ein reichlich se
freies Exsudat mit einer größeren Ansammlung im kleine
Nur die letztere hatte einen fäkulenten Geruch.

Am weitesten von allen ist der Fall 4 vorgeschritten, der
Exsudat überhaupt nicht mehr zeigte, sondern nur zwei al
Abszesse aufwies, von denen der eine im Douglas, der and
halb des Nabels gelegen war. Der entzündlich veränderte
zog nach dem ersten Abszeß hin. Es bestanden ferner
Verwachsungen der Därme untereinander und mit dem P
parietale um die Abszeßhöhle herum.

Fall 13 zeigte unter dem infiltrierten Netz dünnflüssi
während der nach hinten oben abgeknickte Appendix durc
frische Entzündungsvorgänge stark verändert war und eine
am basalen Teil aufwies.

Fall 21 ergab wenig stinkenden Eiter bei Lösung der A
zwischen Netz, Ileum und hinterem parietalem Peritoneum.
gradig infiltrierte Processus lag retrocöcal in Adhäsionen
wies eine Perforation auf.

Ein eigroßes in verdicktes Netz eingeschlossenes Exsudat
im Fall 23. Der Appendix lag in dem Exsudat, enthielt zwe
nebst einer drohenden Perforationsstelle in seiner Mitte.
fanden sich zwischen den Adhäsionen noch zwei Teelöffel e
eitrigen Flüssigkeit.

Fall 17 endlich zeigte in der Cöcalgegend stinkenden Eite
der stark gerötete, nach hinten oben adhärente Appendix
mittlerem und unterem Drittel eine große Perforation au
welcher ein kleiner Kotstein im Begriff war herauszutreten

Schließlich möchte ich noch Fall 14 und 15 besonders
da bei ihnen mir eine Angabe über das Verhalten des P

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orliegt, so daß es zweifelhaft erscheint, in welche unserer drei
n von absoluten Frühoperationen man sie einreihen kann. Im
werden Adhäsionen angegeben, während der nach oben außen
igierte und injizierte Appendix in der Mitte eine beginnende
ion aufwies. Fall 15 zeigte den ins kleine Becken geschlagenen
ix im ersten Stadium der Entzündung. Im basalen Abschnitt
n lag ein Kotstein, an einer Stelle der Wand war ein gräulich
es Infiltrat vorhanden.

ieten diese wenigen 17 Fälle, die nach den subjektiven An-
er Pat. bereits in einem so kurzen Zeitraum nach Beginn des
zur Operation gelangt sind, objektiv bereits ein so mannig-
Bild der pathologischen Entwicklungsvorgänge dar, daß man
; die verschiedenen Stadien der Entwicklung daraus kon-
n kann. Sie bilden die beste Illustration für die weiten
, in denen sich die Differenz zwischen subjektivem Krank-
ühl und objektivem Krankheitsbefund bei der Appendizitis
m frühesten Stadium bewegen kann, und eine genauere Durch-
r vorstehenden Operationsprotokolle dürfte manchem Gegner
hoperation doch zu denken geben.

ersieht daraus, wie häufig man bei der Frühoperation der
e gegenüberstehen kann, daß man dem objektiven Befunde
ine Frühoperation, sondern eine Zwischenoperation zu machen
gen ist. Daß trotzdem unsere sämtlichen Fälle glatt zur
gelangt sind, dürfte zum mindesten nicht gegen den Nutzen
einer Frühoperation, noch einer Zwischenoperation sprechen.
kann sich eben nicht präzise auf eine Operation der Appen-
ach einem zeitlich allein umgrenzten Stadium festlegen und
auch mancher Gegner der Zwischenoperation dazu kommen,
rmaßen unabsichtlich eine Anzahl von Zwischenoperationen
em operativen Befund) zu machen, nach deren Verlauf sich
e Anschauungen der betreffenden Operateure auf Grund ihrer
wenn auch unfreiwilligen Erfahrungen zugunsten oder ungunsten
schenoperation modifizieren dürften.

Erfahrungen der radikaleren Operateure, namentlich die von
nd Sprengel, haben zur Genüge bewiesen, daß bei exaktem
onieren der übrigen Bauchhöhle ein Fortschreiten der In-
auf das freie Peritoneum auch bei Lösung frischer, ent-
er Adhäsionen nicht so sehr zu fürchten ist, als man es
n allgemein getan hatte. Geht doch Rehn auf Grund seiner
rfolge so weit, daß er prinzipiell abgekapselte Abszesse unter
ade des Peritoneums durch die freie Bauchhöhle hindurch

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Auf Grund dieser einwandsfreien Erfolge eines radikale
gehens darf man wohl annehmen, daß der Vorteil einer m
frühzeitigen Entfernung der Infektionsquelle aus dem Körper an
der verminderten Furcht vor der operativen Infektionsmöglich
freien Peritoneums auch die z. Z. noch ziemlich allgemein ver
Abneigung gegen die Zwischenoperation voraussichtlich mehr u
zum Schwinden bringen wird.

Die Therapie bestand neben der Amputation des Appen
in allen Fällen gemacht wurde, in primärer Naht der Bauchde
20 Fällen. Alle 20 waren völlig fieberfrei, bis auf Fall 3, der
kokken im Exsudat hatte und 2 Tage lang mit Fieber post op.
Teilweise Naht der Bauchdecken und Dränage der Peritone
mit Jodoformgaze und Gummi- oder Glasdrän wurde in 5
gemacht. Alle 5 heilten ebenfalls völlig fieberfrei p. p. bis
Dränageöffnung. Breite Tamponade ohne Naht wurde nur in
und 5 gemacht. In beiden Fällen war der Verlauf zuerst fie
Im ersten Fall kam es noch zu einer Retention, und im zwei
wurde 9 Tage nach der Operation die Inzision eines metast
Parotisabszesses links notwendig. Beide Fälle heilten dann g
granulationem, jedoch mußte der letztere Fall mit einer Ba
entlassen werden. Bei den übrigen Fällen ist von persisti
Fistelbildungen nichts erwähnt. Von postoperativen Kompli
seitens der Breslauer Fälle (12—27) sei noch besonders hervorg
daß in 4 Fällen ein kleiner Bauchwandabszeß eintrat, der die
nicht wesentlich verzögerte. Ferner kam in je einem Fall da
leichte Bronchopneumonie, eine trockne rechtsseitige Pleurit
Thrombose der Vena poplitea dextra. In einem anderen F
dem ein perforierter Appendix mit stinkendem Abszeß gefun
die Bauchhöhle trotzdem ohne Dränage geschlossen word
mußte nach 21 Tagen ein Douglasabszeß inzidiert werden,
vermutlich durch entsprechende Dränage bei der ersten O
hätte vermeiden lassen und daher nicht der Frühoperation als
zur Last gelegt werden kann. Zum Schluß mag noch einma
hingewiesen werden, daß das günstige Heilungsresultat dies
sich nicht wesentlich verschlechtert, selbst wenn wir die Fälle
nehmen, bei denen man in 48 Stunden nach Beginn der Besc
bereits vor einer Perforationsperitonitis stand, in denen
Frühoperation mit der Notoperation identisch wurde. Wir
denn eine Mortalitätsziffer von 2,9%.

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Nicht operierte Fälle.

z kurz können wir uns schließlich bei der letzten Gruppe von zur Operation gelangten Fällen fassen, die ja naturgemäß vom gisch-anatomischen, wie vom chirurgischen Standpunkt aus sonderes Interesse beanspruchen können.

Vollständigkeit halber möchte ich nur hervorheben, daß es z. des Geschlechts um 18 männliche und 19 weibliche n handelte. Dieselben verteilen sich auf die einzelnen Alters-en folgendermaßen: Von 10—20 Jahren 16 Fälle, von 21—30: 31—40: 4, von 41—50: 1, von 51—60: 1 und von 61—70 Jahren 1 Fall.

7 Patienten handelte es sich um 1, bei 5 Pat. um 2, bei 4 Pat. nd bei 1 Patienten um 4 vorausgegangene Anfälle.

äußeren Traumen wird ätiologisch nur einmal „schweres Heben" nal eine „Quetschung mit einem 50 Pfund-Gewicht" in der Ileo-end angeführt. Im Fall 2 ging eine lakunäre Tonsillitis voraus, wohl mit Recht in einen ursächlichen Zusammenhang mit der ven Appendixerkrankung bringen kann. Der betr. Patient sollte ur Operation wiederkehren, hat sich aber nicht eingefunden. n fieberfreien Verlauf zeigten im ganzen nur 10 Fälle. Ein niger großes Exsudat bzw. Infiltrat war nachweisbar in 15 Fällen, 5 mal in der Ileocöcalgegend und 1 mal im Douglas lokali-Schmerzhaftigkeit in der Ileocöcalgegend wurde 12 mal nach-n. In 5 Fällen war kein Exsudat nachweisbar. 7 Patienten erten die Operation und wurden entlassen, 1 konnte wegen tiger Furunkulose nicht operiert werden und ist später nicht iedergekommen, ebenso wurde 1 wegen florider Lues nicht

ganz besondere Stellung unter den fieberfreien Fällen nimmt 23 ein, insofern es sich bei diesem um eine „Periappen-ine appendice" handelte. Bei dem Pat. war nämlich vor nach zwei typischen Anfällen durch Geheimrat Garrè der ix entfernt worden. 7 Tage vor der Aufnahme hatten sich dem Pat. ganz spontan allmählich zunehmende Schmerzen leocöcalgegend eingestellt, die den gleichen Charakter hatten, früheren Schmerzanfälle, jedoch ohne Fieber und ohne Er-. Von dem behandelnden Arzt war bei Beginn der Erkrankung skopfgroßer Tumor der Ileocöcalgegend konstatiert worden, der zur Aufnahme in die Klinik um die Hälfte verkleinert hatte. tliche Fälle konnten als beschwerdefrei und relativ geheilt n werden.

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17 Fälle wurden mit Fieber eingeliefert. Dieselben konnten ebenfalls nach allmählichem Abfall des Fiebers in verschieden langer Zeit als fieberfrei und relativ geheilt entlassen werden außer Fall 2, der auf seinen ausdrücklichen Wunsch bereits nach 4 Tagen mit fieberhafter Temperatur entlassen werden mußte, und Fall 25, der nach 42tägiger Behandlungsdauer schließlich ad exitum kam. Der betr. Pat. hatte vor 18 Tagen eine Quetschung mit einem 50 Pfund-Gewicht in der Ileocöcalgegend erlitten. Seit 6 Tagen war plötzlich heftiger Leibschmerz mit Fieber aufgetreten, seit 2 Tagen bestand Stuhlverhaltung, seit 1 Tag häufiges Erbrechen. Der Pat. wurde mit einer Temperatur von 40,1° und einem kleinen, unregelmäßigen Puls von 120 Schlägen in der Minute eingeliefert. Rechts seitlich ließ sich eine Dämpfung bis zum Rippenbogen hinauf nachweisen, eine Resistenz war nicht mit Sicherheit zu fühlen, jedoch bestand starke Druckschmerzhaftigkeit. Der Patient verweigerte einen operativen Eingriff und die Sektion nach 42 Tagen ergab schließlich eine Periappendizitis mit Perforation, eitrige Peritonitis diffusa, zahlreiche Rückperforationen in Dünndarm und Cöcum. Ferner bestand eine Pneumonie beider Unterlappen mit frischen pleuritischen Adhäsionen und ein Tonsillarabszeß rechts.

Die Therapie bestand in allen Fällen in möglichst reizloser, flüssiger Diät, Prießnitz oder Eisblase auf den Leib, ferner hohen Einläufen, um die Darmtätigkeit anzuregen. Opium wurde stets fortgelassen, um eine unnötige Trübung des Krankheitsbildes dadurch zu vermeiden. Bei sehr großen Beschwerden wurde statt dessen Morphium in kleinen Dosen verordnet.

Eine Spontanperforation in den Darm trat im Falle 8 und 9 auf. In beiden Fällen erfolgte darauf ein Rückgang sämtlicher Erscheinungen unter weiterem afebrilen Verlauf. Eine Spontanperforation vor 1 Monat hatte im Fall 20 stattgefunden. Trotzdem bildete sich rechts seitlich wieder ein fieberhaftes Exsudat. Nachdem sich dasselbe unter klinischer Behandlung verkleinert hatte und Patient entfiebert war, wurde Patient ohne Beschwerden entlassen.

Pleuritis fand sich außer in dem oben erwähnten verstorbenen Fall 25 auch im Fall 9 auf der rechten Seite bei einem gleichzeitigen Exsudat rechts im Abdomen. Nach einem Monat erst trat Temperaturabfall und Rückgang der Erscheinungen ein.

Den Ausdruck relative Heilung habe ich für die nicht operierten Fälle absichtlich gebraucht aus der Erwägung heraus, daß man bei diesen Fällen nie wissen kann, ob und wieviel Anfälle später noch eingetreten sind, nachdem die betreffenden Patienten angeblich als geheilt entlassen sind. Als absolut ist eine Heilung der Appendizitis

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eben erst dann zu bezeichnen, wenn eine weitere rezidivierende Erkrankung des Appendix mit Sicherheit ausgeschlossen werden kann. Es ergibt sich von selbst, daß dieses nur möglich ist nach der Entfernung des Appendix auf operativem Wege.

Werfen wir zum Schluß einen kurzen Rückblick auf das Gesamtresultat unserer 550 Appendizitisfälle, so finden wir zunächst bezüglich des Geschlechts das Gesamtverhältnis der männlichen und weiblichen Patienten durchaus entsprechend den Ergebnissen anderer Autoren, wie folgende kleine Tabelle zeigt:

	männlich	weiblich
Garrè	62 %	38 %
Dannehl (Berlin, chirurg. Klinik)	72 „	27 „
Hoffmann (Heidelberg)	72,4 „	27,6 „
Sprengel	64 „	36 „
Hermes (Sonnenburg)	60 „	40 „

Es ergibt sich auch hier wieder die vorwiegende Beteiligung des männlichen Geschlechts an der Erkrankung, auf deren Gründe wir bereits oben näher hingewiesen haben.

Auf die einzelnen Altersdezennien verteilen sich die Kategorien unserer Einteilung folgendermaßen:

	Sicherheitsop.	Spätop.	Notop.	Frühop.	Nicht op.	Summa
1—10 Jahre	16	11	4	1	—	32
11—20 „	98	24	7	8	16	153
21—30 „	124	30	4	11	12	181
31—40 „	68	22	6	7	4	107
41—50 „	27	13	—	—	3	43
51—60 „	10	9	3	—	1	23
61—70 „	3	3	3	—	1	10
71—80 „	—	1	—	—	—	1
Summa:	346	113	27	27	37	550

Es ergibt sich hieraus entsprechend den einzelnen Kategorien auch für die Gesamtsumme der Fälle die höchste Frequenz in das zweite und dritte Altersdezennium fallend, während in den weiteren Dezennien ein schneller und ziemlich gleichmäßiger Abfall der Frequenzziffer erfolgt. Es entspricht dieser Zeitraum eben demjenigen Alter, in welchem die Patienten am häufigsten körperlichen Anstrengungen und Traumen ausgesetzt sind, in welchem sie auch hinsichtlich der Begehung von Diätfehlern, wie kaltes Trinken, Essen von unreifem Obst, vernachlässigte Regelung des Stuhles und Nichtbeachtung etwaiger Darmkatarrhe am unvorsichtigsten sind. Es liegt auf der Hand, daß

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die Folgen eines derartigen Verhaltens sich vornehmlich am Processus als dem prädisponierten Locus minoris resistentiae des Digestionstraktus äußern werden.

Eine kurze vergleichende Übersicht über die Zahl der vorausgegangenen Anfälle bei den einzelnen Kategorien dürfte folgende kleine Tabelle gewähren:

Zahl der Anfälle bei den	1	2	3	4	5	6	7	8	9	10	11	17	Summa
1. Sicherheitsoperationen	96	93	66	38	26	14	4	4	1		3	1	346
2. Spätoperationen . . .	93	12	4	2	1	1							113
3. Notoperationen. . . .	20	6	1										27
4. Frühoperationen . . .	18	6	2	1									27
5. Nichtoperierten . . .	27	6	3	1									37
Summa:	254	123	76	42	27	15	4	4	1		3	1	550

Man ersieht hieraus, daß die Zahl der vorausgegangenen Anfälle weitaus am höchsten ist bei den Sicherheitsoperationen, um bei den Spätoperationen schon erheblich abzunehmen und noch steiler bei den Not- und Frühoperationen abzufallen. Absolut beurteilt sind danach nahezu die Hälfte sämtlicher Fälle, nämlich 254, bereits nach, bzw. bei dem ersten Anfall zur Operation gelangt, eine wenig geringere Anzahl, nämlich 199 Fälle, sind nach dem zweiten und dritten Anfall operiert worden. Mehr als drei Anfälle sind also nur in 97 Fällen vorausgegangen, was für die Gesamtzahl einem Prozentsatz von nur 17,6% entsprechen würde. Es ist also die weitaus überwiegende Mehrzahl, nämlich 82,4% unserer Fälle relativ früh zur Operation gelangt.

Die Entfernung des Appendix konnte in sämtlichen Fällen, also 100% der Fälle, vorgenommen werden bei den Sicherheits- und Frühoperationen. Am seltensten konnte er gefunden und entfernt werden bei den Spätoperationen, nämlich in 22 Fällen = 19,8%. Die Begründung dafür bieten einmal die bei diesen Fällen mehr weniger weit vorgeschrittenen pathologischen Veränderungen, bei denen der Processus bereits größtenteils nekrotisch zerfallen sein kann. Andererseits kann der noch vorhandene Appendix vollständig eingebettet und umhüllt sein von den schützenden Verwachsungen, welche die Wandung der Abszeßhöhle bilden, so daß eine Auffindung desselben ohne eine Kontinuitätstrennung der schützenden Wand und die Gefahr einer Allgemeininfektion des Peritoneums nicht möglich ist. Daß der Processus wesentlich häufiger, nämlich in 74,1% der Fälle, bei der Notoperation gefunden und entfernt werden konnte, ergibt sich von selbst aus der Erwägung, daß diese ja in einem erheblich früheren Stadium

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vorgenommen wird, in dem weder die Veränderungen am Processus, noch die Verwachsungen in der Umgebung so weit vorgeschritten sind, daß sie die Auffindung und Entfernung des Processus erschweren könnten.

Über die Häufigkeit vorausgegangener Symptome und Komplikationen für die einzelnen Kategorien der gesamten 550 Fälle, habe ich folgende Tabelle zusammengestellt:

	Chron. Fälle	Spätfälle	Notfälle	Frühfälle	Nicht op.
1. Blasenbeschwerden . .	17(4,9%)	11 (9,7%)	2 (7,4%)	1 (3,7%)	—
2. Durchfälle . . .	20(5,8 „)	22(19,4 „)	3(11,1 „)	5(18,5 „)	—
3. Blut- oder Kotbrechen	2(0,6 „)	3 (2,7 „)	—	—	—
4. Schmerz im rechten Bein . .	2(0,6 „)	7 (6,2 „)	—	—	—
5. Pleuritis	3(0,9 „)	1 (0,9 „)	—	—	—
6. Venenthrombose	4(1,2 „)	—	—	—	—
7. Frühere Inzisionen . . .	17(4,9 „)	5 (4,4 „)	—	1 (3,7 „)	—
8. Spontanperfor. in andere Organe .	2(0,6 „)	7 (6,2 „)	—	—	2(5,4%)

Danach sind auf die Gesamtzahl der 550 Fälle berechnet frühere Inzisionen vorausgegangen in 4,2%, Spontanperforationen in Blase oder Darm gingen voraus in 2,0%. Ferner wurden Blasenbeschwerden angegeben in 5,6%, Durchfälle in 8,5%, pathologisches Erbrechen in 0,9%, Schmerzen im rechten Bein in 1,6%, Pleuritis in 0,4% und eine vorausgegangene Venenthrombose in 0,4%.

Eine vergleichsweise Betrachtung der Operationsbefunde am Processus ergibt folgende Tabelle, die sich jedoch nur auf die 374 Rostocker und Königsberger Fälle bezieht, da mir die anderen Zusammenstellungen der Breslauer Fälle nicht vorliegen:

	Chron. Fälle	Spätfälle	Notfälle	Frühfälle	Summa
Strikturen . .	57(24,5%)	—	—	3(27,3%)	60(17,2%)
Obliteration. .	41(17,6 „)	—	—	1 (9,1 „)	42(12,0 „)
Retention . . .	60(25,8 „)	—	—	7(63,6 „)	67(19,2 „)
Perforation am Processus .	33(14,2 „)	17(19,5%)	16(84,2%)	—	66(19,0 „)
Perforation am Darm . . .	3 (1,3 „)	2 (2,3 „)	—	—	5 (1,4 „)
Kotsteine und Fremdkörper	30(12,8 „)	13(14,9 „)	4(22,2 „)	3(27,3 „)	47(13,9 „)

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Auf die Gesamtzahl der 349 operierten Fälle verteilt, finden wir danach am häufigsten eine Retention des Inhalts im Processus vertreten mit 19,2%, ihr fast gleichkommend ist die Zahl der ausdrücklich angegebenen Perforationen am Processus mit 19,0%. Strikturen werden in 17,2% angegeben und eine Obliteration des Lumens wird in 12,0% der Fälle angeführt. Relativ selten, nämlich nur in 13,5% wurden Kotsteine konstatiert. Am seltensten fand sich eine Perforation am Darm in 1,4%, ebenso konnte nur in einem Fall = 0,3% ein Fremdkörper im Processus gefunden werden. Interessant ist ferner an der obigen Tabelle das Ansteigen der Frequenzziffer bei den Perforationen von 14,2% bei den chronischen Fällen, auf 19,5% bei den Spätfällen und schließlich auf den sehr hohen Prozentsatz von 84,2% bei den Notfällen. Dieses erklärt sich daraus, daß bei den Sicherheitsoperationen ein großer Teil der früheren Perforationen teils durch gute Vernarbung, teils durch überdeckende Adhäsionen der Beobachtung entgeht, gleichwie auch bei den Spätoperationen in den zahlreichen Fällen, in denen der Processus nicht zu finden ist, über das Vorhandensein der höchstwahrscheinlich vorausgegangenen Perforation eine Entscheidung vielfach nicht zu fällen ist. Es dürften demnach für die zweite Kategorie noch zahlreichere Fälle der Beobachtung entgehen als in der ersten Kategorie. Dagegen bildet für die Notoperation als solche die Perforation die wesentlichste Indikation zum operativen Vorgehen, sobald sie sich durch die ersten Reizerscheinungen des Peritoneums manifestiert, und wird daher in diesen Fällen selten oder nie der Beobachtung entgehen. Im übrigen ergibt sich schon aus der Schwere der Fälle, daß für die letztere Kategorie die Häufigkeit der Perforationen eine ganz besonders hohe sein muß, entsprechend unserem Ergebnis von 84,2%.

Eine ähnliche Steigerung der Frequenzziffer trifft auch für das Vorhandensein von Kotsteinen bei den einzelnen Kategorien zu. Jedoch bewegt sich dieselbe in wesentlich gleichmäßigerer Linie aufwärts, als bei den Perforationen, indem sie von 12,4% bei den chronischen Fällen auf 14,9% bei den Spätfällen ansteigt, um sich bei den Notfällen auf 22,2% und bei den Frühfällen schließlich auf 27,3% zu erheben.

Hier entspricht jedoch das Ansteigen der Frequenzziffer nicht wie bei den Perforationen der Schwere der Fälle, indem hier die Zahl bei den Frühoperationen am größten ist, während sie dort dafür am niedrigsten bzw. =0 ist.

Wir können hier vielmehr konstatieren, daß sich das Ansteigen der Frequenzziffer genau umgekehrt proportional verhält zu der Länge des Zeitraums, der seit Beginn der Erkrankung bis zur Vornahme

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des operativen Eingriffs verflossen ist. Da nun nach der Wahrscheinlichkeitsrechnung für alle 4 Kategorien der Prozentsatz des Vorhandenseins von Kotsteinen ein ziemlich gleichmäßiger sein dürfte, so scheint mir die obige Beobachtung sich aus der Annahme zu erklären, daß, je später der Zeitpunkt der Operation, die Steine um so eher der Beobachtung entgehen können, oder daß in der Zwischenzeit vielleicht eine Zersetzung und Verflüssigung der Kotsteine durch irgendwelche Fermente der meist zahlreich vorhandenen Leukozyten stattgefunden haben könnte. Für wahrscheinlicher und häufiger möchte ich den letzteren Vorgang halten, denn namentlich für die chronischen Fälle ist ein so häufiges Übersehen von Steinen doch wohl auszuschließen, daß gegenüber den Frühfällen eine Differenz von 14,9% zustande käme. In jedem Fall scheint mir dieses gleichmäßige Ansteigen der Frequenzziffer von Kotsteinen umgekehrt zur Entfernung der Operation vom Beginn der Erkrankung das Walten eines reinen Zufalles auszuschließen.

Die gleichzeitige Perforation am Darm, die an sich ja selten zu beobachten ist, weist bei den Spätfällen eine Steigerung auf 2,3% gegenüber 1,3% bei den chronischen Fällen auf, ist also bei den ersteren fast doppelt so häufig vertreten.

Über die Lokalisation der Affektionen am Processus gibt folgende Tabelle Aufschluß, für die jedoch in der Hauptsache nur die chronischen Fälle in Betracht kommen, da bei den anderen Kategorien nur vereinzelt nähere Angaben darüber vorhanden sind.

	Spitze	Mitte	Basis	
Chron. Fälle	11(33,3%)	4(12,1%)	8(24,2%)	Perforation.
Spätfälle.	7(36,8 „)	4(22,2 „)	4(22,2 „)	
Chron. Fälle	24(42,1 „)	17(19,8 „)	11(19,3 „)	Striktur.
Chron. Fälle	5(17,2 „)	1 (3,4 „)	—	Kotstein.
Chron. Fälle	11(26,8 „)	8(19,5 „)	19(46,3 „)	Obliteration.
Summa:	58(16,6%)	34 (9,7%)	42(12,0%)	

Eine Obliteration des Appendixlumens in toto fand sich in 3 Fällen =0,9% der operierten Fälle insgesamt.

Man ersieht aus dieser kurzen Übersicht, daß für die Perforationen, Strikturen und Kotsteine vornehmlich die Spitze des Processus in Betracht kommt, während nur die Obliteration sich häufiger an der Basis mit 46,3% gegen 26,8% an der Spitze findet. Dementsprechend ist auch die Gesamtzahl der Affektionen an der Spitze am höchsten mit 16,6% nach einer Berechnung auf sämtliche 349 operierten Königsberger und Rostocker Fälle vertreten. Nach dieser zeigt die Basis

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des Processus den höchsten Prozentsatz mit 12%, und an letzter Stell kommt die Mitte des Processus mit 9,7% der Gesamtfälle.

Wenden wir uns zum Schluß der Häufigkeit und Art de postoperativen Komplikationen des Krankheitsverlaufes be den einzelnen Gruppen zu, so finden wir deren bei den Rostocke und Königsberger Fällen im ganzen 31 an der Zahl, die einem Prozent satz von 8,3% der gesamten 374 Fälle entsprechen. Von diesen ent fällt nach Gruppen berechnet der weitaus höchste Prozentsatz vo 15% auf die Spätfälle, wie es ja bei dem langsamen Verlauf diese Fälle nicht anders zu erwarten ist. Ihnen folgen die Notfälle mit 10,6% und die nicht operierten Fälle mit 8%. Erheblich seltener finden sich naturgemäß die Komplikationen bei den chronischen Fällen mit 6% und am seltensten bei den Frühfällen, die in ihrem Verlauf sämtlich von Komplikationen frei geblieben sind.

Auf die Zahl der Komplikationen allein berechnet, finden wir am häufigsten die Lungenaffektionen mit 45,1% sämtlicher Komplikationen vertreten. Von diesen ist wiederum am zahlreichsten die Pleuritis exsudativa dextra mit 19,6%, danach die Bronchitis purulenta und Pneumonia dextra mit je 9,6% und am seltensten die Lungenembolie die mit 6,5% in den Journalen verzeichnet ist.

Den Lungenaffektionen am nächsten steht die Flexionskontraktur des rechten Oberschenkels mit 22,6%, die an Häufigkeit fast erreicht wird von der Thrombose der linken Vena femoralis mit 19,6%. Schließlich folgt noch Diabetes in 9,6% und Erysipel in 3,2% sämtlicher Komplikationen des Krankheitsverlaufes.

Betrachten wir nun die einzelnen Gruppen auf die Art ihrer Komplikationen näher, so finden wir zunächst bei den chronischen Fällen am zahlreichsten die Lungenaffektionen mit 3,9% vertreten die sich aus Bronchitis purulenta und rechtsseitiger Pneumonie mit je 1,3%, Pleuritis exsud. dext. mit je 0,8% und Lungenembolie mit 0,4% zusammensetzen. Erheblich geringer ist das Verhältnis der Thrombose der linken Vena femoralis mit 1,7% zu konstatieren. Ein akzidenteller Diabetes ist in 0,4% der Fälle verzeichnet.

Dagegen ist bei den Spätfällen weitaus am häufigsten die Flexionskontraktur des rechten Oberschenkels mit 6,9% der Fälle vertreten. Von Lungenaffektionen findet sich allein die Pleuritis exsud. dext. und zwar in gleicher Häufigkeit, wie die Thrombose der linken Vena femoral. und Diabetes in je 2,2% der Fälle angegeben. Schließlich ist in einem Fall = 1,1% der Spätfälle ein Hauterysipel verzeichnet.

Bei den Notfällen ist nur die Lungenembolie und Flexionskontraktur des rechten Oberschenkels in je einem Falle = 5,3% angeführt während andere Komplikationen fehlen, und bei den nicht operierte

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Fällen ist schließlich als einzige Komplikation die Pleuritis exsud. lext. in 8% der Fälle zu konstatieren. Den letzteren beiden Resultaten dürfte jedoch bei der geringen Anzahl von Notfällen und nicht operierten Fällen eine größere Bedeutung nicht beizumessen sein, da bei so kleinen Zahlen leicht ein Zufallsergebnis in Frage kommen kann.

Werfen wir nun zum Schluß noch einen kurzen Rückblick auf die Mortalität unserer gesamten 550 Fälle nach Gruppen geordnet, so finden wir eine ansteigende Linie derselben in folgender Reihenfolge: Frühoperation 0%, Sicherheitsoperation 0,58%, nicht operierte Fälle 2,7%, Spätoperation 7,9%, Notoperation 70,4%. Stellen wir diesen gegenüber das Verhalten der Komplikationen bei den einzelnen Gruppen nach gleicher Reihenfolge geordnet, so finden wir: Frühoperation 0%, Sicherheitsoperation 6%, nicht operierte Fälle 8%, Spätoperation 15%, Notoperation 10,6%. Es ergibt sich daraus, daß die Zahl der Komplikationen sich in einer der Mortalität genau entsprechenden, ansteigenden Linie bewegt mit einziger Ausnahme der Notoperationen, bei denen die Mortalität steil auf 70,4% in die Höhe schnellt, während die Zahl der Komplikationen auf 10,6% von 15% bei den Spätfällen herabsinkt. Diese Differenz erklärt sich jedoch zwanglos aus der Annahme, daß es bei der überaus hohen und meist sehr frühzeitigen Mortalität dieser Fälle an Zeit zu der Entstehung der bei langsamerem Krankheitsverlauf üblichen Komplikationen an anderen Organen mangelt. Also nicht nur hinsichtlich der Mortalität, sondern auch hinsichtlich der Möglichkeit hinzutretender Komplikationen hat die Sicherheitsoperation und ganz besonders die absolute Frühoperation einen weiten Vorsprung vor dem operativen Eingriff in allen anderen Stadien der Appendizitis voraus.

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496.

(Gynäkologie Nr. 181.)

Überblick über die Entwicklung der modernen gynäkologischen Operationstechnik.

Vortrag, gehalten auf der Versammlung der American gynecological Society in Philadelphia (27. V. 1908).

Von

Prof. Dr. A. Martin,

Berlin.

Die Ehre vor dieser Versammlung zu sprechen weiß ich genügend zu schätzen, da ich seit dem Jahre 1888 den innigen Wunsch empfinde, für die Ernennung als Ehrenmitglied dieser Versammlung meinen Dank persönlich abzustatten. Seit nahezu 40 Jahren fühle ich mich mit den amerikanischen Fachgenossen nahe verbunden: damals hatte ich das Glück, einige ihrer glänzendsten Vertreter, Marion Sims, Emmet und Gaillard Thomas persönlich kennen zu lernen. Seit jener Zeit haben sich die Beziehungen auf eine große Zahl der hervorragendsten Söhne dieses großen Landes weitergesponnen. 21 Jahre sind verflossen, seitdem ich auf dem Meeting in Washington vielen von Ihnen begegnen durfte. Die ehrenvolle Einladung zu unserem heutigen Meeting läßt den seit jenen unvergeßlichen Tagen in mir lebendigen Wunsch, Sie wiederzusehen, in schönster Weise in Erfüllung gehen.

Als ich in diesem Zusammenhang die Entwicklung der Gynäkologie in den letzten fünf Dezennien überblickte, reizte es mich besonders zu verfolgen, welche Wege unser Tun und Handeln in diesem Zeitraum durchlaufen hat.

Damals beschränkte sich unsere Therapie auf medikamentöse und orthopädische Applikationen auf Vulva und Scheide. Zaghaft drang man in die Uterushöhle vor. Eben hatte man in der Diszision des engen Muttermundes nach dem Vorgehen von Sir James Simpson und E. Martin einen wesentlichen Fortschritt erkannt, ebenso in der

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Verwendung des Chassaignacschen Ecraseur und der elektrokaustischen Schlinge — während man anfing, die noch sehr zweifelhaft erkennbaren abdominalen Neubildungen etwas zuversichtlicher durch die Laparatomie anzugreifen, besonders nachdem Atlee, Peaslee, Spencer Wells, Baker Brown, Köberlé und Keith mehr als vereinzelte Erfolge erzielt hatten. Marion Sims war es vorbehalten, ebenso wie Gustav Simon und Hegar der vaginalen Methode durch die ausgiebigere Freilegung des Scheidengewölbes zur Plastik am Collum und in der Scheide weitere Bahnen zu eröffnen. Emmets Trachelorrhaphie und die erfolgreicheren Fisteloperationen von Simon und Bozeman erschienen als Triumphe dieses Entwicklungsstadium. Auch die intrauterine Therapie wurde durch die breite Dilatation der Cervix (Preßschwamm und Laminaria, Ellingersches Dilatatorium) und die Eröffnung des Kavum in dieser Zeit erfolgreich weiter ausgebildet. Es wurde damit der nachhaltigeren Einwirkung auf dessen Mukosa frei Bahn geschaffen für Curette und medikamentöse Applikationen.

Der nächste Schritt vorwärts geschah in der abdominalen Chirurgie: Nicht nur, daß wir lernten — mit Köberlé und Keith, Péan und Karl Schröder an der Spitze — neben den Ovarial- auch die Uterusgeschwülste anzugreifen: wir wagten uns auch an die entzündlichen Geschwulstmassen heran. Lawson Tait, Hegar und auch ich durften damals die Möglichkeit derartiger abdominaler Operationen mit nicht unbefriedigenden Erfolgen belegen. Sehr bald ergab sich, daß manche dieser Geschwulstmassen der ektopischen Eieinbettung ihre Entstehung verdankten: wie wurden wir von der Häufigkeit dieser verhängnisvollen Komplikation überrascht!

Inzwischen hatte W. A. Freund den Weg gefunden, durch eine Kombination abdominaler und vaginaler Operation das verhängnisvollste Übel, den Uteruskrebs, auszurotten. Zwar entsprachen zunächst die Erfolge nicht den anfänglichen Erwartungen. Freunds Operation wies aber Emil Rieß den Weg, der seit einem Jahrzehnt in der Ausbreitung auf das pelvine Bindegewebe und die retroperitonealen Drüsen allgemein als die typische Karzinomoperation angesehen wird.

Freunds Vorgehen gab den Anstoß, den karzinomatösen Uterus auf vaginalem Wege auszurotten. Czerny, Billroth, Schröder folgten dieser Anregung. Damit wurde ein gewaltiger Schritt vorwärts getan. Wir lernten in der vaginalen Uterusexstirpation ein Mittel kennen, um auch bei nicht malignen Erkrankungen mit dem Uterus seine erkrankten Adnexorgane vaginal nicht nur zu exstirpieren, nein, sie auch gegebenenfalls zu erhalten, nachdem wir ihre kranken Teile entfernt

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und ihre unwegsam gewordenen Abschnitte wieder wegsam gemacht hatten.

Eine weitere Etappe bilden die Versuche, die bis dahin orthopädisch behandelten Uterusdeviationen operativ zu heilen. Die Ideen Sängers und Olshausens haben ein weites Feld operativer Tätigkeit eröffnet. Erst durch die Erfahrungen mit der Ventrifixur wurde die Alexander-Adamsche Operation weiter verbreitet, um ihrerseits jene wesentlich in den Hintergrund zu schieben.

Den Siegeslauf der Laparatomie zu unterbrechen war Dührßen und Mackenrodt vorbehalten, als sie zu Anfang der letzten Dekade des verflossenen Jahrhunderts einen sicheren vaginalen Weg in den Innenraum des kleinen Beckens und zu allen Beckenorganen zeigten. Fast schien es, daß die Abdominaloperation nur den großen Geschwülsten in der Bauchhöhle vorbehalten werden sollte! Nach kurzen Jahren setzte die Reaktion ein, vornehmlich auf dem Gebiete der ektopischen Schwangerschaft, dann auf dem der entzündlichen Adnexerkrankungen, trotzdem wir durch die raumgebenden vulvo-vaginalen Inzisionen Dührßens und besonders die von Schuchardt angegebene seitlich schräge, eine bessere Narbenbildung sichernde, lernten den Zugang zum Scheidengewölbe und den darüber liegenden Gebilden sehr ausgiebig freizulegen. Unverkennbar ist der Enthusiasmus für die vaginalen Operationen im Laufe des jetzigen Jahrhunderts abgeflaut, so daß heute selbst so überzeugte Vorkämpfer wie Mackenrodt davon abrücken und sich des abdominalen Verfahrens in überwiegender Häufigkeit bedienen.

Die operative Technik hat in dieser Beziehung eine eigenartige Wellenbewegung durchlaufen. Nach einer kurzen Periode überwiegender Bevorzugung vaginaler Operationen entwickelte sich die Laparatomie zur Vorherrschaft, bis die vaginale Technik weiter ausgebildet wurde. Dann aber gewinnt nach kurzen Jahren die Laparatomie wieder einen Vorsprung, der anscheinend in sieghaftem Vordringen den vaginalen Operationen einen sehr bescheidenen Platz übrigzulassen sich anschickt.

Wir fragen angesicht dieses Werdeganges, wie ist es überhaupt gekommen, daß die gynäkologischen Operationen sich in dieser weitgehenden Weise entwickeln konnten? Der Weg wurde vorbereitet durch die wachsende Einsicht in die pathologische Anatomie. Sie gab die Unterlage für die Diagnose, um deren Ausbildung B. S. Schultze, der Nestor der deutschen Gynäkologen, sich hochverdient gemacht hat. Nicht erst wenn der Tumor die Bauchdecken wölbt oder das kleine Becken von den erkrankten Organen völlig verlegt wird, lernten wir die Prozesse differenzieren. Nach Karl Ruges Rat entnehmen wir Teile der Uterusschleimhaut und bauen auf deren mikroskopischen Bildern weitere

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Schlüsse auf. Heute kommen uns die biologisch-chemischen Methode zunutze, um durch das Kultur- und Impfverfahren, weiter durch di Untersuchungen des Blutes, der Se- und Exkrete die Sachlage weit gehend klarzustellen — das wollen wir dankbar anerkennen, wen wir uns auch nicht darüber täuschen, daß nur zu oft noch viel meh Licht erwünscht ist. Gerade auf diesem Felde liegt noch viel Arbei vor uns. So z. B. gilt es noch den Zusammenhang zwischen Genital leiden und Appendix und gewissen drüsigen Organen, die von Hega und Freund gegebene Anregung über die Bedeutung des Infantilismu für die gesamte Pathologie zu erforschen und zutreffend zu bewerten

Wir müssen rückhaltlos bekennen, unsere operative Tätigkeit wär einer solchen Ausdehnung nicht fähig gewesen, wenn wir nicht auf opferreichem Wege durch die Antisepsis zur Asepsis durchgedrunge wären. Sie alle haben diesen Entwicklungsgang selbst durchlebt; wi vereinigen uns in der Huldigung für Holmes und Semmelweiß Pasteur, Koch und Lister, um nur diese Namen zu nennen. Die Saat ihrer Arbeit ist in aller Welt aufgegangen und verbreitet ihre Segen auf allen Gebieten der Medizin; nicht zuletzt aber sind wi Gynäkologen es, die in heißem Bemühen mitgearbeitet haben. In de unermüdlichen Ausgestaltung unserer operativen Technik beginne wir die reiche Ernte vor uns zu sehen. Seit einem Lustrum liegt hinreichendes, modern bearbeitetes Material vor uns, um nicht nur die primären Erfolge unserer Operationen an großen Zahlen zu prüfen — wir sind in das Stadium der Kontrolle der Dauerresultate ein getreten. Wir dürfen uns nicht verhehlen, daß uns diese Prüfung manche Enttäuschung eingetragen hat: sie hat uns Lücken und Fehl schlüsse gezeigt, die uns zur Selbstkritik und zu erneuter Arbeit anregen

Betrachten wir nun unter dem Eindruck der so verschärften Kritik den heutigen Stand unserer operativen Technik, so beansprucht die Laparatomie den Vorzug unbeschränkter Klarstellung der pathologi schen Verhältnisse in der ganzen Bauchhöhle vor der vaginalen Ope ration. Das ist sicher weitgehend zuzugeben, wenn auch die letztere in ihrer heutigen Ausbildung durch die breite Eröffnung des Becken bodens für den Raum unterhalb des Beckeneingangs — soweit es hier lokalisierten Prozessen gilt — an Klarheit nichts zu wünschen übrigläßt

Noch vor einem Lustrum mußte ein sehr schwerwiegender Einwand gegen die Abdominaloperation in der statistisch unbestreitbar höheren Lebensgefahr und in den oft ihr anhaftenden Komplikationen erblickt werden.

Es ist ohne weiteres zuzugeben, daß in ersterer Beziehung wesent liche Fortschritte gemacht worden sind. Unsere Aseptik hat gegen früher wesentlich durchgreifender den Feind, die septische Infektion, bekämpfen

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lehrt. Er verfolgte um so mehr die Laparatomie, weil sie das Peri-
neum den pathogenen Keimen in größerer Ausdehnung freilegt. Es
ird allgemein nach möglichster Abkürzung der Operation getrachtet.
ie Schädlichkeiten der Narkose werden verringert; die Inhalations-
rkose wird abgekürzt, durch die Lumbalanästhesie ersetzt. Die Für-
rge für die Steigerung der Widerstandskraft der Kranken hat durch-
eifende und evidente Erfolge gezeitigt. Gleichzeitig damit ist die
cherheit der Bauchschnittvernarbung gewachsen. Die Schnittführung
ch Pfannenstiel verleiht der Narbe unbestreitbar eine nachhaltigere
iderstandskraft gegen die herniöse Dehnung.

Das erkennen wir rückhaltlos an. Die Erfahrung lehrt uns aber,
ß diese Narben vor einer solchen Dehnung nicht mit voller Sicher-
eit geschützt sind. Selbst bei völlig aseptischem primärem Heilungs-
rlauf werden die physiologischen Umbildungsvorgänge des weiblichen
örpers (Schwangerschaft, Klimakterium) verhängnisvoll, noch mehr,
enn etwa ungeeignete Dehnungsansprüche und erneute pathologische
eränderungen hinzukommen. Erfolgt aber die Heilung aus irgend-
inem Zufall oder einer nicht erkennbaren Lücke der Asepsis nicht per
rimam, dann wächst die Gefahr der Narbendehnung in erschrecken-
er Weise. Sicher erreichen die Meister im Fach lange Reihen un-
störter Heilung, aber auch sie sind nicht dagegen gesichert, daß aus
r Geschwulst, welcher die Operation gilt, eine Verunreinigung der
auchwunde eintritt. Die Gefahr wächst, wo es sich um die Operation
n Entzündungsprodukten handelt, sie begleitet alle diejenigen Fälle,
welchen eine Dränage des Operationsfeldes geboten erscheint.

Noch bedenklicher sind die Spätfolgen, welche wir auch nach
ealer primärer Heilung, nach einem ungestörten Operationsverlauf,
erschreckender Häufigkeit aber nach der Lösung von Verwachsungen
treten sehen: die Verwachsungen der Intestina und des Omen-
m mit der Bauchwunde untereinander und mit dem Stumpf
r entfernten Geschwülste. Die langjährige Kontrolle unserer
perierenden lehrt uns die Bedeutung dieser Komplikationen mehr und
hr würdigen. Keine Modifikation unserer Methoden schließt sie mit
cherheit aus; nicht die ängstliche Vermeidung einer Insultierung
s Peritoneum während der Operation, nicht die sogenannte Peri-
nealisierung der entstandenen Defekte, nicht die Versuche durch
lge Substanzen oder physiologische Kochsalzlösungen die Berührung
r betreffenden Organe zu beeinflussen, nicht die frühzeitige An-
gung des Motus peristalticus — geben sichere Gewähr gegen diese
omplikationen. Sie bleiben der Laparatomie anhaften, jedenfalls in
nem verhängnisvoll größeren Maßstab als den vaginalen Opera-
onen. Gewiß sind die Beckenorgane auch nach diesen gegen Ver-

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wachsungen nicht gesichert; solche Verwachsungen treten aber, jeden falls soweit meine recht ausgiebigen Erfahrungen zeigen, wesentlich seltener hervor.

Die Narbenbildung im Becken erfolgt — ich möchte sagen mi konstanter Regelmäßigkeit, deren sehr seltene Ausnahmen eben di Regel bestätigen — frei von Narbengeschwüren. Selbst da, wo es notwendig, das Wundgebiet zu dränieren, wo infektiöses Material de Tiefe entnommen wurde, sind Unregelmäßigkeiten der Narbe seh seltene Spätkomplikationen. Auch bei vaginalen Operationen werden sicherlich gelegentlich pathogene Keime über das Operationsgebie verschmiert; in der Regel kommt es sehr schnell zu einer Abkapse lung nach oben, Wundsekrete werden durch die Scheide entleert oder jedenfalls von da aus leicht erreicht.

Sicher bedarf es für die vaginalen Operationen einer bestimmten Schulung, ich möchte sagen: einer minutiöseren Operationsarbeit. Nicht immer will es leicht gelingen, das Scheidengewölbe freizulegen, in das Peritoneum vorzudringen, die hier liegenden Organe dem Auge zugängig zu machen: die eben erwähnten Vorteile lohnen reichlich die aufgewandte Mühe. Mehr noch, als nach einer aseptisch und unblutig verlaufenden Laparatomie, gleichen vaginal operierte gesunden Wöchnerinnen, welche nach Abschluß der Geburt nach kurzer Rast ihres Lebens wieder froh werden.

Daß die modernen Narkosenverfahren einen Unterschied zwischen beiden Methoden nicht bedingen, sei hier nur ebenso erwähnt, wie daß für die Rekonvaleszenz nach keiner dieser Operationen heute noch eine lange Dauer der Rückenlage verlangt wird. Je früher sich die Kranke nach eigenem Behagen bewegt, dann das Bett verläßt, um so sicherer bleiben ihr die Gefahren der nachteiligen Rückwirkungen einer strengen Rückenlage auf Herz und Darm erspart.

Daß die vaginalen Methoden nur für ein beschränktes Gebiet in Frage kommen können, ist von vornherein anzuerkennen, aber es ist nicht statthaft zu sagen, z. B. daß Tumoren des Uterus und der Ovarien nur so lange vaginal angegriffen werden dürfen, als sie noch im Becken liegen: eine solche Beschränkung kenne ich nicht! Die Grenze liegt nicht darin, daß etwa die Tumoren nur mit einem sehr beschränkten Segment in das Becken hineinragen, sie liegt in der freien Verschieblichkeit. Auch kleine Geschwulstmassen, welche verwachsen sind, soll man nicht vaginal angreifen; andererseits können aber auch umfangreichere vaginal operiert werden, wenn sie zerkleinerungsfähig sind — solange ihre Oberfläche nicht durch peritonitische Verwachsungen mit der Nachbarschaft schwer kontrollierbar geworden ist. Gewiß können wir nach der Methode von D. v. Ott solche

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Verwachsungen in manchem Falle auch dem Auge zugängig machen und unter der Kontrolle der Beleuchtung sicher trennen — als Regel betrachte ich solche Verwachsungen als Kontraindikationen. Gelegentlich entgehen uns auch recht feste Verwachsungen noch vor der Operation. Sehr oft weist die Anamnese auf eine vorausgegangene Peritonitis hin. Wir lernen auch in dieser Beziehung mit reifender Erfahrung! Aber wenn wir schließlich unerwartet auf solche Verwachsungen stoßen, was hindert uns, die vaginale Operation abzubrechen und abdominal zu Ende zu führen? Nur ganz vereinzelt bin ich hierzu gezwungen gewesen, die Kranken haben keinen Nachteil davon gehabt.

Da wir in wachsender Häufigkeit Frühstadien der Neubildungen zur Beobachtung bekommen, diejenigen aber der Ovarien dann überwiegend häufig noch leicht beweglich sind, bevorzuge ich für diese den vaginalen Weg.

Eine ideelle Konkurrenz zwischen beiden Methoden kommt außer für die Neubildungen des Uterus für die plastischen Operationen im Becken in Frage. Nur zu oft handelt es sich dabei um gleichzeitige Erkrankung des Uterus selbst und der Adnexe. Diese Aufgabe hat an Bedeutung außerordentlich gewonnen, seitdem uns neben den Lageanomalien die entzündlichen Adnexerkrankungen und ganz besonders die ektopische Eiinsertion — fast möchte ich sagen täglich — Indikationen für operative Eingriffe geben!

Die Frage der Uterus-Deviationen hat durch die guten Erfolge der Alexander-Adamsschen Operationen für die vaginalen Methoden in den Augen vieler an Bedeutung verloren und doch sehen wir darnach recht häufig Mißerfolge, besonders aber in erschreckender Weise Narbendehnungen und herniöse Ausstülpungen. Die Tatsache, daß bis heute noch immer neue Operationsmethoden zu ihrem Ersatz erfunden werden, spricht eine beredte Sprache! Mag man die Vaginifixur, die Festlegung des Uterus an die Scheide, verlassen, so verdient jedenfalls die Kürzung und Fixierung der Ligamenta rotunda von der Scheide aus volle Beachtung. Ich habe die Vaginifixur unmittelbar nachdem Mackenrodt und Dührßen uns den Weg durch das vordere Scheidengewölbe gezeigt, ganz besonders deswegen in sehr großer Ausdehnung geübt, weil sie uns gestattet, die so außerordentlich häufigen perimetritischen Pseudomembranen zu lösen. Nur in den Fällen ganz allgemeiner Verwachsung durch feste knorpelig harte Schwielen stoßen wir dabei auf ernste Schwierigkeiten. Diese Fälle wird man kaum vaginal angreifen. Viel bedeutungsvoller aber ist es, daß nur zu oft schwer tastbare zarte Bänder und Zügel den Uterus mit seiner Nachbarschaft verbinden; dabei ist ihm eine gewisse

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Beweglichkeit geblieben. Diese Membranen sind sehr häufig die Ursache der Mißerfolge der Alexander-Adamsschen Operation. Das Verfahren von Goldspohn soll diesem Übel begegnen; es erfreut sich in Deutschland nur eines beschränkten Kreises von Freunden. Goldspohn selbst übt es nur noch sehr selten, besonders mit Rücksicht auf die in Amerika so oft betonte Notwendigkeit, bei allen entzündlichen Genitalleiden die Appendix zu kontrollieren.

Der Einwand, daß die Vaginifixur der Entwicklung des schwangeren Uteruskörpers hinderlich sei — ist hinfällig solange nicht das Korpus sondern die Gegend des unteren Uterinsegmentes festgelegt wird. Dann entfaltet sich, wie hinreichend ausgedehnte Erfahrungen beweisen, das Korpus ungestört als Fruchthalter. Unteres Uterinsegment und Cervix dehnen sich während eines geduldig abgewarteten Geburtsverlaufes.

Die von Freund, Wertheim, Schauta, Pfannenstiel u. a. ausgehenden Vorschläge, durch die Einlagerung des umgestülpten Uterus dem Vorfall der gesamten Genitalien zu begegnen, insbesondere aber ihn zur Stütze der verlagerten Blase zu machen, geben eine weitere, sehr bedeutungsvolle Indikation für vaginale Operationen.

Die Therapie der entzündlichen Adnexerkrankungen ist in den letzten Jahren in ein neues Stadium gerückt, seitdem wir ihre Ätiologie besser erkannt haben. Viel häufiger, als wir es früher annehmen wollten, klingen die gonorrhoischen Infektionen in Tube und Peritoneum nach einem akuten, oft sehr bedrohlichem Stadium ab. Unter geeigneter Pflege tritt Heilung, ja bis zur vollen Funktionsfähigkeit ein! Auch bei den septischen Erkrankungen und den tuberkulösen hat eine etwas ruhigere Auffassung Platz gegriffen: nur eine Minorität dieser Fälle bietet eine Indicatio vitalis. Außerordentlich häufig finden wir lange Zeit nach dem klinisch unverkennbaren ersten Erkrankungsstadium diese Prozesse abgeheilt, sei es, daß uns eine andere Erkrankung zur operativen Freilegung zwingt oder daß Rezidive den Heilungsablauf stören. Dann treffen wir den Eiter eingetrocknet, steril — dann finden wir dicke Schwarten und Schwielen, die auf eine jahrelange Dauer der Erkrankung hinweisen. In der Zwischenzeit haben sich die Trägerinnen subjektiv eines vortrefflichen Befindens erfreut! Neben solchen Fällen sehen wir andere, die vor interkurrenten Störungen im Laufe einer langsamen Genesung bewahrt, schließlich ohne Operation zu einer vollständigen Ausheilung kommen. Das mahnt uns doch sehr ernstlich zu erwägen, ob es richtig ist, da wo eine ernste Lebensgefahr nicht vorliegt, gleich diese Organe zu entfernen, zumal es sich sehr häufig um jugendliche Personen handelt, bei Gonorrhöe, Tuberkulose, Wochenbetts- oder Operationsinfektionen.

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Ob es vorzuziehen ist, in frischem Stadium, „à chaud" abdominal oder vaginal zu operieren, wird viel behauptet und bestritten. Ich habe es in den letzten Jahren vorgezogen, frisch entzündliche Fälle vaginal zu operieren. Purulente Saktosalpingen, ausgedehnte peri- und parametritische Abszesse inzidiere ich durch das Scheidengewölbe. Unter Dränage genesen diese Kranken nicht nur bis zur völligen Euphorie und normalem Lokalbefund; ich habe mehrfach darnach Schwangerschaft und normales Wochenbett beobachtet. Aber die große Mehrzahl dieser Fälle sehen wir im nicht frisch entzündlichen Zwischenstadium. Wir entschließen uns erst dann zur Operation, wenn die Allgemeinbehandlung und die eine Resorption befördernde Lokaltherapie erfolglos geblieben sind. Dann kann man wenig umfangreiche Geschwülste dieser Art auch aus den alten Verwachsungen sehr gut vaginal operieren. Umfangreichere Massen mit unklaren Verwachsungen werden besser abdominal angegriffen.

Schwangere Tuben geben in ihrem Entwicklungsgang sehr verschiedene Indikationen. Daß vorgeschrittenere Fälle, welche über etwa den dritten Monat hinausgekommen sind, zur Domäne der abdominalen Operationen gehören, liegt auf der Hand. Vorher aber sind ungestörte ektopische Schwangerschaften mit großer Sicherheit vaginal zu operieren. Streitig ist die Wahl, wenn der Fruchtkapselausbruch erfolgt. Auch hier ist zu unterscheiden, ob es zu einem Tubenhämatom gekommen ist oder zu einer Hämatokele; erstere habe ich mit voller Sicherheit auch bei mehr als Faustgröße vaginal operiert, ebenso auch letztere, wenn sie abgekapselt erschien. Die Blutstillung ist vaginal mit voller Sicherheit durchzuführen, Koagula und Eisack zu entfernen. Ich lasse gern, wenn es angängig ist, die entbundene Tube zurück, getreu dem Grundsatz, nur das zu entfernen, was für den Erfolg der Operation nötig ist. Aber ich konzediere rückhaltlos, daß wir bei mangelnder Übung, mangelnder Assistenz im Hause der Patienten, auf dem Lande und unter sonst ungünstigen Verhältnissen rascher und sicherer durch die Laparatomie dazu kommen, die blutende Tube zu versorgen, und das ist unsere erste Aufgabe in den Fällen drohenden Verblutungstodes. Eventuelle Komplikationen der Bauchnarbe müssen die vom Verblutungstod Geretteten mit in den Kauf nehmen.

Erst in der letzten Zeit eröffnet Schauta dem vaginalen Verfahren wieder eine weite Perspektive, indem er durch eine sehr weitgehende vaginale Operation das Collumkarzinom angreift. Er berichtet über glänzende primäre und Dauererfolge an einem großen Material. Die Exstirpation des parametranen Bindegewebes gelingt dabei sicher, wie ich mich durch mehrfache Operationen an der Leiche überzeugt habe.

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Schauta verzichtet auf die Exstirpation der retroperitonealen Drüsen! Gewiß entfernt er sich damit von den heutigen Anschauungen der Chirurgen, aber wir dürfen nicht verkennen, daß unsere bisherigen Durchforschungen solcher exstirpierten Drüsen sehr ernste Bedenken anregen. Hier stellt sich uns ein Problem, das unser aller Mitarbeit herausfordert!

Ich bekenne mich, wie diese Skizze zeigt, als warmen Fürsprecher der vaginalen Operation, soweit durch diese mit Aussicht auf sicheren Erfolg dem Übel beizukommen ist. Auch für mich ist die Laparatomie die technisch leichtere, sie ist mir aber ultima ratio. Nur zu oft drängen wenig ausgedehnte, dabei aber recht beschwerliche, Leben und Lebensgenuß behindernde Krankheitsprozesse zur Operation. Wir müssen dann überlegen, ob wir nicht durch eine für uns vielleicht schwierigere, dafür aber doch sicheren Erfolg versprechende Methode zum Ziele kommen. Ich halte die Vermeidung einer Bauchschnittnarbe immerhin, selbst wenn mit der abdominalen Operation eine größere Operationsgefahr nicht verbunden ist, für sehr wesentlich im Interesse unserer Kranken. Bemühen wir uns frühzeitig die Entwicklung der Krankheitsprozesse zu erkennen, lernen wir in sorgsamer klinischer Beobachtung den Werdegang derselben beurteilen, greifen wir rechtzeitig ein, wo ein Eingreifen nicht zu umgehen ist, dann stehen wir oft vor der Möglichkeit, insbesondere Neubildungen und entzündliche Prozesse zu operieren, welche einer vaginalen Operation noch zugängig sind.

Ich verschließe mich nicht dem Einwand, daß da, wo eine Kontrolle des Wurmfortsatzes geboten erscheint, die Eröffnung der Bauchdecken sicherer zum Ziele führt. Aus eigener Anschauung habe ich den Eindruck gewonnen, daß aus Furcht vor der Möglichkeit einer späteren Appendizitis dieses Gebilde zweifellos oft ohne zwingende Berechtigung exzidiert wird. Ebenso werden die Bauchdecken nur zu häufig ohne dringende Indikation gespalten, wo ein einfacher vaginaler Eingriff vollständig zur Abhilfe vorhandener Erkrankungsprozesse ausreichen würde.

Die vaginalen Operationen erfordern eine exaktere Diagnose, eine minutiösere Technik. Innerhalb ihrer gegebenen Grenzen führen sie sicher zum Ziele und bewahren in größerer Ausdehnung vor verhängnisvollen Spätwirkungen als die abdominalen.

Mögen diese Ausführungen zu erneuter Verwendung dieser Methoden anregen!

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497/98.

(Gynäkologie Nr. 182/83.)

Die Erfolge und Dauererfolge der Hebosteotomie an der Kieler Universitätsfrauenklinik.

Von

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Mit sechs Tabellen.

Über die operative Erweiterung des engen Beckens sind aus der Kieler Universitätsfrauenklinik bisher zwei Publikationen hervorgegangen: H. Hohlweg, 4 Fälle von Pubiotomie, Z. f. Gyn. 1905, S. 1281 und die unter meiner Anleitung gefertigte Inaugural-Dissertation: C. Salomon, Ein Beitrag zur Statistik der Pubiotomie (5 Fälle), Kiel 1907. Auf diese Arbeiten zurückgreifend, und ferner fußend auf den seit dieser Zeit ausgeführten Schambeinschnitten möchte ich das hervorheben, was wir aus den zwar nicht sehr zahlreichen, aber gut ausgewählten und sorgfältig weiter beobachteten Fällen gelernt haben.

Im ganzen wurde bisher das Os pubis 20mal durchsägt, und zwar wurde nur im 1. Falle die offene Durchschneidung des Knochens nach Gigli vorgenommen, die nächsten 12 Fälle nach der Döderleinschen subkutanen Schnittmethode mit dem Döderleinschen Instrumentarium behandelt und die letzten 7 Fälle nach der von Kroemer[1]) angegebenen subpubischen Schnittmethode mit der Bummschen Nadel operiert. Das Herumleiten der Nadel geschah bei den nach Döderlein operierten Fällen von der oberen Inzisionswunde aus, meist nur unter indirekter Kontrolle des in die Scheide gelegten Zeigefingers, während bei dem Kroemerschen Verfahren der in die untere Wunde eingeführte Zeigefinger zum unmittelbaren Schutze der Harnblase gegenüber der von unten nach oben dringenden

1) P. Kroemer, Über Versuche, den primären Verlauf und die Dauerresultate der Hebosteotomie zu bessern. Hegars Beiträge zur Geb. u. Gyn. 1907, Bd. 12, H. 2, S. 261 ff.

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Bummschen Nadel und der umgekehrt wandernden Säge an der hinteren Fläche des Os pubis lag.

Ich lasse nun zunächst die 20 Fälle in einer tabellarischen Übersicht folgen (siehe diese); soweit sie schon publiziert sind, nur unter Hervorhebung der wichtigsten Punkte, im übrigen mit genaueren Angaben, um dann an der Hand der Erfolge und Dauererfolge für Kind und Mutter die Indikationsstellung und Technik der Operation zu beleuchten und den Wert festzustellen, den diese Operation unter und gegenüber den bisher gültigen und geübten Verfahren für uns gewonnen hat.

Wenn man sich ein Urteil darüber verschaffen will, welchen Gewinn die Hebosteotomie gebracht hat, so muß man in Erwägung ziehen, daß sie im wesentlichen eine Operation im Interesse des Kindes ist bei einem Mißverhältnis zwischen dem Beckenkanal und dem Kopf eines vollwertigen lebenden Kindes, sei es, daß das Mißverhältnis hervorgerufen ist durch größere Differenzen in den Maßen von knöchernem Geburtsweg und Geburtsobjekt, sei es bei geringeren Maßunterschieden durch mangelnde Konfigurationsmöglichkeit des Schädels, durch Versagen der konfigurierenden Kräfte, durch ungünstige Einstellung des Kopfes, oder aber durch Summation von mehreren dieser Faktoren. Führen wir die Operation aus, so müssen wir sicher sein, oder mindestens genügend begründete Aussicht haben, daß das Kind, um dessentwillen wir das Mißverhältnis zwischen Kopf und Becken auf dem Wege des beckenerweiternden Schnittes beseitigen, auch wirklich lebend geboren wird, und nicht nur lebend, sondern auch weiter existenzfähig zur Welt kommt. Stirbt das Kind trotz der Beckendurchsägung intra partum oder an einer auf die Entbindung zurückzuführenden Schädigung ab, so war unsere Indikationsstellung oder aber unser geburtshilfliches Verhalten nach Vollendung der vorbereitenden Operation fehlerhaft, da wir ohne Berücksichtigung des Kindes ein schonenderes Verfahren einschlagen konnten.

Wenn wir unser Material daraufhin prüfen, so finden wir, daß tatsächlich alle 20 Kinder lebend entwickelt worden sind = 0% Mortalität. 1 Kind starb aber am 2. Wochenbettstage an einem diffusen intrakraniellen Bluterguß (Fall 6). Der Kopf wurde nach Durchsägung des Schambeins ohne Schwierigkeit und ohne nennenswerten Kräfteaufwand mittels Forceps in und durch das Becken hindurch gezogen. Vor der Hebosteotomie waren außerhalb der Klinik energische Zangentraktionen seitens des behandelnden Arztes ohne Erfolg geblieben. Danach ist es wohl mehr als wahrscheinlich, daß der resultatlose Forceps die subdurale Blutung und somit den Tod des Kindes verschuldet hat. — Weist schon dieser Fall auf die verderbliche, das kindliche Leben vernichtende Wirkung des hohen Forceps bei engem

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Becken hin, so werden wir in dieser Auffassung noch bestärkt, wenn wir die andern Fälle berücksichtigen, bei denen der Beckenerweiterung ein Zangenversuch vorausging. Trotz schonender Traktion mit dem Forceps, trotzdem beim Mißlingen sofort von weiteren Extraktionsmanövern Abstand genommen wurde, und der Kopf nach Durchsägung des Beckens dem Zangenzug leicht folgte, kam das bis zum Operationsbeginn ganz lebensfrische Kind in Fall 1 asphyktisch zur Welt und wurde erst nach 20 Minuten zum Schreien gebracht. — In Fall 12 bewirkten kräftige, nicht zum Ziele führende Traktionen mit der Simpsonschen Achsenzugzange eine lange und ziemlich tiefe Druckspur auf der linken Wange des Kindes. Nach der Durchtrennung des Os pubis gelang die Beckenpassage des kindlichen Kopfes ohne jeden Widerstand. — In Fall 16 endlich verursachte der dem Schambeinschnitt vorausgegangene allerdings recht energische Zangenversuch eine ungewöhnlich starke Impression am linken Stirnbein und eine nicht unbeträchtliche Weichteilverletzung am rechten Auge. Man muß sich darüber wundern, daß das Kind überhaupt diese Verletzung überstanden hat.

Aus diesen Erfahrungen geht zur Evidenz hervor, wie unrichtig es ist, der vorbereitenden Knochenoperation einen Entbindungsversuch in Gestalt der hohen Zange vorauszuschicken. Wir schädigen dadurch das Kind in ganz unnötiger Weise, stellen den, eine exakte Indikationsstellung vorausgesetzt, annähernd sicheren Erfolg für das kindliche Leben in Frage und machen den Nutzen der Operation illusorisch. Ich gehe noch weiter und möchte behaupten, daß die beckenerweiternde Operation in einem Fall, bei dem das Kind schon mittels hoher Zange unter Anwendung großer Körperkraft bearbeitet worden ist, und bei dem man die Überzeugung gewinnt, daß das Kind schon schwer gelitten hat, nicht mehr berechtigt ist, weil wir die notwendige Garantie für ein lebensfrisches Kind trotz vorhandener regelmäßiger Herztätigkeit nicht mehr bieten können. Im Anschluß an so rigorose und in ihrer Wirkung resultatlose Entbindungsmanöver sollte nur noch die Perforation die gegebene Operation sein. Früher, als wir die Hebosteotomie nicht hatten, bzw. als sie noch nicht genügend fundiert war, konnte man gegen eine Operation auf Biegen und Brechen nichts einwenden. Wenn das Kind der Einwirkung der elementaren Gewalt des hohen Forceps standhielt, so war das eben der Gewinn eines Lebens. Jetzt darf man die hohe Zange bei engem Becken nur als Versuch für die Fälle zugestehen, denen die Hebosteotomie versagt bleiben muß. — Unter hoher Zange verstehe ich die Anwendung des Forceps in jenen Fällen, bei denen der Kopf zwar fest auf den Beckeneingang aufgepreßt ist und mit einem mehr oder weniger großen

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Segment ins Becken hineinragt, aber weder mit seinem größten Um fang den Beckeneingang passiert hat, noch mit seiner größten Zirkum ferenz im Beckeneingang steht.

Im übrigen finden wir nur noch eine Kopfverletzung verzeichne eine tiefe löffelförmige Impression des hinteren Scheitelbeins (Fall 10 In diesem Fall, der ursprünglich für den Kaiserschnitt aus relativ Indikation bestimmt war (rachitisch plattes Becken mit Conj. ver von 6,2 cm) konnte an der Indikationsstellung, wie wir später noc sehen werden, nicht festgehalten werden. Das Kind wäre sicher ver loren gewesen und der Perforation verfallen, wenn man nicht noc einen Rettungsversuch mit der Hebosteotomie gemacht hätte. De Versuch gelang, weil das Kind die mittlere Größe wenig überstieg die Schädelknochen weich und elastisch waren. Nur als Ausnahm von der Regel gehört dieser Fall in den Rahmen der Hebosteotomi hinein. Er zeigt nur zu deutlich, daß der Schambeinschnitt auch seine Grenzen hat als Operation mit wirklichem Nutzeffekt. Sehr große Mißverhältnisse lassen sich auch durch die operative Spaltung des Beckenringes nicht beseitigen.

Dem Todesfall an intrakranieller Blutung reiht sich noch ein wei terer (Fall 13) an. Dieses Kind wurde durch Wendung und Extrak tion leicht asphyktisch entwickelt, durch bloße Hautreize schnell wiederbelebt. Am 7. Wochenbettstage starb es plötzlich, nachdem es noch 1 Stunde vorher reichlich Brust genommen hatte. Deutliche Zeichen einer Erkrankung, speziell Fieber und Unruhe, waren nicht hervorgetreten. Die Sektion ergab lediglich kleine pneumonische In filtrate der linken Lunge (Aspirationspneumonie).

Demnach sind alle Kinder = 100% lebend geboren worden aber nur 18 gesund aus der Klinik entlassen = 90%. Von diesen 18 entlassenen Kindern starben 2, offenbar infolge mangelnder Pflege nach 4 Wochen resp. 3 Monaten (Fall 4 u. 5).

Hätte man in den 20 Fällen die beckenerweiternde Operation nicht vorgenommen, so wären die Kinder 16mal sicher nicht lebend zur Welt gekommen, 4mal mit größter Wahrscheinlichkeit verloren ge wesen. Es ist demnach auf der Seite des Gewinnes ein großer Erfolg zu buchen. Die Operation hat das voll gehalten was wir von ihr erwarteten. Sie hat die Mortalität der Kinder auf ein Minimum beschränkt.

Von der im Interesse des Kindes unternommenen Operation muß man nicht allein einen Erfolg für das Kind verlangen, sondern auch gleichzeitig eine Gefahrlosigkeit des operativen Eingriffes für die Mutter. Es darf auf keinen Fall die kindliche Mortalität auf Kosten der Mortalität und Morbidität der Mutter herabgesetzt werden

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Von den 20 Müttern ist 1 gestorben = 5% Mortalität.[1]) Dieser Fall ist schon ausführlich durch Hohlweg publiziert und in der Dresdener Kongreßtabelle von Döderlein (Verh. d. D. Gesch. f. Gyn. XII, S. 138, Nr. 59) registriert worden. Ich wiederhole, daß diese Frau am 4. Wochenbettstage einer eiterigen (Staphylokokken) Peritonitis erlegen ist, und daß die unmittelbar post partum placentae geschehene Abimpfung von der fötalen Fläche der Nachgeburt Staphylokokken neben Bacterium coli ergeben hat, während die Knochenwunde bei der Autopsie keine Spur von Reizung, geschweige denn Eiterung aufwies. Mit Recht ist wohl anzunehmen, daß diese Frau auch ohne Hebosteotomie an der bereits bestehenden Infektion (vielfache Untersuchungen durch Hebamme und Arzt außerhalb der Klinik, über 3 Tage hin sich erstreckende Geburt bei vorzeitigem Fruchtwasserabfluß) zugrunde gegangen wäre. Somit würde dieser Fall die Hebosteotomie sicherlich nicht direkt belasten und diskreditieren. Aber man könnte einwenden, eine Perforation des zwar lebenden, aber nicht mehr ganz intakten Kindes (Infektion des viele Stunden offenen Eisackes) hätte die völlige Erweiterung der unnachgiebigen Cervix (Bossischer Dilatator) unnötig gemacht, man hätte langsamer und schonender operieren können; vielleicht wäre dann auch die manuelle Plazentarlösung erspart geblieben. Da man einem solchen Einwand nicht jede Berechtigung absprechen kann, darf man diesen Todesfall nicht ohne weiteres aus der Mortalitätsquote der Hebosteotomie streichen.

Von den der Mutter intra operationem drohenden Gefahren steht in erster Linie die Blutung. Sie war in unsern 20 Fällen meist gering oder nur mäßig stark, 7mal ziemlich stark, anscheinend immer rein venös und durch bloße Kompression ohne Schwierigkeit zu beherrschen. Am stärksten machte sie sich in Fall 5 geltend, wo im ganzen Genitalgebiet ausgesprochene Varicenbildung vorhanden war. Offenbar reichten die Varices, obwohl die weniger beteiligte Seite zur Operation gewählt war, in das Sägeschnittgebiet nicht nur vor, sondern auch hinter dem Os pubis hinein. Bei sehr intensiver und ausgedehnter Entwickelung ektatischer Venen ist jedenfalls große Vorsicht geboten. In unserem Fall entstand im Labium majus der Operationsseite ein überhühnereigroßes Hämatom, das sich allmählich zurückbildete und nach 5 Wochen auf Haselnußgröße verkleinert war. Auch zwei weitere Blutergüsse, ein hühnereigroßes Hämatom im Labium majus sin. (Fall 11) und ein kleineres prävesicales Hämatom (Fall 2)

1) Korrekturbemerkung: Inzwischen sind 3 weitere Hebosteotomien mit glücklichem Ausgang für Mutter und Kind ausgeführt worden, so daß nunmehr bei 23 Fällen die mütterliche Mortalität = 4,3% beträgt.

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vereiterten nicht, sondern wurden langsam resorbiert, innerhalb von 8 bzw. 6 Wochen. Schwerste Blutungen aus dem Sägebezirk oder unangenehme Folgen von Blutergüssen im Bereich des Knochenschnittes resp. dessen Umgebung haben wir demnach nicht erlebt.

Als außerordentlich schwerwiegende Komplikation der Hebosteotomie muß ferner die Verletzung der Harnblase angesehen werden. An unserem Material ist keine Läsion der Harnblase konstatiert worden. Falls eine solche passiert wäre, hätten wir sie wohl mit Sicherheit finden müssen, da wir jedesmal am Schlusse der Operation eine Kontrolle der Harnblase durch Auffüllen und sorgfältiges Forschen nach etwaiger Blutbeimengung vorgenommen haben. Die Vermeidung einer Harnblasenläsion in unsern 20 Fällen führen wir darauf zurück, daß bei dem rein subkutanen Operieren nur ein stumpfes Führungsinstrument, die stumpfe Nadel von Döderlein, unter Kontrolle von der Vagina aus angewandt wurde, während den Schutz der Harnblase gegenüber der scharfen Bummschen Nadel immer der vom Kroemerschen subpubischen Weichteilschnitt aus hinter das Os pubis gelegte Zeigefinger übernahm. Bei einer großen Serie von Operationen, wo man mit dem Zufall nicht mehr zu rechnen braucht, wird die Blasenverletzung, die in ihren gefährlichen Konsequenzen recht deutlich durch die Hammerschlagsche[1]) Erfahrung gekennzeichnet ist, nur dann umgangen werden können, wenn man prinzipiell Sägeführer und Säge nur unter direkter Fingerdeckung um das Os pubis herumleitet. Würde sich herausstellen, was bisher meines Wissens nicht beobachtet ist, daß noch nach Placierung der Säge beim ersten Sägezug, also vor dem Verschwinden der Säge im Knochen, die hinten anliegenden Gewebe (Harnblase, Durchtrittsschlauch mit fötalem Kopf) angesägt werden können, so müßte auch noch dieser Akt unter geeigneter Deckung vor sich gehen. — Aber nicht nur eine unmittelbare Läsion wird durch solch Vorgehen vermieden, sondern auch die nachträgliche Zerreißung der Harnblase beim Klaffen der Knochenwunde. Der von unten hinter das Os pubis dringende Zeigefinger hat eben nicht nur den Zweck des direkten Schutzes der umgebenden Weichteile, sondern verfolgt vor allem auch die Tendenz, die Harnblase und ihren Befestigungsapparat am Os pubis in ausreichendem Maße abzulösen. Zweifellos schützt keine Technik der Hebosteotomie vor der schweren Komplikation der Blasenläsion auch nur mit annähernder Sicherheit, wenn

1) S. Hammerschlag, Warnung vor poliklinischer Ausführung der Hebosteotomie. Zentralbl. f. Gyn. 1907, S. 1001—1003.

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nicht diese Ablösung von dem Os pubis vor der Knochendurchsägung geschieht, und die Harnblase so lange zurückgehalten wird, als in ihrer unmittelbarsten Nähe Sägeführer oder Säge arbeiten. Denen, die da einwenden, die Blutung sei bei der Ablösung der Harnblase wesentlich stärker, entgegne ich, daß wir bezüglich der Blutstillung nie Schwierigkeiten gehabt haben. Falls man nach Kroemer den Klitorisschenkel vom absteigenden Schambeinast ablöst und sich dann hart an die Hinterfläche des Os pubis hält, wird wohl kaum die Blutung einen exzessiven Grad erreichen. Man vermeidet damit die direkte Verletzung des Corpus cavernosum clitoridis, nennenswerter arterieller Gefäße, des Plexus pudendalis und des Plexus vesicalis.

Außer der Blutung und der Harnblasenläsion sind die für den Schambeinschnitt ihrer Lokalisation und Form nach typischen Scheidenverletzungen zu fürchten, weil damit gerade das eintritt, was man verhüten wollte: die Kommunikation der Knochenwunde mit dem Geburtskanal. Einer von der Vagina ausgehenden Infektion der Wunde mit ihren komplizierten Verhältnissen ist damit Tür und Tor geöffnet.

Solche kommunizierende Scheidenrisse haben wir nicht weniger als 7 erlebt. In allen diesen Fällen wurde nach Döderlein, resp. in Fall 1 nach Gigli hebosteotomiert und 6mal wegen fötaler, mütterlicher oder gemischter Indikation sofort im Anschluß an die Hebosteotomie, 1mal nach 33stündigem vergeblichen Warten auf eine Spontangeburt mit Zange entbunden. Von den 12 nach Döderlein operierten Fällen wies die Hälfte die kommunizierende Scheidenverletzung auf. In welchen Fällen kam sie zustande? Es handelte sich 4mal um Erstgebärende, 3mal um Zweitgebärende. Zu diesen Erstgebärenden zählen 2 alte Ip. mit allgemein gleichmäßig verengtem Becken und engen unnachgiebigen Weichteilen (Fall 4: 38j. Ip. u. Fall 12: 31j. Ip.), 1 28j. Rachitika mit plattem Becken und schlecht dehnbaren Geburtswegen (Fall 6) und 1 21j. Parturiens mit allgemein verengt rachitisch-plattem Becken, bei der das Journal betont, daß eine genauere digitale Beckenuntersuchung wegen der Enge der Weichteile nur sehr schwer gelang (Fall 11). — Von den 3 Zweitgebärenden hatten 2 ein allgemein gleichmäßig verengtes Becken (Fall 1 u. Fall 7), die 3. ein rachitisch plattes, dabei leicht allgemein verengtes Becken (Fall 9). Bei Fall 1 klaffte die nach Gigli offen angelegte Knochenwunde um 7 cm, bei Fall 7 ist ein enger und hoher Schambogen besonders hervorgehoben, und bei Fall 9 waren die Hypoplasie der Genitalien und die Narben von der 1. Entbindung (Dammriß 2.° und tiefe Scheidendamminzision) Momente, welche die Erweiterung der Scheide erschwerten.

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Die nach Döderlein hebosteotomierten 6 Frauen ohne kommunizierende Scheidenverletzung waren 1 I p., 2 II p., 1 III p. und 2 IV p. Nach der Beckenform verteilt findet sich darunter kein allgemein gleichmäßig verengtes Becken (im Gegensatz dazu 4 allgemein gleichmäßig verengte Becken mit kommunizierender Scheidenverletzung), 2 allgemein verengte rachitisch platte und 4 rachitisch platte Becken. In keinem dieser Fälle war der Schambogen verengt, 5mal weit oder sogar sehr weit. Die Weichteile waren wohlentwickelt und für die Geburt gut vorbereitet, speziell ist bei der einzigen von 5 in Betracht kommenden Erstgebärenden, die keinen kommunizierenden Scheidenriß erlitten hat, nachdrücklich darauf hingewiesen, daß Damm und Scheide gut aufgelockert und dehnbar gewesen seien (Fall 3). Als entbindende Operation, die auch in diesen 6 Fällen unmittelbar an die Knochendurchsägung angeschlossen wurde, diente 2mal die Zange und 4mal die innere Wendung und Extraktion.

Vergleichen wir die beiden gegenübergestellten Gruppen von Operationsfällen miteinander, so können wir leicht ersehen, daß nicht allein das der Hebosteotomie auf dem Fuße folgende Entbindungsverfahren, speziell die Zange, an dem Zustandekommen der kommunizierenden Scheidenverletzung die Schuld trägt, sondern noch andere Faktoren für die Entstehung der unangenehmen Komplikation heranzuziehen sind, nämlich die Form des Beckenausgangs und die Beschaffenheit der Weichteile. Ungünstig liegen in dieser Richtung die Fälle von allgemein gleichmäßig verengtem und allgemein verengtem platten Becken, weil mit der Allgemeinreduktion der Beckenlichtung ganz gewöhnlich eine mangelhafte Entwickelung der weichen Geburtswege, speziell eine abnorme Enge der Scheide und eine Hypoplasie der äußeren Genitalien einhergeht. Noch schwieriger können die Verhältnisse werden, wenn dazu auch noch der Elastizitätsverlust der Gewebe einer alten Erstgebärenden oder eine Narbenrigidität im Gebiete der Vagina und des Vestibulums komplizierend hinzutritt. — Nach unseren Erfahrungen läßt sich das Bersten der Scheidenwand entsprechend der Knochenwunde bei engem Beckenausgang und ungünstiger Beschaffenheit der Weichteile, sei es Hypoplasie oder Rigidität, mit der bisher besprochenen Operationstechnik (Gigli und Döderlein) nicht verhüten; ob durch Abwarten der Spontangeburt, ist möglich, aber keineswegs sicher. Jedenfalls darf man auf die Spontangeburt schon deshalb nicht bauen, weil man jeden Augenblick gezwungen werden kann, auf fötale oder mütterliche Indikation hin doch die Geburt operativ zu beenden.

Wenn ich unsere Fälle daraufhin kritisch betrachte, so finde ich

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kaum einen heraus, bei dem man noch hätte warten können, und in dem einzigen Fall, wo es geschah (Fall 11), warteten wir 33 Stunden vergeblich auf die Spontangeburt, um dann doch nach der für die Kreißende und für die Umgebung qualvollen Wartezeit mit Zange entbinden zu müssen. Und die kommunizierende Scheidenzerreißung, deren Verhütung wir doch mit unserm Warten im Auge hatten, wurde nicht vermieden.

So rationell und selbstverständlich auch das Abwarten der Spontangeburt zunächst erscheint, viel Nutzen darf man sich davon nicht versprechen, weil es gerade in den Fällen, wo wir es am nötigsten brauchen, im Stich läßt. Bei dem hypoplastischen Genitalapparat fehlt es eben an den eine Spontangeburt garantierenden Austreibungskräften. Natürlich will ich nicht bestreiten, daß man in dem einen oder andern Fall mit der Spontangeburt Erfolg haben kann, vielleicht auch einmal in solchem Falle, wo voraussichtlich bei sofortiger operativer, vor allem Zangenentbindung eine schwerere Weichteilverletzung entstanden wäre. Im Hinblick aber auf die so oft erforderliche schleunige Entbindung haben wir alle Veranlassung, uns umzusehen, ob wir nicht imstande sind, unsere Hebosteotomietechnik dem offenbar häufigen Bedürfnis der sofortigen Geburtsbeendigung anzupassen und so zu verändern, daß die kommunizierenden Scheidenrisse sich entweder ganz verhüten lassen oder wenigstens zu den Seltenheiten gehören.

In der Tat meine ich, daß die Kroemersche Schnittführung, die sich auf die Art und Form der bei Zangenoperation nach Hebosteotomie von selbst zustande gekommenen Risse gründet, den natürlichen Verhältnissen also Rechnung trägt, in hervorragender Weise den bisher unvermeidlich scheinenden Weichteiltraumen vorzubeugen vermag. Seit dem 1. 12. 07 haben wir 7 Hebosteotomien mit nachfolgender künstlicher Entbindung (3 verschiedene Operateure) nach dieser Methode ohne Rißkomplikation ausgeführt. Von diesen Fällen sondere ich 2 aus (Fall 17 u. 18), weil hier wegen des weiten Schambogens und der günstigen Weichteilverhältnisse (Vp. u. IVp.) auch bei Anwendung des Döderleinschen Verfahrens die Rißkomplikation nicht zu fürchten gewesen wäre. In Fall 15 (allgemein verengtes rachitisch plattes Becken mit gut mittelweitem Schambogen einer 24j. I p.) gelang es trotz sehr enger Scheide mit Zuhilfenahme einer anderseitigen Scheidendammzision ein mittelgroßes Kind durch innere Wendung und Extraktion ohne Rißverletzung zu entwickeln. — In Fall 14 u. 19, 34j. II p. und 34j. IV p., wurde die Rißverletzung vermieden bei allgemein verengtem rachitisch platten Becken mit hohem, engen Schambogen: innere Wendung und Extraktion eines über mittelgroßen bzw. kleinen Kindes. — In Fall 20 wurde bei allgemein gleichmäßig verengtem Becken mit

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engem und hohem Schambogen ein 4400 g schweres Kind, das in 2. unvollkommener Steißfußlage sich eingestellt hatte, durch Extraktion entwickelt. — In Fall 16 endlich glückte die Zangenextraktion eines 4330 g schweren Kindes bei einer 27j. IIp. mit allgemein gleichmäßig verengtem Becken ohne Schädigung der so gefährdeten Scheidenpartie. — Diese letzten beiden Beobachtungen fallen um so mehr ins Gewicht, als wir bei allen andern allgemein gleichmäßig verengten Becken (4mal: 2 Ip. u. 2 IIp.) die typische Rißverletzung mit der Döderleinschen Methode nicht hatten umgehen können. — Die von Kroemer[1]) geratene gleichseitige Scheidendamminzision haben wir bisher nicht in Anwendung gezogen. Bei abnormer Enge der Scheide ist die von Pfannenstiel (Monatsschr. f. Geb. u. Gyn. 1906, Bd. 24, S. 415) als prophylaktischer Entspannungsschnitt empfohlene Spaltung des Scheidenrohres auf der der Hebosteotomie entgegengesetzten Seite mit gutem Erfolge angewandt worden.

Welchen Einfluß hatte denn nun die mit der Knochenwunde kommunizierende Scheidenverletzung auf den Verlauf des Wochenbettes? Von den 7 Wochenbetten verliefen 2 fieberlos (niemals Temperatur in axilla über 38,0°), bei absolut glatter Wundheilung (Fall 1 u. 9). Beide Male wurde der kommunizierende Riß möglichst exakt vernäht, das Vaginalrohr also wieder in sich geschlossen. Während in Fall 1 die nach Gigli angelegte Hebosteotomiewunde ausgiebig nach oben und unten mit Jodoformgazestreifen dräniert und nur die Hautwunde mit einigen Catgutnähten zusammengezogen wurde, erfolgte in Fall 9 auch eine Naht der beiden Inzisionswunden oberhalb und unterhalb des durchsägten Schambeins. — In Fall 4 blieb das große Wundgebiet, trotzdem die Frau am 4. Wochenbettstage einer Staphylokokkenperitonitis erlag, infolge unmittelbarer sorgfältiger Vernähung des kommunizierenden Scheidenrisses laut Sektionsbefund frei von Infektion. — Im Fall 12 heilte die dicht vernähte Scheidenrißwunde primär, ebenso die durch Naht geschlossene obere Inzisionswunde; an der mit 2 langen Jodoformgazestreifen dränierten unteren Inzisionswunde aber stellte sich bei zweimaliger Temperaturerhöhung auf 38,2° in axilla eine mäßig starke eiterige Sekretion ein. — In Fall 11 wurden die beiden Inzisionswunden sofort nach Durchsägung des Knochens vernäht und ebenso der kommunizierende Scheidenriß, der nach langem vergeblichen Warten auf Spontangeburt bei der dann unvermeidlich gewordenen Zangenextraktion entstanden war. Unter leichtem Temperaturanstieg entwickelte sich hier ganz allmählich eine Thrombose der linken Vena

1) P. Kroemer, l. c., S. 262, Fig. 1.

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emoralis ohne stärkere Schwellung des linken Beines. — In Fall 6 örte ein schmerzhaftes Ödem des Operationsgebietes und eine eiterige ronchitis mit pneumonischem Infiltrat im rechten Unterlappen das Wochenbett. Die Erscheinungen wurden von einem leichten, bis 3° in axilla ansteigenden Fieber begleitet, das gut 14 Tage hindurch ührte. Aus der schon längere Zeit völlig geheilten Rißwunde stieß ch $2^1/_2$ Monate post hebosteotomiam ein dünner 3 cm langer und cm breiter Knochensequester ab. Es liegt nahe, anzunehmen, daß ne Infektion des ausgedehnten Wundbezirkes auf embolischem Wege dem entzündlichen Lungenprozeß, an Ort und Stelle zu der anfangs bemerkt gebliebenen Knochennekrose führte. — Fall 7 schließlich igte die bedrohlichsten Folgeerscheinungen, eine schwere Thrombo-lebitis im Bereich der Knochenwunde und eine von hier ausgehende eptikopyämie mit 2 Schüttelfrösten am 15. und 17. Wochenbettstage. – Sicher ist es nicht zufällig, daß gerade diese beiden letzten Fälle durch einen fieberhaften, und der letzte Fall sogar ernsten Verlauf des Wochenbettes kompliziert waren. Die Ursache dafür ist in dem Unterlassen der sofortigen Naht des kommunizierenden Scheidenrisses zu suchen. Die Jodoformgazedränage nach der Scheide wirkte *nicht* infektionshemmend, sondern infektionsfördernd. Hat man die Entstehung einer kommunizierenden Scheidenverletzung nicht verhüten können, so bietet der allseitige vollkommene Nahtabschluß des Operationsgebietes, vor allem aber nach der Vagina zu, noch die beste Gewähr für ein normales Wochenbett mit ungestörter Wundheilung. Sollte es trotzdem zu einer Infektion der Wunde kommen, so ist jederzeit die Möglichkeit gegeben, dem entzündlichen Sekret durch einfaches Öffnen der unteren Inzisionswunde genügenden Abfluß zu verschaffen und durch Einlegen eines Dränrohres eine Verhaltung des Sekretes zu verhindern.

In den 13 Fällen, wo gröbere Weichteilverletzungen vermieden wurden, bzw. Risse überhaupt nicht eintraten, war das Wochenbett mal fieberlos (Achselhöhlentemperatur nicht über 38,0°). In Fall 15 war 38,2° am 10. Tage p. op. auf Koprostase, 38,5° am 23. Tage p. op. auf eine Angina zu beziehen; im übrigen verlief die Rekonvaleszenz ungestört. — 2mal (Fall 3 u. 14) fand das 39° etwas überschreitende Fieber innerhalb der ersten 10 bzw. 8 Tage seine Erklärung in einer ausgedehnten eiterigen Bronchitis, bei glatter Wundheilung und normaler Genitalrückbildung. — 1mal (Fall 5) verursachte ein vorübergehendes schmerzhaftes Ödem der Operations- und angrenzenden Inguinalgegend bei hühnereigroßem Hämatom im Labium majus leichte Temperaturerhöhung (am 2. und 3. Wochenbettstage 38,4° in axilla, sonst normale Temperatur). — In Fall 16 entwickelte sich bei nur an-

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fänglich starkem Ödem des linken Labium majus unter flüchtigem Anstie der Körperwärme, bis zur Höchsttemperatur von 38,4° in der Achsel höhle, und lange anhaltender ausgesprochener Pulsbeschleunigung ei embolischer Prozeß der rechten Lunge. Der Sägeschnitt war in diesem Fall versehentlich durch das weit medialwärts reichende For. obturatorium hindurchgeführt worden. — Bei dem 19. Falle, der bereits fiebernd i die Klinik eintrat, vermochte die Naht der Inzisionswunden, obwoh sie sofort nach Durchsägung des Knochens noch vor der an di Hebosteotomie anschließenden künstlichen Entbindung mit vaginalem Kaiserschnitt angelegt wurde, eine Infektion der Knochenwunde und ihrer Umgebung nicht zu verhüten. Es folgte ein schwerer pyämischer Zustand, der nach vier auf den 3.—5. Wochenbettstag sich verteilenden Schüttelfrösten in ein leicht fieberhaftes Stadium überging und allmählich in Heilung auslief. Aus dem durch den unteren Inzisionsschnitt dränierten Infektionsherd entleerte sich reichlich Eiter und nekrotische Gewebsfetzen (Streptokokken, ebenso im Sekret des Genitaltraktus).

Die absolute Morbiditätsziffer des Wochenbettes bei unsern Hebosteotomien mit kommunizierenden Scheidenrissen beträgt demnach (5 von 7 Fällen) 71,4%, die auf Erkrankung des Hebosteotomie-Wundbezirkes reduzierte 57,1%. Bei unsern Hebosteotomien ohne kommunizierende Scheidenverletzungen dagegen beläuft sich die absolute Wochenbettsmorbidität auf 46,1% (6 von 13 Fällen), die auf Erkrankung des Knochenwundbereiches reduzierte auf 23%. Fassen wir alle Wochenbettserkrankungen der 20 Hebosteotomien zusammen, so erhalten wir eine Gesamtmorbidität von 55%. Es handelte sich:

1 mal um Koprostase, später in demselben Falle Angina,
2 mal um ausgedehnte eiterige Bronchitis,
1 mal um eiterige Peritonitis (vor Einlieferung in die Klinik infiziert), ohne Infektion der Hebosteotomiewunde,
7 mal um leichtere oder schwerere Erkrankungen der Hebosteotomiewunde und deren Folgen:
  1 mal leichte Eiterung aus der dränierten unteren Inzisionswunde,
  1 mal stärkeres Ödem im Wundbereich bei Hämatom des Labium majus,
  1 mal Schenkelvenenthrombose auf der Operationsseite,
  2 mal Thrombophlebitis mit Lungenembolie,
  2 mal Wundeiterung mit Pyämie.

Vorläufig ist die Wochenbettsmorbidität nach Hebosteotomie eine recht hohe. Es wird aber gelingen, sie künftig wesentlich

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günstiger zu gestalten. Wie unsere Erfahrungen lehren (Fall 4 u. 19), müssen wir nur alle sicher infektiösen Fälle und solche, die außerhalb der Klinik bei langer Geburtsdauer, womöglich vorzeitigem Blasensprung, vielfach von Arzt und Hebamme untersucht worden sind und uns auf Infektion sehr verdächtig erscheinen, von der Operation ausschließen, weil hier die Hebosteotomie für die schon sehr gefährdete Mutter eine schwere Komplikation darstellt, während die für die Mutter schonende Perforation des ohnehin womöglich schon infizierten Kindes die besten Chancen für die Erhaltung des mütterlichen Lebens bietet (cf. den Standpunkt Döderleins Verh. d. D. G. f. Gyn. 1907, 12, S. 170 ff. und v. Franqués ebendaselbst S. 231). Wir müssen ferner die Indikation nicht allein nach der Form des verengten Beckeneingangs, sondern unter Berücksichtigung aller Beckenverhältnisse, speziell des Beckenausgangs und unter besonderer Würdigung der weichen Geburtswege stellen und mit einer gut ausgebildeten Technik die Gefahren der Hebosteotomie (Blutung, Hämatombildung, Harnblasenverletzung, kommunizierende Scheidenrisse und deren Folgen) sorgsam umgehen lernen. Alsdann wird unter beträchtlicher Herabsetzung der Morbiditätsziffer auch die Wochenbettsdauer wesentlich verkürzt werden. Bei glattem Verlauf der Operation und reaktionsloser Wundheilung braucht der Klinikaufenthalt nicht länger zu währen, als nach einer unkomplizierten spontanen Entbindung. In Fall 17 und 18 standen die Operierten am 4. Tage, in Fall 20 am 9. Tage auf und wurden am 14. bzw. 12. und 15. Tage nach Hause entlassen.

Da 1 Hebosteotomierte gestorben ist, und sich 1 (Fall 19) noch in der Klinik befindet, sind im ganzen 18 Frauen zur Entlassung gekommen. Sie waren sämtlich beschwerdelos, ihr Gang ungestört und schmerzfrei. — Eine dauernde Schädigung der Artt. sacroiliacae infolge der Kontinuitätstrennung des Beckenringes ist bis jetzt niemals eingetreten. Wohl klagte eine Frau (Fall 14: allgemein verengtes rachitisch plattes Becken mit Conj. vera 8,1 cm) bei den ersten Gehversuchen über leichtes Schmerzgefühl in der Kreuzdarmbeinfuge auf der nicht operierten Seite. Dieser Schmerz war aber binnen wenigen Tagen völlig geschwunden. Auch die noch in der Klinik befindliche Frau (Fall 19: allgemein verengtes rachitisch plattes Trichterbecken, auffallend schlecht aufschließbar) klagte während der ersten Wochen p. op. über heftige Schmerzen im Kreuz, vor allem in der Gegend der rechten Art. sacroiliaca. Die Schmerzen sind jetzt nach ca. 6 Wochen nur noch angedeutet, die Kreuzdarmbeinfuge auf Druck nicht empfindlich. Die Frau ist außer Bett und lernt allmählich immer besser gehen, so daß voraussichtlich auch in diesem Fall ein gutes

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definitives Gehvermögen erzielt wird. — Die Sägestelle markier sich bei der Aufnahme des Entlassungsbefundes entweder als ei Delle oder als ein schmaler, bis höchstens ½ cm breiter Knochenspa Kallus wurde 10mal ganz vermißt, 8mal in geringer Menge und n 1mal als dickerer Wulst (Fall 1) gefunden. Die Knochenenden war meist nur leicht, manchmal gar nicht und 4mal deutlicher gegeneinand verschieblich. Eine Schmerzhaftigkeit im Operationsgebiet bestan niemals. Die Weichteilwunden, auch die Rißwunden in der Vagin waren dicht geschlossen und unempfindlich. Von den 3 intra oper tionem entstandenen Hämatomen waren nur noch Spuren vorhande Bezüglich der vorderen Scheidenwand war ein Unterschied gegen über den Situsverhältnissen vor der Operation nicht zu konstatieren nur 1mal zeigte sich eine Zystozele mäßigen Grades, die vor de Operation nicht bemerkt worden war (Fall 11).

Betreffs des Dauererfolges ziehe ich nur die Hebosteotomierte heran, bei denen die Operation mindestens ½ Jahr zurückliegt. Da sind 12 Fälle, über deren Zustand und Ergehen ich entweder schrift lichen bzw. mündlichen Bericht erhalten habe (4 Fälle), oder die ich selbst habe nachuntersuchen können (8 Fälle). Alle Frauen gaben an, voll arbeitsfähig und in ihrem Gehvermögen nicht ge schädigt zu sein. In Fall 2 hatte einige Wochen hindurch leichte Ermüdbarkeit des der Operationsseite entsprechenden Beines ohn nachweisbare Atrophie oder Degeneration der Muskulatur bestande In den 8 nachuntersuchten Fällen (½ Jahr bis 2¾ Jahr p. op.) wurd an der Sägestelle kein oder kein nennenswerter Kallus, 2mal ein schmale, wenig vorspringende Knochenleiste gefühlt. Ein wirklicher Knochenspalt existierte in keinem Falle mehr; die nur in 1 Fall (Fall 11 7 Mon. p. hebost.) noch etwas gegeneinander beweglichen Knochen enden hatten sich lückenlos zusammengefügt. Von einer Bruch pforte oder Vorwölbung eines Baucheingeweides im Operations gebiet war bisher bei genauester Untersuchung in keinem Falle etwas zu entdecken. Die Zystozele in Fall 11 hatte sich nicht vergrößert und machte keine Beschwerden. — Eine weitere Geburt haben wi bei den Hebosteotomierten bis jetzt nicht zu beobachten Gelegenheit gehabt. 1 Operierte (Fall 3) ist zur Zeit Gravida mens. VIII. Von einem reellen Maßzuwachs der Conj. vera infolge der Knochendurch sägung haben wir uns in diesem Falle ebensowenig wie bei den andern nachuntersuchten Frauen überzeugen können. Für die bevorstehende Geburt haben wir die Wiederholung der Hebosteotomie geplant.

Entsprechend den guten primären Erfolgen für Kind und Mutter und entsprechend den bisher günstigen Dauerresultaten ist die Zahl der Hebosteotomien an unserer Klinik von Jahr zu Jahr gewachsen

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e sind allmählich immer mehr Fälle von engem Becken der Hebosteotomie zugefallen, wie sich leicht aus der folgenden Übersicht ernehmen läßt:

1903: 1 Fall,
1905: 3 Fälle,
1906: 5 Fälle,
1907: 7 Fälle,
1908: 4 Fälle im ersten Vierteljahr.

Wie stellen wir nun an unserer Klinik die Indikation für die Hebosteotomie, und wie grenzen wir sie jetzt ab gegenüber den konkurrierenden Operationen: hohe Zange, Perforation des lebenden Kindes, prophylaktische Wendung, künstliche Frühgeburt und Kaiserschnitt aus relativer Indikation?[1])

Die Gefahren der hohen Zange bei engem Becken, speziell für das Kind, habe ich schon bei der Besprechung unserer Hebosteotomie-Kinder erläutert, ihre Unrichtigkeit als Voroperation vor der Hebosteotomie betont, und sie nur für die Geburtsfälle reserviert, bei denen die beckenerweiternde Operation nicht vorgenommen werden soll, weil die dazu notwendigen Vorbedingungen fehlen: erfahrener, operationsfähiger, technisch bewanderter Geburtshelfer, geschulte Assistenz, geeignete Räumlichkeit. Hier darf die hohe Zange bei eingetretener Konfiguration und günstiger Einstellung des Kopfes nur als Rettungsversuch für das Kind betrachtet werden, bei dessen Mißlingen trotz energischer, aber nicht roher Handhabung des Instrumentes die Perforation des lebendes Kindes die alleinige logische Konsequenz ist. Muß man die hohe Zange außerhalb der Klinik als notwendiges Übel anerkennen, so sollte sie in der Klinik aus der Reihe der geburtshilflichen Operationen bei engem Becken ganz gestrichen werden. Um Mißverständnisse zu vermeiden, hebe ich noch einmal hervor, daß in die Rubrik hohe Zange nicht solche Fälle eingereiht sind, bei denen der Kopf bereits mit seiner größten Zirkumferenz im Beckeneingang steht oder schon den Beckeneingang passiert hat. — Anders steht es mit der Perforation des lebenden Kindes. Das Gebiet dieser Operation ist ja außerordentlich eingeschränkt worden, gerade weil

[1]) J. Pfannenstiel, Die Indikationsstellung zur Behandlung der Geburt bei Beckenenge. D. Med. Wochenschr. 1906, S. 1654—1656; J. Pfannenstiel, Diskussionsbemerkung zu dem Vortrag Rühl, Zur Technik der Pubiotomie in der Mittelrhein. Ges. f. Geb. u. Gyn. Monatsschr. f. Geb. u. Gyn. 1906, Bd. 24, S. 415; J. Pfannenstiel, Diskussion über beckenerweiternde Operationen. Verhandlungen D. Ges. f. Gyn. 1907, XII, S. 351—356.

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wir im Besitze der Hebosteotomie den hohen Forceps wenigst klinisch beseitigen konnten. Außerhalb der Klinik bleibt die Pe ration des lebenden Kindes nach erfolglosem hohen Forceps die v geschriebene Operation. Aber auch in der Klinik wird ein gewis Prozentsatz lebender Kinder der Perforation anheimfallen, nämlich d Kinder, die schon vor der Einlieferung der Kreißenden in die Kli mit hoher Zange vergeblich bearbeitet worden sind und na unserer Überzeugung schwer gelitten haben, und die Kind schwer infizierter oder auf schwere Infektion höchst ve dächtiger Gebärender. Bei diesen dürfen wir zwecks Entbind keine komplizierten Wundgebiete schaffen, weil wir sie gegen fektion nicht zu schützen vermögen. Ohne die Garantie für Geburt eines intakten Kindes leisten zu können, würden wir der Anlegung von Wunden die schon bedrohte Mutter noch me gefährden, während wir ihr durch die viel weniger eingreifende u schonendere Perforation des zwar lebenden, aber bereits mehr od weniger geschädigten Kindes weit bessere Chancen bieten. Freili ist es nicht immer leicht, darüber ins klare zu kommen, ob ei fiebernde Kranke als infiziert anzusehen ist oder nicht. Sind darüber im Zweifel, so entscheidet das Interesse der Mutter. — die prophylaktische Wendung betrifft, so gibt sie nur dann Resultate, wenn es sich nicht um eine Enge des ganzen Gebur kanales, sondern nur um einen engen Beckeneingang handelt, also das platte Becken mit weitem Schambogen, und um weite de nungsfähige Weichteile. Hat sich bei einem platten Becken mäßig verkürzter Conj. vera, etwa bis 8 cm herunter, der Kopf u günstig eingestellt, in Hinterscheitelbeineinstellung, Stirnlage oder G sichtslage, oder bleibt die Konfiguration des Kopfes bei der Vorde scheitelbeineinstellung ungenügend, so ist die prophylaktische Wendu indiziert, vorausgesetzt natürlich, daß die Größe und Härte des Kopf den Erfolg nicht illusorisch erscheinen lassen, und die Wendung übe haupt noch ausgeführt werden darf. Allgemein gleichmäßig vereng und allgemein verengte platte Becken, sowie auch platte Becken engen oder rigiden Weichteilen eignen sich nicht für die prophyl tische Wendung, weil die schnell zu überwindenden Widerstände v zu groß sind (Pfannenstiel). Erscheint der Kopf bei plattem Beck und mäßiger Verkürzung der Vera auffallend groß und hart, ist d Zeit für die Wendung womöglich verpaßt, oder ist die Conj. ve stärker verkürzt, etwa von 7,5 cm abwärts, so kommt nur die He osteotomie in Frage. Als untere Grenze für die Hebosteotomie b plattem Becken lassen wir 7 cm oder wenig darunter gelten. Ist de Kopf nur mittelgroß und konfigurabel, der Beckenausgang sehr ge

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einig, so kann man auch noch ausnahmsweise weiter mit der Vera- größe heruntergehen: Fall 3 Conj. vera von 6,6 cm und Fall 10 Conj. ra von 6,2 cm. Mit solcher Verkürzung des geraden Durchmessers m Beckeneingang sind wir bereits in das Gebiet des Kaiserschnittes n relativer Indikation eingetreten. Diese Operation war auch für n Fall 10 in Aussicht genommen.[1]) Die besonderen Umstände (ein- hende Narkosenuntersuchung seitens mehrerer Untersucher am Tage r der Geburt und unerwarteter Blasensprung mit Nabelschnurvorfall le vor die Vulva, bevor die Kreißende auf das Geburtsbett gelagert rden konnte) zwangen aber, auf die geplante Operation zu ver- chten. Die schnell ausgeführte Hebosteotomie und anschließende Wendung und Extraktion unter Zuhilfenahme der Walcherschen Hängelage ergab ein ausgetragenes lebendes Kind, allerdings mit iner nicht unerheblichen Impression des hinteren Scheitelbeins. In diesem Fall ist die Walchersche Hängelage mit Vorteil für das Kind nd ohne Schädigung der hinteren Beckengelenke ausgenutzt worden. Gegen ihre Anwendung bei der subkutanen Hebosteotomie ist nichts einzuwenden (Werth), falls es sich nicht um ein schlecht aufschließ- bares Becken mit geringer Exkursionsbreite der Artt. sacro-iliacae handelt.

Für die allgemein gleichmäßig verengten und allgemein verengten ienen Becken mit engem Schambogen liegt die Indikation zur Heb- steotomie bei wesentlich größeren Maßen, etwa bei einer Conj. vera on 8—7,5 cm, und reicht bei ungünstiger Relation zum Kopf sogar ch höher bis zu 9 cm Vera und mehr. Diese Fälle sind deshalb ungünstig, weil mit der allgemeinen Beckenverengerung ganz ge- öhnlich eine Hypoplasie und mangelhafte Dehnbarkeit der weichen eburtswege vergesellschaftet ist (Pfannenstiel). Außerdem pflegt e Wehentätigkeit eine mangelhafte zu sein, und die Artt. sacroiliacae nd zuweilen so wenig beweglich, daß die Spaltung des Beckenringes ur zu einem geringen Klaffen der Knochenwunde und somit nur zu nem minimalen Raumgewinn führt. Bei diesen Beckenformen mit r Hebosteotomie 7,5 cm Vera nach unten zu überschreiten ist sehr kant, weil dann die Kreuzdarmbeinfugen zu leicht geschädigt und e Weichteile schwersten Verletzungen ausgesetzt werden. Hier be- nnt bei den allgemein verengten Becken der Kaiserschnitt aus rela- ver Indikation in sein Recht zu treten. — Welch schöne Erfolge mit r Hebosteotomie auch bei den allgemein verengten Becken erzielt erden können, wenn man sie nur richtig wertet, ersieht man aus ll 14 u. 15. In Fall 14 (II p. mit allgemein verengtem rachitisch

1) R. Werth, Verhandl. d. D. Ges. f. Gyn. 1907, XII, S. 343 u. 344.

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platten Becken: Conj. vera 8,1 cm) fiel bei 3markstückgroßem Os die kräftig pulsierende Nabelschnur vor. Nach Hebosteotomie Eröffnung des äußeren Muttermundes mittels drei Inzisionen wu ein gut mittelgroßes lebendes Kind durch Wendung und Extrak gewonnen. — In Fall 15 (Ip. mit allgemein verengtem rachitisch pla Becken: Conj. vera 8,1 cm) machten sich Kompressionserscheinun der vorliegenden Nabelschnur bemerkbar bei unerweitertem Os Nach Aufheben aller Hindernisse (Hebosteotomie, vaginaler Kai schnitt, tiefe Scheidendamminzision) lieferte die Wendung und traktion ein lebendes mittelgroßes Kind.

Wie sollen wir uns aber verhalten, wenn wir in der Schwang schaft ein enges Becken zu beurteilen haben, wie stellen wir zu der prophylaktischen Operation intra graviditatem, zu künstlichen Frühgeburt? Wir haben bei dem verarbeiteten M terial 6mal Gelegenheit gehabt, 5—6—8 Wochen ante terminum üb die künstliche Frühgeburt zu entscheiden. Wir haben sie in drei Fällen abgelehnt; und zwar in Fall 7 (IIp. mit allgemein gleichmä verengtem Becken: Conj. vera 8,4 cm; relativ kleiner Kopf), weil noch auf eine Spontangeburt bei ausgetragenem Kinde hofften, ander seits bei eintretenden Schwierigkeiten die Hebosteotomie in Reser hatten. — In Fall 14 (IIp. mit allgemein verengtem rachitisch plat Becken: Conj. vera 8,1 cm, querer Durchmesser des Beckeneinga 11,4 cm; enger Schambogen) erschien uns der Erfolg für das Ki bei frühzeitiger Entbindung zu unsicher. — In Fall 2 (IIp. mit a gemein verengtem rachitisch platten Becken: Conj. vera 6,9; weit Beckenausgang), in Fall 5 (IVp. mit allgemein verengtem rhachitis platten Becken: Conj. vera 7,0 cm; nicht verengter Schambogen), Fall 11 (Ip. mit allgemein verengtem rachitisch platten Becke Conj. vera 6,85 cm; mittelweiter Schambogen) und in Fall 13 (IV mit rachitisch plattem Becken: Conj. vera 7,2 cm; sehr weiter Scha bogen) kam die künstliche Frühgeburt wegen der zu starken Ve kürzung der Conj. vera nicht in Frage. Wenn aber auch die Ei leitung der künstlichen Frühgeburt durch die Hebosteotomie ei wesentliche Einbuße erfahren hat, so wird sie doch immer ihre G tung behalten. Als Maße kommen für sie in Betracht: bei allgeme verengten Becken Verae von etwa 8—8,5 cm, bei platten Becken Ver von etwa 8—7,5 cm.[1 u. 2]) Falls die künstliche Frühgeburt bei den a gegebenen Maßen nicht früher als in der 36. Woche eingeleitet wi

1) R. Werth, Diskussion über beckenerweiternde Operationen. Verh. d. Ges. f. Gyn. 1907, XII, S. 345 u. 346.

2) J. Pfannenstiel, ebd. S. 352—356.

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ist die Chance des frühgeborenen Kindes sehr gut und die Pro-
e auch nach der Entlassung aus der Klinik günstig, falls es sich
Mütter handelt, die mit der Pflege des Kindes vertraut sind und
dieselbe angelegen sein lassen.

Die genannten Zahlen können natürlich nur einen Anhalts-
kt für unser Handeln beim engen Becken geben. Jeder
will nach genauester Feststellung der Becken- und Weichteilver-
nisse, nach Prüfung des Geburtsobjektes und seiner Relation zu
Geburtswegen und nach Berücksichtigung des Allgemeinzustandes
Mutter und der lebenswichtigen Organe für sich beurteilt werden.
müssen streng individualisieren und nicht nur bezüglich
Indikationsstellung, sondern auch bezüglich der Technik
Operation, wenn wir uns für die Hebosteotomie entscheiden.
delt es sich um platte Becken oder um allgemein verengte platte
ken mit weitem Beckenausgang, um eine gute Entwickelung, Ela-
ität und Sukkulenz der weichen Geburtswege, so brauchen wir die
stehung einer schwereren Rißverletzung nicht zu fürchten, wenn
uns einigermaßen an die festgesetzten Maße halten. Ob wir dann
ch Döderlein oder Kroemer operieren, ist in solchem Falle
ichgültig. Ist aber ein enger Beckenausgang vorhanden (allgemein
ichmäßig verengtes oder allgemein verengtes plattes Becken mit
hem und engem Schambogen) und entsprechend hypoplastische,
e und wenig dehnbare Weichteile, dann heißt es entspannen, wo
n nur kann. Unter solchen Umständen muß man die Scheiden-
nd an der so sehr gefährdeten Sägestelle mobilisieren, also die
oemersche Schnittführung wählen, ev. einen entspannenden
eidendammschnitt hinzufügen. Es macht durchaus den Eindruck,
ob wir dadurch schwereren Rißverletzungen (Scheide und Harnblase)
dem Wege gehen können, zumal wenn wir gleichzeitig den Rat
annenstiels[1]) berücksichtigen, beim Herumhebeln des Kopfes um
Symphyse nicht die Sägestelle als Hypomochlion zu benutzen,
ndern den Kopf mehr nach hinten und unter Belastung der nicht
erierten Seite herauszuleiten. Haben wir uns in einem Falle mit
stigen Beckenausgangs- und Weichteilverhältnissen, bei dem wir
o die Entstehung eines kommunizierenden Scheidenrisses nicht zu
ärtigen haben, für die Hebosteotomie entschieden, obwohl ein
ämischer Zustand besteht oder mit der entfernten Möglichkeit
er Infektion zu rechnen ist, so empfiehlt sich dagegen sicherlich
hr das Döderleinsche Verfahren gegenüber dem Kroemerschen,
il bei der Döderleinschen Methode die untere dem Genitalkanal

[1]) J. Pfannenstiel, Monatsschr. f. Geb. u. Gyn. 1906, Bd. 24, S. 415.

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näherliegende Wundöffnung die bei weitem kleinere is
Knochenwundgebiet deshalb vor einer ev. drohenden
seitens der Geburtswege leichter bewahrt bleiben kann.
waigen komplizierenden Rißverletzungen, sei es der Scheid
der Harnblase, müssen sofort durch Naht exakt geschlossen
Auch ohne daß sich eine Verletzung der Harnblase ereignet h
zweckmäßig in den ersten Tagen ein Dauerkatheter eingele
man auf diese Weise imstande ist, bei der Harnentleerung de
von dem Wundgebiet fernzuhalten und häufigere intensivere Beweg
des durchsägten Beckens fürs erste zu verhüten. — Einen Be
gurt haben wir nur in den ersten 10 Fällen angewandt, dann
fortgelassen, weil sich eine derartige Fixierung des Beckenringes
unnötig erwiesen hat. — Die unter exakter Vereinigung der Rän
vernähten Inzisionswunden bleiben am besten ohne Deckverband u
nur unter dem Schutze eines desinfizierenden und austrocknend
Streupulvers (Dermatol). Unter einem Heftpflasterverband kommt
zu leicht eine sekundäre Infektion der Wunde zustande.

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Geburtsverl… zur Oper…	das Kind: Dauer-Resultat	Bereits publiziert	Indikationsstellung:
50stündiger … ext. ganz e… rk gebeugte B… n Beckeneingan… Symphyse gen… g. T. 36,8°, Pu… e Herztöne da… ßig. Trotz kräf… igkeit kein G… ritt. Vergeb… ge.	21. V. 07, *über 4 J. alt*: Gesundes, kräftiges Kind. Rechts vor dem Tub. par. kleine narbige Stelle ohne Haare.	*Hohlweg* Z. f. Gyn. 1905, Nr. 42, S. 1281. — Döderleins Kongreßtabellen V. d. D. Ges. f. Gyn. XII, S. 116, Nr. 17.	Starkes Mißverhältnis zwischen Kopf und Beckeneingang.
ca. 10stündiger …hentätigkeit Os…: Nach …. Fruchtwasse…erer Er…eflossen. Kopf… Beines …r dem Beckenei… Degene…hts abgewichen; …er Bein… Ungestörte H…zt *ganz* Kindes. T. 36… hlbefin…	VII. 05, *gut 1/4 J. alt*: Kind prächtig gediehen, gesund.	*Hohlweg* l. c. — Döderleins Kongreßtabellen l. c. S. 136, Nr. 57.	Wegen der starken Verkürzung der C. vera 6 W. ante terminum auf die Einleitung der künstlichen Frühgeburt verzichtet und Hebosteotomie bei rechtzeitiger Geburt beschlossen.
14stündiger krä… …tätigkeit bei erw…gemein… …tswegen Kopf in…terleibs… …agung extrame…*lehindert*. … dem Beckenein… …t der Symphyse… …r verlaufend; 1. … 1 Stunde *Mekoni*…hend der …ei kindliche He…ervorra… …ernd regelmäßig …rsprung; …entlich an Freque…eine Be… …etzt. T. 37,5° m…enenden …la.	…en ersten 10 Mona… …kräftig, dann „eng…he Krankheit", hat …t im 3. Lebensjahre …fen gelernt.	*Hohlweg* l. c. — Döderleins Kongreßtabelle l. c. S. 136, Nr. 58.	Fötale Indikation zur Geburtsbeendigung. Starkes Mißverhältnis zwischen dem ungünstig eingestellten Kopf und Beckeneingang. Leichter Temperaturanstieg intra partum.
…ittiger Fruchtwass… …h mehr als 60stün… …ft sehr wechselnde… …igkeit *Os ext. 5*… …pf beweglich auf d… …eingang; 1. Stellun… …pfgeschwulst. *Abg*… *…hlichem Mekonium*… …lmäßige Herztätig… …des. Keine Tem… …hung. Puls etwas b… …. Kreißende fühlt s… …d und erschöpft.	Staphy… …riechen… …r auße… …ke eit… …ndat i… Der Z… Reizun…	Kin…chen alt, Todesurs…e nicht bekannt. A… 51 … bip… to-… um… ent…	*Hohlweg* l. c. — Döderleins Kongreßtabelle l. c. S. 138, Nr. 59.	Fötale und mütterliche Indikation zur Geburtsbeendigung. Wegen des dringenden Verlangens der „alten Erstgebärenden" nach einem lebenden Kinde im Interesse des Kindes Hebosteotomie nach Dilatation mit Bossi.

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499.

(Gynäkologie Nr. 184.)

Über Sterilität.[1]

Von

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Unter dem hier abzuhandelnden Thema verstehe ich die Unfähigkeit des geschlechtsreifen Weibes zur Fortpflanzung trotz eines in normaler Weise stattfindenden geschlechtlichen Umganges.

Bei der großen Wichtigkeit der Sterilität im Haushalte der Familie, des Staates und der Nationen ist es nicht zu verwundern, daß schon die ältesten Völker ihr Augenmerk auf die Tauglichkeit zur Fortpflanzung richteten.

In ihren Sitten und Gebräuchen finden wir eine ganze Reihe Einrichtungen, die offenbar diesem Zwecke dienten oder doch mindestens ihn nebenbei verfolgten.

So zerstören nach Ploß die chinesischen Kinderwärterinnen den neugeborenen Mädchen den Hymen durch Einführen des Fingers in die Scheide. Die Exzision der Klitoris in Abessinien soll darin ihren Grund haben, daß die abessinische Klitoris wegen ihrer Größe den späteren Zeugungsakt hindern oder erschweren könnte. In dieser Weise beschnittene Weiber hält man in der Tat in Ecuador zur Kohabitation und Fortpflanzung für fähiger und geschickter.

Die Sitte, junge heranwachsende Mädchen für einige Zeit aus dem Dorfe zu entfernen und sie über ihre kommende Bestimmung zu unterrichten, der Brauch, die eingetretene erste Menstruation als Zeichen der erlangten Fortpflanzungsfähigkeit festlich zu begehen, die dabei vorgenommenen Manipulationen, wie Kneten der Brüste, Einschieben von Gegenständen in die Vagina lassen ihren Zweck ohne weiteres erkennen.

1) Nach einer am 12. Februar 1908 gehaltenen Probevorlesung.

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Nach indischem Brauche ist es für einen Vater eine Schande, eine mannbare Tochter noch ledig zu Hause zu haben. Und bei anderen Völkerschaften mußte Ehelosigkeit begründet werden, Ledigsein galt als Vergehen oder Schande, während Heirat und Zeugung prämiiert wurden.

Im Alten Testamente sah man die Fruchtbarkeit als eine besondere Gnade des Himmels an. Die Verpflichtung der alten Juden, die kinderlose Witwe des Bruders zu heiraten, diente doch nur der Fortpflanzung.

Bei den Griechen und Römern brachte die Niederkunft einer Frau nicht nur persönliche und gesellschaftliche, sondern auch rechtliche Vorteile.

Im altdeutschen Rechte war Unfruchtbarkeit ohne weiteres ein Grund zur Ehescheidung und in Japan kann man heute noch zu einer sterilen Gattin eine zweite hinzunehmen.

Es ist sonach nicht zu verwundern, daß schon die alten Ärzte, wie Hippokrates, Plinius u. a. sich mit der Sterilität beschäftigt haben.

Aus dem Mittelalter sei nur an die Ansicht von Soranus aus seiner Abhandlung über die Zeichen der mutmaßlichen Fruchtbarkeit erinnert: Da die große Mehrzahl der Ehen nicht um der Wollust willen, sondern der Erzielung von Nachkommenschaft wegen geschlossen wird, so ist es ganz sonderbar, daß man dabei mehr auf ahnenreiche Abstammung und Vermögen Gewicht legt, statt zu berücksichtigen, ob die Frau zur Konzeption fähig und zum Gebären gut gebaut ist.

Neben diesen altruistischen Interessen spielt die Sterilität auch für das Einzelwesen nicht selten eine bedeutende Rolle. Wenn wir für unsere heutige Zeit der Ansicht des unverbesserlichen Weiberfeindes Schopenhauer, wonach jedes Weib, das kinderlos stirbt, den einzigen Zweck, zu dem es taugt, verfehlt hat, auch nicht generell zustimmen können, so drückt sich doch in dem von Ellen Key geforderten „Rechte auf die Mutterschaft“ ein starker Trieb aus, den zu befriedigen das Weib ein Bedürfnis und Recht habe. Ihre Worte „Liebe will Zukunft, nicht Augenblicke“ verraten die Sehnsucht nach dem Kinde, den Mutterschaftstrieb.

In seinem bekannten Buche „Also sprach Zarathustra“ sagt Nietzsche: „Alles im Weib ist ein Rätsel und alles im Weib hat seine Lösung: Sie heißt Schwangerschaft. Der Mann ist für das Weib ein Mittel, der Zweck ist immer das Kind.“

Gewiß betrachten nicht wenige Frauen der Gegenwart nicht das Kind als unmittelbaren Zweck der Liebe, aber das Weib, das die Mutterschaft überhaupt nicht und nie wünscht, ist doch wohl eine Ausnahme.

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Zur Illustration dafür, wie hoch einzelne hervorragende Geister die Mutterschaft einschätzten, genüge der kurze Hinweis darauf, daß Napoleon I. die Frau für die beste hielt, die am meisten Kinder geboren hat. Und Zola bezeichnet in seinem Romane „Fécondité" als modernes Schönheitsideal ein Weib mit einem Kinde an der Brust und einem an den Knien. (Kisch.)

Ehe wir selbst die Frage beantworten, ob von unserem heutigen ärztlichen Standpunkt aus die Zeugung um jeden Preis so hoch zu bewerten sei, wollen wir erst die Ursachen der Sterilität untersuchen. In der Formulierung des Themas liegt es begründet, daß die vom Manne ausgehenden Veranlassungen der Sterilität unerörtert bleiben. Da aber erfahrungsgemäß in ca. $^1/_3$ aller sterilen Ehen die Schuld allein an dem Manne liegt, so sei eigens hervorgehoben, daß in praxi keine gegen Sterilität gerichtete Therapie eingeleitet werden soll, ehe die Potentia coëundi und generandi des Mannes erwiesen ist. Erstere muß anamnestisch feststehen und bezüglich der letzteren legen wir bekanntlich ein sehr großes Gewicht auf die normale Beweglichkeit der Samenfäden unter dem Mikroskope. Die Verläßlichkeit dieses Kriteriums hat Torkel in den letzten Tagen angezweifelt. Er weist auf die Versuche von Fick hin, wonach die Spermatozoen des Frosches, die durch Frierenlassen unbeweglich waren, nach dem Auffrieren wieder beweglich werden können, ohne indes befruchtungsfähig zu sein.

Noch schwerer ist die Beurteilung der Sterilitätsursachen beim Weibe: Das Ei ist uns überhaupt nicht zugänglich. Die Betastung der Generationsorgane ist sehr schwer und bei der Bewertung unserer Befunde führen subjektive Momente leicht zu einer Überwertung: so spielt z. B. in Marienbad die Fettleibigkeit eine große Rolle als Sterilitätsursache, in Franzensbad die Gonorrhöe und bei einem renommierten Operateur das Myom, weil sich hier jeweils Kranke bestimmter Kategorien ansammeln. Unter den polnischen Jüdinnen fand Fraenkel die Gonorrhöe weit seltener als Sterilitätsursache denn sonst, weil sie bei der frühen Heirat der jungen Leute überhaupt seltener sei.

Sodann ist die Fertilität der Frau an gewisse äußere Momente geknüpft, die zwar zum großen Teil selbstverständlich sind, die aber dennoch berücksichtigt werden müssen, um nicht fälschlich von Sterilität zu sprechen: Die Dauer der Geschlechtsreife währt in unserer Zone ca. vom 16—50 Jahre. Die Unfruchtbarkeit jenseits dieser Grenzen bedarf als physiologische Erscheinung keiner weiteren Erörterung. Bei anderen Völkerschaften ist es wieder anders. Mütter im Alter von 10 Jahren soll es z. B. bei den Buschmännern geben. Das frühe Erlöschen der Konzeptionsfähigkeit bei den Orientalen ist bekannt. Fruchtbarkeit im höheren Alter findet sich am ehesten im nördlichen

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Europa: Irland, England, Dänemark, Schweden. Doch sind Konzeptionen jenseits des 50. Jahres im ganzen nicht gar so selten. Ich kenne selbst einen Fall von Niederkunft im 58. Jahre, 28 Jahre nach der letzten Geburt und 10 Jahre nach Eintritt des Klimax.

Während wir gewöhnt sind, Fruchtbarkeit nur zu finden in den zeitlichen Grenzen der Menstruation, weicht dieser Fall hiervon erheblich ab. Er bestätigt die Ausnahme und illustriert die Tatsache, daß Menstruation und Ovulation nicht immer nebeneinander hergehen müssen. Anhaltende Amenorrhöe verbürgt ja noch nicht absolute Sterilität. Wir kennen Beobachtungen von mehrfachen Niederkünften, ohne daß die betreffenden Mütter einmal in ihrem Leben menstruiert hätten.

Auch innerhalb der Grenzen des geschlechtsreifen Alters dürfen wir noch nicht von Sterilität reden, wenn nicht gleich der ersten Kohabitation die Konzeption folgt, wie es beim Tiere die Regel ist. Das ist wichtig für die Definition der sterilen Ehe. Nur in etwa 15% aller Fälle erfolgt die erste Geburt 10 Monate nach der Heirat, in 6% läßt sie 3 und mehr Jahre auf sich warten. Nach 5 Jahren aber konzipieren nur noch $^1/_{40}$ aller Frauen. Daraus ergibt sich, wie falsch es ist, nach einjähriger Ehe von Sterilität zu reden. Mit einiger Wahrscheinlichkeit können wir das erst nach 3 oder besser noch nach 5 Jahren tun.

Zur Erklärung der zeitlichen Differenz im Eintritt der ersten Konzeption nach der Heirat hat man auf verschiedene Momente hingewiesen. Ich will nur einiges erwähnen: Statistische Erhebungen über die Geburtenzahl haben ergeben, daß einzelne Monate ganz besonders fruchtbar waren. Man hat danach ein allgemeines zeitliches Konzeptionsoptimum im Frühling annehmen wollen und glaubte, daß zwar einesteils die in diese Zeit fallenden Feste und Vereinsversammlungen die Vereinigung der Geschlechter begünstigen, andererseits aber doch auch mit der Wiederkehr des Frühlings, der Besserung der Erwerbs- und Nahrungsverhältnisse eine Hebung der Lebens- und Reproduktionskraft verknüpft sei. Ein für die einzelne Frau verschiedenes zeitliches Konzeptionsoptimum hat Cohnstein konstruieren wollen, da ihm aufgefallen war, daß Mütter von mehreren Kindern immer um dieselbe Jahreszeit niederkamen.

Sodann hat man dem Alter, in dem die Ehe eingegangen wird, einen Einfluß zugeschrieben. Von den jugendlichen Frauen, die zwischen 15 und 19 Jahren heiraten, gebären nur etwa 20% innerhalb der ersten 10 Monate nach der Ehe, während diese Zahl z. B. bei denen, die mit 33 heiraten, sich verdoppelt. (Kisch.)

Dazu kommt noch, daß zuweilen mangelhafte Übung im sexuellen

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erkehr unfreiwillig, oder die soziale Stellung und die von ihr
)hängenden wirtschaftlichen Verhältnisse den Zeitpunkt der ersten
eburt freiwillig hinausschieben.

Diese allgemeinen Gesichtspunkte dürfen nicht unerörtert bleiben,
enn es sich darum handelt, den Ursachen der Sterilität im Einzel-
lle nachzuspüren. Das Hauptgewicht ist aber doch auf die somatische
ntersuchung des speziellen Individuums zu legen.

Wenn wir bedenken, daß zur Reproduktion des Einzelwesens in
er Hauptsache drei Bedingungen seitens des Weibes erforderlich sind:

1. die Bildung und der Austritt eines Eies aus dem Follikel,
2. die Wegsamkeit der von Sperma und Ei zu durchwandernden Bahnen,
3. die Möglichkeit der Beherbergung und Bebrütung des befruchteten Eies,

) zerfallen die Sterilitätsursachen in natürlicher Weise in drei große
ruppen:

I. **Anomalien der Keimbildung,**
II. **Anomalien, die zur Kontaktbehinderung zwischen Sperma und Ovulum führen,**
III. **Anomalien der Nidation und Bebrütung.**

Sind die die Sterilität verursachenden Momente angeboren, dann
eden wir von angeborener, sind sie erworben, von erworbener
terilität.

Primäre Sterilität liegt vor, wenn nach 5jähriger Ehe überhaupt
eine Konzeption erfolgt; sekundäre Sterilität haben wir, wenn min-
lestens 5 Jahre lang nach einer Konzeption, mag sie mit Abort oder
echtzeitiger Niederkunft enden, eine erneute Befruchtung nicht mehr
rfolgt.

Als absolute Sterilität bezeichnen wir den Zustand, bei dem ein
Konzeptionshindernis vorliegt, das auf keine Weise zu beseitigen ist,
. B. Defekt des Uterus. Relativ steril nennen wir dagegen z. B. einen
otenten Mann, der mit einem bestimmten, zeugungsfähigen Weibe
inen befruchtenden Beischlaf nicht auszuüben vermag.

Ich habe diese einzelnen Termini angeführt, weil sie in der Lite-
atur sehr durcheinander geschmissen werden und dadurch viel Ver-
irrung angerichtet wird.

Betrachten wir nun

die Anomalien der Keimbildung!

Sie lassen sich wieder in drei Gruppen zerlegen:

a) In einer ersten Gruppe finden wir pathologisch-anatomische Veränderungen lokaler Natur (am Ovarium);

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b) in einer zweiten Gruppe handelt es sich um pathologisch-anatomische Veränderungen allgemeiner Natur (am übrigen Körper,

c) in einer dritten Gruppe haben wir kein anatomisches Substrat.

Ia. Unter den die Keimbildung beeinträchtigenden lokalen Affektionen verdienen zunächst

die Entwicklungsanomalien des Ovariums

Erwähnung.

Bei gänzlichem Fehlen der Ovarien ist die Sterilität ohne weiteres selbstverständlich.

Es genügt aber auch schon der Defekt oder die mangelhafte Entwicklung der funktionierenden Substanz, des Follikelapparates. Sei es nun, daß überhaupt keine Eier gebildet werden, oder daß diese, nur mangelhaft gereift, im Follikel abortiv zugrunde gehen oder wenigstens nicht befruchtungsfähig sind. Eine etwa eintretende Menstruation beweist nichts. Wir sehen sie ja auch bei vielen heranreifenden Mädchen der Zeugungsfähigkeit lange vorausgehen.

Die Diagnose dieser Zustände ist nicht leicht und trotzdem sehr verantwortungsvoll.

Auf den Defekt oder die rudimentäre Ausbildung der Ovarien deuten zuweilen schon die mangelhaft entwickelten sekundären Geschlechtscharaktere hin. Zeichen von Hypoplasie und Infantilismus am übrigen Genitale oder am Körper sind wertvolle Indizien. Wegen ihrer häufigen Vergesellschaftung mit diesen Prozessen kommt einer etwa bestehenden Tuberkulose oder Chlorose große Bedeutung zu.

Handelt es sich um einen Defekt, dann fehlt beim Tastbefund das Ovarium. Aber nur der geübte Untersucher, der sich zutrauen darf, keinen Schlupfwinkel übersehen zu haben, ist berechtigt, mit einiger Wahrscheinlichkeit Defekt der Keimdrüse anzunehmen, wenn er sie nicht gefühlt hat.

Das mangelhaft ausgebildete Ovarium fühlt sich bei Fehlen des Follikelapparates klein und an seiner Oberfläche glatt an. In einem abnormen Hochstand haben wir vielleicht noch eine Reminiszenz infantiler Verhältnisse.

Selbstverständlich ist nur die Beteiligung beider Ovarien von Bedeutung. Ich betone das, weil nach einer alten Theorie des Parmenides und Anaxagoras die Knaben aus dem einen und die Mädchen aus dem anderen Eierstock stammen sollen.

Nächst den Entwicklungsanomalien kommt den

Tumoren

(angeborenen und erworbenen) eine Bedeutung zu.

Ovarialtumoren

verdrängen entweder das funktionierende Gewebe ganz oder die letzten

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ollikel erliegen dem Drucke der gegen sie vordringenden Geschwulst. ies besonders bei gutartigen Tumoren genügen noch die minimalsten rarialreste zum Zustandekommen einer Konzeption, wie das Zuammentreffen von Kolossaltumoren und Gravidität beweist.

Am radikalsten räumen die doppelseitigen malignen Geschwülste t der funktionierenden Substanz auf. Dazu vermindern sie durch hwere konstitutionelle Schädigungen die Reproduktionskraft in sich eblich, so daß Sterilität eintreten kann, obschon anatomisch noch llikelgewebe nachzuweisen ist.

Auch den

Uterustumoren

uß man einen Einfluß auf die Keimbereitung zuerkennen. Die bei erusmyom häufig zu beobachtende kleinzystische Degeneration der varien stellt eine schwere Schädigung des Follikelapparates dar und e von Bulius beschriebene Angiodystrophia ovarii bedeutet auch ch Pfannenstiel eine so eingreifende Ernährungsstörung des Eierockes, daß seine normale Funktion aufhört. Indes darf ich die Anht Hofmeiers nicht unerwähnt lassen, wonach bei Myom die Eierockstätigkeit und damit auch die Konzeptionsmöglichkeit länger als ast wachgehalten wird. Die Häufigkeit der Sterilität bei Myom ist iner Meinung nach nur eine scheinbare. Aber er blieb mit seiner uffassung ziemlich vereinzelt.

Unter den die Keimbildung störenden lokalen Affektionen wären dlich noch

entzündliche Prozesse

nennen, die sich im Innern des Eierstockes oder an seiner Oberiche abspielen.

Parenchymatöse Entzündungen können die Eibildung durch Zerörung des Follikelapparates aufheben, durch Schädigung desselben e Eireifung erheblich beeinträchtigen oder doch durch Erkrankung s Eies selbst die Befruchtungsfähigkeit herabsetzen, z. B. durch Verderungen an der Eiperipherie, die den Samenfäden den Eintritt möglich machen.

Erkrankungen, die vom Stroma aus auf das Parenchym übergreifen, ben selbstverständlich dieselben Folgen haben. Gehen sie mit hronischer Induration und Verdickung einher, wie bei Stauungsprosen z. B. nach schweren unkompensierten Herzfehlern, dann kann er Follikelapparat dem Drucke des wuchernden Bindegewebes ganz rliegen oder die Follikel werden von ihm so umlagert, daß sie nicht hr nach der freien Oberfläche hin platzen können.

Diesen letzten Effekt haben auch alle jene entzündlichen Erkrankungen, die mit Adhäsions- und Schwartenbildung an der Eier-

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stocksoberfläche einhergehen, auch ohne daß sie zu krankhaften Veränderungen im Ovarium selbst führen.

Für alle die genannten Erscheinungen kommt ätiologisch die ganze Summe der akuten und chronischen Entzündungen resp. Eiterungen, die sich im Eierstock gelegentlich als Perioophoritis, Oophoritis oder Pyovarium etablieren, in Betracht: Puerperalfieber, Strepto- und Staphylokokkeninfektion bei irgendwelchen anderen Gelegenheiten, metastatische Eiterungen nach Sepsis, Typhus u. dgl., Tuberkulose und Gonorrhöe.

Eine der wichtigsten Rollen spielt in praxi gerade die Gonorrhöe, freilich weniger durch Zerstörung des Parenchyms, als vielmehr durch Veränderungen an der Oberfläche, die das Platzen des Follikels hindern. Des anderen durch sie bedingten Momentes, der Verlegung des Tubenlumens, habe ich im nächsten Abschnitt zu gedenken.

Alle diese Prozesse können ausheilen. Und wenn der Follikelapparat nicht gänzlich zerstört ist, kann auch diese Form von Sterilität heilbar sein.

Anhangsweise sei der Traumen gedacht, die direkt oder durch Blutung das Ovarium zerstören, ähnlich wie es bei der von Ploß berichteten Sitte oder Unsitte, junge Mädchen durch Zusammendrücken der Eierstöcke steril zu machen, geschieht.

Ib. Ich komme nun zu den **Affektionen allgemeiner Natur** am übrigen Körper, welche die Keimbildung hemmen können.

Sie können vorübergehende oder dauernde Sterilität verursachen. Ihr Einfluß ist zuweilen sekundär an den Generationsorganen (Atrophie, Klimacterium praecox) nachzuweisen, in anderen Fällen fehlt jede Spur hiervon. Darum ist die Art ihrer Bedeutung auch nicht immer klar, aber das Faktum derselben steht fest. Gemeinsam ist ihnen allen eine Konsumption der Kraft des Organismus. Diese Rolle spielen im Körperhaushalt:

α) jede Sorte lang anhaltenden Fiebers (Typhus, Puerperalfieber usw.),
β) schwere Blutkrankheiten (Anämie, Leukämie usw.),
γ) Stoffwechsel- und Konstitutionsanomalien (Diabetes, Fettleibigkeit, Chlorose usw.),
δ) schwere Nerven- und Geisteskrankheiten (Basedow, Psychosen usw.),
ε) eine Reihe Gifte, unter denen Alkohol und Morphium eine große Rolle spielen,
ζ) chronische Infektionen (Lues und Tuberkulose usw.).

Ich kann sie nicht alle einzeln erörtern. Nur die wichtigsten: **Tuberkulose, Chlorose, Fettleibigkeit und Alkoholismus** seien herausgegriffen.

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Die Bedeutung der **Tuberkulose** liegt jedenfalls in der genannten weren konstitutionellen Schädigung. Die Konsumption der Körperft bedeutet eine Konsumption der Reproduktionskraft.

Bezüglich der **Chlorose** nahm man früher an, daß es durch mangelte Kongestion zu den Genitalien nicht zur Eireifung und zum ıtzen des Follikels komme. Heute halten wir die Chlorose für e Entwicklungskrankheit, die mit der konstitutionellen Minderrtigkeit den Keim zur Sterilität in sich trägt.

Auf die Rolle der **Fettleibigkeit** wirft die Beobachtung am Tier bedeutungsvolles Licht. Die Tierzüchter wissen, daß Mästung der rtilität schadet. Die gemästete Truthenne hört auf Eier zu legen. Am Menschen ist aufgefallen, daß während sonst unter ca. 10 Ehen sterile sich befindet, dies bei Fettleibigen schon unter 4 der Fall . Das ist schon Hippokrates bekannt gewesen, der die geringe rtilität der Szythinnen auf ihre Adipositas zurückführt. Amenorrhöe er spärliche Menstruation bei fetten Frauen deuten oft auf eine ıngelhafte Funktion der Ovarien hin. Die Wirksamkeit zahlreicher gen Sterilität empfohlener Badekuren hat in der Beseitigung der ttleibigkeit ihren Grund.

Worin liegt nun der Einfluß derselben begründet?

Zunächst ist daran zu erinnern, daß es sich für manche Fälle gar cht um Anomalien der Keimbildung handelt, sondern nur um mechasche Kohabitationsbehinderung, die dann zu dem in Rede stehenden apitel nicht gehört.

Für die Mehrzahl der Fälle müssen wir indes mit P. Müller anehmen, daß die Ablagerung von Nährmaterial in Form einer Fetthäufung die Bildungsvorgänge im Ovarium hindert, aber ohne eine nderung der Eierstöcke selbst zu verursachen, so daß nach Schwund es Fettes wieder Ovulation eintreten kann.

F. Fraenkel sucht den Grund in einer ausbleibenden Rückbildung es Corpus luteum. Dadurch sistiere die Ovulation und ihr folge le Fettleibigkeit. Er weist auf das Tier hin, wo mit Persistenz des Corpus luteum Ovulation und Brunst aufhören. Das zeitliche Verhältnis wischen Ausbleiben der Ovulation und Eintritt der Fettleibigkeit ist lso umgedreht, eine Reihenfolge, die wir auch für die klimakterischen Veränderungen als möglich zulassen müssen. Ohne die Frage weiter u diskutieren, kann man jedenfalls das sagen: das Ovarium ist ein eines Reagens auf die Vorgänge im Körperhaushalt. Seine Nichtnktion zeigt an, daß im Körper ein Übermaß von Ernährungsstoffen ntweder abnormer Weise wie beim Diabetes oder physiologischer Weise wie bei der Laktation oder durch Ansatz an unrichtigen Stellen nd in unrichtiger Form (Adipositas) verbraucht wird.

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Ob der mangelhaften sexuellen Erregsamkeit Fettleibiger eine Bedeutung zukommt, wäre erst zu entscheiden, wenn es sicher feststünde, daß Fettleibige, wie Schopenhauer meint, weniger erregbar sind und daß die Voluptas sexualis auf das Zustandekommen der Befruchtung Einfluß hat. Ich komme auf diese Frage noch zurück.

Auf die Möglichkeit, daß **Alkohol** die Fertilität beeinträchtige, ist man namentlich in England aufmerksam geworden, wo bekanntlich konzentrierte Alkohole konsumiert werden. Indes fand eine französische Arbeit dies nicht bestätigt. Erhebungen über 402 Pariser Arbeiterfamilien ergaben, daß 81 Trinkerehen unter ihnen sogar fruchtbarer waren, als die anderen. Wenn indes der Alkoholismus zu schweren körperlichen Schädigungen geführt hat, dann kann er nach ziemlich allgemeiner Ansicht die Konzeption hindern und zwar wahrscheinlich durch Beeinträchtigung des Keimes. In anderen Fällen sollen auch chronische Oophoritis oder die beim Alkoholismus zuweilen einsetzende Fettleibigkeit eine Rolle spielen.

Ic. Haben wir bisher immer noch für die Erklärung einer Unfruchtbarkeit einen somatischen Befund verantwortlich machen können, so fehlt dieser in einer dritten Gruppe von **Sterilität ohne somatischen Befund** gänzlich. Es bleibt uns dann nur übrig, unsere Unkenntnis durch Hypothesen und rein empirisch gewonnene Tatsachen zu bedecken.

Zunächst eine Reihe äußerer Momente. Solche sind z. B. der Einfluß von Klima und Jahreszeit. Sie sind bekanntlich für die Fruchtbarkeit des Tieres von großer Wichtigkeit. Für den Menschen haben sie wenig Bedeutung. Wichtiger ist dagegen der plötzliche Wechsel der Lebensweise und des Klimas: so sollen die Europäerinnen in den Tropen ihre Fertilität verlieren, wie auch gewisse sonst wildlebende Tiere in der Gefangenschaft sich nicht mehr fortpflanzen, sei es daß sie sich überhaupt nicht mehr begatten, oder die Begattung fruchtlos bleibt.

Auf die Begünstigung der Fertilität durch Ernährungs- und Temperaturverhältnisse hat schon Darwin hingewiesen. Nur ein Beispiel! Die wilde Ente legt im Jahre 5—10 Eier, während ihre zahme Schwester es auf 80—100 bringen kann. Im warmen Taubenschlag finden sich zuweilen schon im Januar Eier, während dies sonst erst im Frühling der Fall ist.

Zu diesen äußeren Momenten kommen nun noch einige in den Einzelwesen begründete hinzu.

Von altersher hat man die Blutsverwandtschaft als Grund der Sterilität angesehen. Darum verbot schon Moses die Ehe zwischen

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hen Blutsverwandten. Unsere Beobachtungen am Tiere belehren
ıs desgleichen.

Das Aussterben vornehmer Familien hat man auf die Inzucht rückgeführt. So fand z. B. Goehlert in einigen europäischen Herrherhäusern 32% sterile Ehen gegen sonst ca. 10%. Zur Erklärung eser Erscheinung hat man die Vermutung einer verringerten Ovuion ausgesprochen. Diese soll in nervösen Einflüssen infolge der stinktiven Abneigung gegen den leiblichen Vetter ihren Grund haben.

Damit haben wir nun die Frage nach der Bedeutung der Konitution, des Temperamentes und der Geschlechtslust beihrt. Sie ist schwer zu beantworten. Oft genug stehen uns nur ganz dividuelle Anschauungen zu Gebote.

Zunächst der erste Punkt, Konstitution und Temperament. 'ährend der Koran die Frauen mit brauner Haut für fruchtbarer ilt als die mit heller, liegt doch wohl unter der Voraussetzung, daß iebe am besten auf dem Boden sexueller Harmonie gedeiht, dem ekannten: „ich liebe die schlanken, die blonden, die Mädchen mit leichem Gesicht“ eine gegenteilige Auffassung über die sexuelle rauchbarkeit zugrunde. In dem oben erwähnten Rat des Soranus, ıan solle bei der Wahl einer Frau auf ihre vermutliche Fruchtbarkeit ücksicht nehmen, ist die Anerkennung von somatischen Zeichen der exuellen Leistungsfähigkeit ausgedrückt: Ein lebhafter brünetter Mann nd eine sanfte Blondine sollen besonders gut zueinander passen.

Die Bedeutung der Geschlechtslust, des Orgasmus, für die ıusstoßung eines Eierstockeies wird sehr verschieden beurteilt. Sicher ıt jedenfalls das: zum Zustandekommen einer Konzeption ist der)rgasmus des Weibes nicht unbedingt nötig. Die Schwängerung nach lotzucht, oder, wenn man diese wegen der im letzten Augenblick vielleicht doch noch eingetretenen sexuellen Erregung nicht für stichıaltig hält, die Konzeption in tiefer Narkose oder anderen bewußtosen Zuständen beweisen dies.

Dieser Argumentation steht aber eine andere nicht schlecht gestützte Ansicht gegenüber. Zur Illustration der Bedeutung einer geschlechtlichen Erregung für den Ablauf physiologischer Vorgänge überhaupt wies schon Schopenhauer darauf hin, daß die jungen Städterinnen bei der Fülle der sexuellen An- und Aufregungen früher menstruieren, als ihre Schwestern auf dem Lande. Ja Kisch erzählt von einer Dame, die nach eigener Aussage willkürlich konzipieren könne, je nachdem sie sich zu geschlechtlicher Erregung hinreißen lasse oder nicht. Und bei gewissen Völkern soll sich auf das absichtliche passive Verhalten sub coitu ein direktes System zur Konzeptionshinderung aufgebaut haben.

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Auch bei uns kann man, wie Kisch hervorhebt, zuweilen die Beobachtung machen, daß die treulose Ehefrau von ihrem Ehemann trotz Erfüllung aller sonstigen Bedingungen nicht empfängt, während sie vom Buhlen, zu dem sie die Liebe im Herzen trägt, schwanger wird. Sie liefert uns das Beispiel einer auf Dispareunie begründeten relativen Sterilität. Ein bekannter historischer Beleg ist Napoleon I., der aus seiner Ehe mit Josefine keine Kinder hatte, obschon sie schon Nachkommen besaß, während er mit Marie Luise einen Sohn zeugte.

Der Wert, den van Swieten der sexuellen Erregung für das Zustandekommen einer Konzeption beimaß, ergibt sich deutlich daraus, daß er, wegen Sterilität der Kaiserin Maria Theresia gefragt, den Rat gab: mamillam esse titillandam.

Indes so interessant diese ganze Untersuchung ist, ich kann nicht weiter auf sie eingehen. Ihre Bewertung fällt zusammen mit der Frage des weiblichen Sexualempfindens überhaupt. Das ist das Geheimnis des Weibes. Aber wenn wir vom Standpunkte des sittlich ernsten Arztes ein wenig hineinleuchten, dann können wir keine unberufenen Eindringlinge sein. Nur einzelne Proben der divergenten Anschauungen! Nach Hegar empfindet das Weib sexuell weniger als der Mann. Lombroso meint, daß sich das Weib sehr gerne vom Manne umwerben läßt, aber seinen Wünschen gibt es sich nur wie ein Opfertier hin. Und Adler hat sogar ein eigenes Buch geschrieben über die mangelhafte Geschlechtsempfindung des Weibes.

Nach anderen Anschauungen ist das Weib überhaupt lauter Geschlecht, eine breite Geschlechtsfläche und muß es sein, weil seine ganze Karriere von der Sexualität abhängt. Kisch ist der Ansicht, daß der Sexualtrieb des Weibes als eine machtvolle, elementare Gewalt die Begattung verlangt, auch wenn Furcht vor Konzeption besteht oder von Konzeption keine Rede mehr sein kann. In Irrenanstalten, wo alle anerzogenen Hemmungen wegfallen, soll sich das deutlich zeigen. Einer solchen Auffassung entstammen wohl auch die Worte Diderots: „Das einzige, was die Frau von Grund aus gelernt hat, ist: das Feigenblatt, das ihre Stammutter Eva ihr vererbte, mit Anstand zu tragen."

Auch die Frauen selbst beurteilen den Sexualtrieb ihrer Stammesschwestern verschieden. Rosa Mayreder z. B. unterscheidet verschiedene Typen.

Sie sehen, meine Herren, wir befinden uns auf einem sehr unsicheren Gebiete. Ein Untersucher hat diese Unsicherheit mit den geistreichen Worten gekennzeichnet: „Die Frau ist ein Komma, der Mann ein Punkt. Hier weist du woran du bist, dort lies weiter."

Zum Schlusse dieses ganzen Abschnittes noch der kurze Hinweis

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auf, daß die Keimbildung zuweilen beeinträchtigt wird durch Homo-
ualität und geschlechtliche Überanstrengung, wie die Sterilität von
)stituierten, auch beim Fehlen anderer Sterilitätsursachen zeigt.
Endlich nimmt man, so paradox es auch klingen mag, eine gewisse
liche Disposition an, entweder so, daß es sich um eine ererbte
were Entwicklungsfähigkeit der Eier handelt, oder so, daß den
:rmatozoen der Eintritt in das Ei erschwert ist, analog den Beob-
ıtungen von Schenk, der an verschiedenen Tiereiern sah, daß der
ıtritt der Samenfäden verschieden schwer ist.
Die in früherer Zeit gemachte Annahme, daß die Frauen, die mit
em männlichen Zwilling zur Welt kamen, steril seien wegen mangel-
fter Ausbildung des Genitalapparates, besteht jedenfalls in diesem
ıne nicht zu Recht.

Anomalien, die zu einer Kontaktbehinderung zwischen Sperma und Ovulum führen.

Sie zerfallen in drei Gruppen:

a) Momente, welche die Deportation des Samens im Scheidengewölbe erschweren oder ausschließen,
b) Momente, welche die Retention des an normaler Stelle abgesetzten Samens beeinträchtigen,
c) Momente, welche die Wanderung der Spermatozoen nach oben unmöglich machen.

Zu der ersten Gruppe gehören kurz gesagt alle jene Anomalien, e rein mechanisch oder auch nur reflektorisch eine Immissio :nis hindern.

Die mechanisch wirksamen sind angeborene Defekte oder rudi-entäre Entwicklung der Scheide, Verengerung des Introitus durch umoren, Entzündungen, narbige Schrumpfungen, Totalprolapse usw.

Wenn gelegentlich auch die Ablagerung von Sperma am äußeren ienitale für eine Konzeption genügen kann, so finden die Samenfäden on hier aus doch nur ausnahmsweise den Weg nach oben, so daß ie Sterilität bei Erkrankungen der genannten Art gut verständlich ist.

Totalprolaps führt indes seltener zu Sterilität; denn es wird der 'orfall bei der Kohabition reponiert oder, wenn dies nicht der Fall st, so kann unter Umständen das Sperma direkt in die Cervix uteri rgossen werden.

Die reflektorisch ausgelösten Hindernisse der Kopulation werden lurch den Vaginismus dargestellt. Wir verstehen darunter eine ıochgradige Hyperästhesie des Introitus, so daß das Vordringen des 'enis einesteils wegen heftiger Schmerzen nicht mehr zugelassen wird, ınderenteils wegen krampfartiger Kontraktion des Constrictor cunni

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nicht möglich ist. Ob dieser Erscheinung eine der vielen beschuldigten anatomischen Veränderungen oder eine rein nervöse Ursache (Abneigung gegen den Mann) zugrunde liegt, soll hier unerörtert bleiben.

Zum Verständnis der aufgestellten zweiten Gruppe (fehlende oder mangelhafte Retention des Samens im Scheidengewölbe) weise ich zunächst darauf hin, daß wir unter normalen Verhältnissen die Bildung einer kleinen Samenlache im hinteren Scheidengewölbe, von der aus die Aszension der Samenfäden stattfindet, annehmen. Dieser Mechanismus kann gestört sein durch alle angeborenen oder erworbenen Affektionen, bei denen das Scheidengewölbe fehlt oder räumlich reduziert ist: Hypoplasie, Atrophie, entzündliche Veränderungen, Lageanomalien.

Mit dem Scheidengewölbe fehlt auch das Receptaculum seminis und der Same fließt aus der Vagina ab, besonders wenn der Scheidenschlußapparat durch Erschlaffung und Defekte im Beckenboden und Dammrisse Not gelitten hat, da nach Kisch gewöhnlich eine Kontraktion des Constrictor cunni das Sperma eine Zeitlang unter einer gewissen Spannung retiniert hält. Indes darf nicht unerwähnt bleiben, daß nach anderer Ansicht gerade die Aktion des Konstriktor den Samenabfluß befördert. So soll es Frauen geben, die durch willkürliche Kontraktion des Beckenbodens sich des Spermas im Strahl erledigen, ehe Konzeption eintreten kann.

Ich komme nunmehr zu den Ursachen, welche die Wanderung der Samenfäden nach oben erschweren. Sie zerfallen in mechanische und chemische.

Unter den ersteren finden wir zunächst eine Kategorie, die den Samenfäden den Zugang zum Wege, der in sich normal sein kann, erschwert. Zum Verständnis wieder ein kurzer Hinweis auf die Physiologie! Im Orgasmus soll der Uterus tiefer treten, sich der Glans penis nähern oder es soll mindestens der in der Cervix steckende Kristellersche Schleimpfropf in die Samenlache eintauchen, so daß an ihm die Spermatozoen wie an einer Leiter emporsteigen. Wenn dem so ist, dann fehlt mit dem ausbleibenden Orgasmus auch die beschriebene Leiter.

Eine ähnliche Erschwerung des Zuganges zum Uterus soll in der Retroflexio liegen, weil die Portio in den Samensee nicht eintaucht.

In einer zweiten Kategorie handelt es sich um Verlegung des Pfades, auf dem die Wanderung nach oben stattfinden soll. Am Collum uteri sind es neben, durch Narbenschrumpfung oder Tumoren erworbenen Stenosen namentlich angeborene Verengerungen oder Knickungen durch Lageanomalien, auf die Sims seinerzeit ein so großes Gewicht legte. Wir sind heute geneigt, die Hauptursache der Sterilität

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ht in diesen mechanischen Momenten zu suchen, sondern in r, der abnormen Enge des Cervicalkanals oder der Lageanomalie grunde liegenden Hypoplasie und konstitutionellen Minderwertigkeit. i erworbenen Retroflexionen liegt die Schuld oft genug in den ichzeitig bestehenden entzündlichen Prozessen des Endo- oder Peritriums, welch letztere die Lageanomalie veranlaßt haben können.

Im Cavum uteri kommen Verödung der Höhle durch Tumoren er medikamentöse Einwirkungen in Betracht. Bezüglich des letzten nktes sei an den Vorschlag von Pincus erinnert, die Vaporisation s Cavum uteri zur Erzeugung von Sterilität auszuführen.

Der Tubenkanal kann eine Beeinträchtigung erfahren durch angerene Schlängelung, durch Knickung infolge abnormer Verwachsungen d Befestigungen und hauptsächlich durch entzündliche Verödungen ines Lumens oder Verklebungen seines Fimbrienendes, wie wir sie i Tuberkulose, septischen Infektionen und Gonorrhöe oft finden. ie Hauptrolle fällt in praxi der Gonorrhöe zu, auf deren ernste Beutung zuerst Noeggerath in klassischer Weise hinwies. Wenn auch e Angaben Doctors, daß über 80% aller Männer, und die Statistik von laschko, wonach von den über 30 Jahre alten alle zweimal vor der Hei gonorrhoisch infiziert waren, die Wirklichkeit hinter sich läßt (Erb), illustriert sie doch die erschreckende Häufigkeit der Gonorrhoe nd ihre wichtige Rolle in der Ätiologie der Sterilität. Sehr viele rauen empfangen zugleich mit dem Sperma die Gonokokken ihres lannes und werden steril oder die im ersten Wochenbett sich ausildenden Pyosalpingen führen zur Einkindsterilität.

Anhangsweise sei hervorgehoben, daß man für die Wanderung er Spermatozoen auch darin ein Hindernis erblickte, daß ein reichcher von oben kommender Sekretstrom sie herausschwemme oder ie Samenfäden durch ein besonders zähes Sekret sich nicht hindurchinden können.

Diesen Sekretanomalien kann auch noch ein chemischer Einfluß uf die Beweglichkeit der Samenfäden anhaften. Es ist z. B. bekannt, aß diese am besten gedeihen in dem alkalischen Cervixsekret, während ie im sauren Sekret absterben (Katarrh). Solchen Änderungen der ekretreaktion liegt häufig ein bakterieller Prozeß zugrunde. Die akterien können dabei das Endometrium so verändern, daß die permatozoen untergehen oder vielleicht scheiden sie den Samenäden gefährliche Toxine aus. Wir wissen das noch nicht.

Anomalien der Nidation und Bebrütung des Eies.

Haben sich Ovulum und Sperma auch getroffen, so brauchen wir für das Zustandekommen einer normalen Konzeption noch die Nidation auf dem Endometrium.

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Diese kann zunächst schon daran scheitern, daß das Tubenlumen verlegt ist und das befruchtete Ei auf seiner Wanderung aufgehalten wird. Ich verweise nur auf die ganze Ätiologie der Extrauterin-gravidität.

Aber auch im Cavum uteri glücklich angekommen, hindern lokale und allgemeine Erkrankungen die Implantation.

Unter den lokalen Affektionen nenne ich nur die angeborene Unfähigkeit des Uterus, ein Ei zu beherbergen, wie sie so oft der Hypoplasie eigen ist, ferner gut- und bösartige Tumoren, die bald das Cavum uteri verlegen, bald die Schleimhaut gänzlich zerstören, Lageanomalien, sämtliche entzündlichen Erkrankungen des Endometriums u. a. m. Hinsichtlich bakterieller Endometritiden sei für die Gonokokken eigens darauf hingewiesen, daß nach mancher Ansicht eine Eiimplantation auf einem gonorrhoischen Endometrium denkbar ist.

In anderen Fällen liegt die letzte Ursache in schweren konstitutionellen Schäden, Stoffwechsel-Blutkrankheiten, Herz-Nierenleiden, welche die Unfähigkeit der Eibebrütung bedingen. Solchen Momenten kommt zuweilen eine um so größere Bedeutung zu, als es nach Ansicht einzelner Autoren mit Ausnahme der Atresie für den Eintritt der Spermatozoen ins Cavum uteri kein absolutes Hindernis gibt.

Sämtliche bis jetzt besprochenen Sterilitätsursachen werden wirksam ohne unser Zutun, ja oft genug gegen unseren direkten Wunsch und Willen. Von dieser unfreiwilligen Sterilität unterscheidet sich die beabsichtigte, freiwillige, die wir kennen als **fakultative Sterilität** und **operative Sterilisierung.**

Unter **fakultativer Sterilität** verstehen wir die absichtliche Hinderung der Konzeption durch präventiven geschlechtlichen Verkehr. Den bedeutenden Einfluß dieses Faktors illustriert eine Statistik von Körösi, die sich auf die Fruchtbarkeit der Frauen zwischen 40 und 44 Jahren erstreckt. Unter den Neuvermählten betrug sie ca. 20%, unter den übrigen nur 5%. Da es sich um gleiches Alter handelt, kann die Verminderung der Fertilität auf $^1/_4$ nur durch das Nichtwollen der länger Verheirateten erklärt werden.

Die Idee, die Fortpflanzung zu modifizieren, ist alt, wie die Festlegung eines gesetzlichen Alters für die Heirat, die Aussetzung von Kindern, Kindesmord, künstlicher Abort usw. zeigen. Zum ersten Male trat gegen Ende des 18. und zu Anfang des 19. Jahrhunderts der Engländer Thomas Robert Malthus öffentlich für sie ein. Er empfahl Beschränkung der Fortpflanzung durch sexuelle Abstinenz und späte Heirat, weil er für das Ausreichen der Nahrung fürchtete. Seine Anschauungen fanden Beifall zunächst bei dem sog. Malthusschen Bund. Die Malthussche Liga in England und der Sozialharmonische Verein in

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itschland empfahlen dann zu diesem Zwecke antikonzeptionelle tel.

Aber trotzdem blieb diese ganze Lehre nicht unangefochten. Ich ne nur Liebig und Bebel, welche die Anschauung von der Unzuglichkeit der Nahrung sehr in Frage zogen. „Die Menschen verıren, nicht sie vermindern, das ist der Rat, der im Namen der tur an die Menschheit geht", ruft ein anderer Gegner des Malthusen Systems. Roosevelt, der Präsident der Vereinigten Staaten, t mit Bezug auf Konzeptionsverhinderung[1]): Jene, welche sich ihrer rantwortlichkeit durch den Hang nach Unabhängigkeit, Bequemlicht und Wohlleben entziehen, begehen ein Verbrechen gegen ihre ıse und sollten der Gegenstand der Verachtung und des Abscheus ein gesundes Volk sein.

Doch wollen wir die gegenteilige Anschauung nicht minder gewichr Persönlichkeiten nicht ungehört lassen. Hegar empfiehlt die gulierung der Fortpflanzung, um die Qualität auf Kosten der Quanit zu verbessern. Und der Hygieniker Gruber hält die Massenıgung für nutzlos, weil ihr ein Massensterben entspricht. Ja ein ıtor versteigt sich zu der Behauptung: „Die Zeit wird kommen, wo ler Patriot es als seine Pflicht ansehen wird, nicht im Krieg möghst viel Feinde zu töten, sondern im Frieden möglichst wenig nder zu zeugen."

Noch eine Reihe Momente individueller Natur, die sich auf Gendheit der Mütter und Kinder, wirtschaftliche Interessen u. a. beehen, sind von den sog. Neomalthusianisten beigebracht worden. h kann auf sie nicht einzeln eingehen. Ihre hohe Bewertung in ankreich kommt zum Ausdrucke in dem bekannten: „Ah l'amour, ımour — c'est le plaisir d'un jour pour le regret d'neuf mois." ınen wurden ebensoviele Bedenken ethischer, moralischer, rechtcher Natur entgegengehalten.

Wir sehen Ansicht gegen Ansicht. Jede für sich gut gestützt. Welıer soll der Arzt folgen, wo im eigenen Lager Fehde herrscht? So ellt Forel die Forderung, man solle die Zeugung von der Befrieigung des Sexualtriebes trennen als allgemeinberechtigt auf, während aenger[2]) sagt: gut geschultes geburtshilfliches Personal wird mehr ur Gesundung der Frauenwelt beitragen als die Humanitätsbestreungen der Neomalthusianer . . . Woferne die Frau elend ist durch chädigungen im Wochenbett, so gilt es, diese zu beseitigen. Wofern ie blaß ist infolge Mangels guter Nahrung, so ist es Aufgabe der Gesellschaft, ihr zu helfen.

1) Zit. nach Kisch, l. c. S. 420.
2) Zit. nach Kisch, l. c. S. 425.

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Wir haben also keine Normen. Im eigenen Innern müssen wir die Entscheidung für unser Handeln suchen. „Wie würde ich für mich in diesem Falle handeln?“, das ist die einzige verläßliche Richtschnur.

Daß es nicht Sache des Arztes sein kann, mit einem Rate rein die Bequemlichkeit zu unterstützen, liegt in seinem Berufe begründet.

Daß er aber seinen Rat da nicht verweigert, wo die Mutterschaft physische Gefahren bringt, ist eine Pflicht, die ihm eben wieder sein Beruf auferlegt. Wenn es einem verkommenen Verbrecher von der Straße erlaubt ist, in der Notwehr einen hochverdienten Mann zu töten, dann muß es einer Frau erlaubt sein, durch Verhinderung der Schwangerschaft ihr Leben zu retten.

Schwerer ist die Frage zu beantworten, ob auch soziale Rücksichten den Arzt bestimmen sollen, antikonzeptionelle Mittel anzuraten. Kisch lehnt das rundweg ab. Es sei Sache der Nationalökonomen, da zu helfen.

Wenn es sich nur um soziale Momente handelt, dann geht sie den Arzt als solchen nichts an; darin muß man Kisch beipflichten. Aber wie oft sind Armut, schlechte Ernährung, mangelhafte Gesundheit, ausgehungerte, elende, widerstandslose Kinder zusammen vergesellschaftet! Da berühren sich die Indikationen des Arztes, des Hygienikers, des Rassenhygienikers und des Nationalökonomen oft so eng, daß man sie unmöglich trennen kann.

Ich kann auf die uns zu Gebote stehenden Mittel zur Erreichung der fakultativen Sterilität leider nicht mehr eingehen. Sie sind zum Teil in den oben abgehandelten Sterilitätsursachen schon genannt und zerfallen in:

A. physiologische,
B. artifizielle.

Unter den physiologischen wäre zu nennen die gänzliche sexuelle Abstinenz, wie sie Tolstoi in seiner Kreutzersonate predigt, die zeitweilige sexuelle Abstinenz nach Capellmann und Cohnstein, die auf ein zeitliches Konzeptionsoptimum Rücksicht nimmt. Dieses besteht nach Capellmann vor und nach der Menstruation und nach Cohnstein, wie erwähnt, zu bestimmten, für jede Frau verschiedenen Jahreszeiten, die sich nach dem Monate der früheren Geburten berechnen lassen. Allein das hat sich nicht bestätigt und das Capellmannsche Verfahren ist zum mindesten unzuverlässig.

Die häufige Amenorrhöe und seltene Konzeption während der Laktation ist bekannt. Man empfahl darum, die Männer sollen durch Saugen an der Brust die Laktation wachhalten und so Sterilität künstlich

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orrufen. Allein nach den neuesten Untersuchungen von Heil
nt der Laktation nur etwa in 50% Amenorrhöe zu und tatsächlich
n wir trotz Laktation erneute Konzeption nicht so selten.
n die Bedeutung der Nahrung und des Orgasmus des Weibes
ich schon erinnert.
ie artifiziellen Mittel zerfallen in mechanische, chemische
operative.
ie ersten übergehe ich ganz.
ie chemischen Mittel bestehen in medikamentösen Spülungen.
sollen entweder die Spermatozoen direkt abtöten oder physikalisch
liminieren durch Wegschwemmen.
n operativer Sterilisierung haben wir schon die Zerquetschung
Ovarien kennen gelernt. Ob seiner tollen Ungeheuerlichkeit er-
ne ich den Vorschlag Weinholds, man solle den jungen Mädchen
Genitalien bis zur Heirat durch eine metallische Versiegelung ab-
ießen. Das erinnert an die bei gewissen Völkern übliche Infibu-
n zum Schutze der Keuschheit.
Praktisch diskutabel sind der künstliche Abort, die Castratio
rina durch Verschorfung des Endometriums nach Pincus, die
tration, gelegentlich die Uterusexstirpation unter Belassung
Ovarien. Von der Castratio uterina ist nicht viel zu halten; sie
unzuverlässig und gefährlich. Für die anderen Methoden bedarf es
anderer Indikationen, die sich nach den Besonderheiten des Einzel-
s richten.
Das eigentliche Verfahren ist die Durchtrennung der
ben, die Tubensterilisation. Schon seit längerer Zeit bekannt,
Kehrer sie besonders empfohlen. Ihre Indikationsstellung
lt die größten Anforderungen an das ärztliche Verantwortlichkeits-
ußtsein. Man darf nicht vergessen: der Eingriff ist doch nicht
z ungefährlich; die Konzeptionsfähigkeit ist dauernd zerstört, die
handenen Kinder können sterben und die Reue kommt zu spät.
nhafte Gynäkologen, wie Koßmann, lehnen sie daher ganz ab.
hrer selbst ließ sie nur zu nach gründlicher Belehrung der Ehe-
ten, nach Beratung mit dem Hausarzte, nach schriftlicher Festlegung
Situation, wenn Schwangerschaft oder Geburt der Gesundheit der
u Schaden bringen.
Es gibt auch Operateure, die aus rein sozialen Momenten die
bensterilisation ausführen und die Genesung garantieren. Jedoch
Möglichkeit, sich auch anders zu helfen, die eventuelle Gefahr
r Operation, um deretwillen sie zum Teil nicht gewünscht, zum
il vom Arzt abgelehnt wird, machen die Tubensterilisation aus
ründen sozialer Natur zu einer seltenen Operation. Garantien aber,

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wie die genannten, halte ich für falsch und unerlaubt. Man kann in der Medizin nichts garantieren.

Sie sind geeignet, ein leichtsinniges Verlangen nach der Operation wachzurufen und bringen dann den Arzt nur in Verlegenheit, da er ein solch unbegründetes, leichtsinniges Trachten nach künstlicher Sterilisierung ablehnen muß; denn selbstverständlich muß sich das ärztliche Gewissen gegen den Mißbrauch der Tubensterilisierung kehren. Dies wird freilich nicht allzuoft der Fall sein, da einesteils der Entschluß, sich operieren zu lassen, anderenteils das Bewußtsein der dauernden Unfruchtbarkeit den Wunsch nach der Operation nur selten aufkommen lassen.

Aus diesem Grunde wird es daher kaum dahin kommen, daß der Staat an der Tubensterilisation Interesse hat, ähnlich wie an der absichtlichen Verstümmelung zum Zwecke der Militärdienstuntauglichkeit.

M. H. Wir haben eine Summe Momente kennen gelernt, die Sterilität verursachen, die gleichzeitig zum großen Teil eine Konzeption für Mutter, Kind, Familie und Staat auch gar nicht wünschenswert erscheinen lassen. Angesichts ihrer wird oft die Sterilität als ein Glück zu betrachten sein und sich manchesmal der Wunsch regen, daß die Heirat nicht die einzige Karriere des Mädchens wäre, ohne die sie sonst brotlos ist. Wir haben soeben ein Gebiet verlassen, das an den sittlichen Ernst des Arztes und an seine Kunst, zu individualisieren die größten Anforderungen stellt. Feste Normen gibt es nicht.

Die einzige Richtschnur für sein Handeln trägt der Arzt in seiner eignen Brust. Und da zeigt sich dann so recht das Homersche „ἰητρὸς γὰρ ἀνὴρ πολλῶν ἂν τάξιος ἄλλων“ und das Billrothsche Wort: „nur ein guter Mensch kann ein guter Arzt sein“.

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(Innere Medizin Nr. 147/48.)

Über kardiopulmonale Geräusche.[1])

Von

Prof. Hermann Müller,

Zürich.

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rrando discimus. — Die eigenen Fehler und die Fehler anderer in der Kunst der Diagnostik unsere besten Lehrmeister. Es jetzt 16 Jahre her, daß ich zum erstenmal so recht eindringlich eine Art von Herzgeräuschen aufmerksam wurde, welche schon hlige Male Anlaß zu Fehldiagnosen gegeben hat und tagtäglich gibt.

m Mai 1890 kam in meine Behandlung der 14jährige Jüngling Albert B., Höschi 45 Zürich V. Sohn eines Küfermeisters. Der Jüngling hatte in der Primarschule rn und Scharlach durchgemacht und war in den letzten Jahren sehr viel ad; er war streng gewachsen, hatte häufig Nasenbluten und wurde von verdenen Ärzten behandelt. Im Winter 1889/90 hatte er sehr viel die Schule umt, wollte nie arbeiten. — Bei meinem ersten Besuche am 3. V. 1890 war bettlägerig, klagte über große Müdigkeit, über häufiges Herzklopfen und zeitAtemnot. Die Untersuchung ergab normale Gesichtsfarbe, gute Ernährung, Anämie. Die Herzdämpfung schien etwas vergrößert zu sein, die Grenze war wenig außerhalb von der Mammillarlinie und die rechte ganz außerhalb vom rechten Sternalrand. An der Herzspitze hörte ich ein lautes endes, systolisches Geräusch und der 2. Ton an der Hörstelle der nonalis war sehr deutlich akzentuiert.

Die Untersuchung der übrigen Organe ergab normalen Befund. Vom 3. bis ai besuchte ich den Pat. im ganzen 7mal, untersuchte jedesmal das Herz und ein allerdings nicht immer gleichlautes systolisches Geräusch an der Mitra. Ich erklärte der Mutter, daß ich ein Geräusch am Herzen höre und annehmen e, daß eine Herzklappe nicht vollkommen schließe, und daß der leichte Herzr wahrscheinlich auf den überstandenen Scharlach zurückzuführen sei. — Die

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[1]) Nach einem Vortrage, gehalten an der LXXI. Versammlung des ärztlichen alvereins in Olten 27. Okt. 1906 und in der Gesellschaft der Ärzte von Zürich ebr. 1907.

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Mutter kann heute noch erzählen, daß sie damals selbst das Ohr auf die entblößte Brust gelegt und ein „rauschendes" Geräusch wahrgenommen habe. Am 19. V. wurde Pat. von mir entlassen und ich sah ihn seither nie wieder. – Ich hatte gar keinen Grund an der Richtigkeit meiner Diagnose, „leichte Insuffizienz der Mitralis", zu zweifeln, denn ich hatte mehrere Male sehr sorgfältig untersucht und es schien alles zu stimmen. Da erfuhr ich einige Wochen später, daß der Jüngling von seiner Mutter noch zwei andern Ärzten zugeführt wurde und daß beide keinen Klappenfehler konstatieren konnten; der eine derselben hat mir das im 2. Akte einer ärztlichen Gesellschaft mitgeteilt. Pat. ist seit einigen Jahren in Amerika verheiratet und befindet sich vollkommen wohl. Vom Vater habe ich noch vor kurzer Zeit vernommen, daß anno 1890 vor mir 4 Ärzte seinen Sohn behandelt hatten und daß zwei derselben — sehr bekannte Zürcher Ärzte – ebenfalls einen Herzfehler konstatiert und eine kurze Lebensdauer vorausgesagt hatten.

Der Fall hat mich zum genauern Studium des unerklärlichen Herzgeräusches, das ich so sicher hörte und nicht als ein akzidentelles anämisches Blasen taxieren konnte, und das zwei andere zuverlässige Beobachter nach mir nicht mehr hörten, angeregt. In den folgenden Jahren sind mir nun eine ganze Anzahl ähnlicher Fälle vorgekommen, wo andere Ärzte die gleiche Fehldiagnose machten, die mir passiert war. So habe ich mehrere junge Männer untersucht, die gelegentlich zu mir kamen, weil sie wegen Herzfehler militärfrei wurden, trotzdem sie noch nie irgendwelche Beschwerden hatten. Bei der Untersuchung, die bei der militärischen Ausmusterung in stehender Position vorgenommen worden war, sei ein lautes Geräusch am Herzen gehört worden. Ein anderes Mal stellte sich bei mir ein Herr zur Untersuchung ein, der wegen Herzfehler nicht in die Lebensversicherung aufgenommen wurde. Und einmal konsultierte mich eine Frau, der nach vorhergehender Untersuchung durch zwei Ärzte Herzfehler konstatiert und deshalb der künstliche Abortus eingeleitet worden war. Alle hatten keinen Herzfehler.

Ein besonders klassischer Fall, der mir unvergeßlich bleiben wird, war im Jahre 1897 lange in meiner Beobachtung.

Herr Max M., geb. 17. VII. 1879 Zürich V, ist der einzige Sohn aus neuropathisch stark belasteter Familie. Eine Tante väterlicherseits ist seit vielen Jahren hochgradig hysterisch, ich hatte sie selbst vor Jahren vorübergehend an neurasthenischer Stenokardie behandelt, und eine andere Tante, in deren Familie ich seit langer Zeit Hausarzt bin, war einmal mit angioneurotischem Ödem des Gesichtes behaftet. Der Vater hat schon öfter schwere Attacken von Gelenkneuralgien durchgemacht, der junge Mann selbst litt mehrere Male an sehr schmerzhaften Gelenkaffektionen, die ich unbedingt für neuralgisch erklären mußte. —

Am 20. Mai 1896 wurde ich mittags 12 Uhr zum Pat. gerufen wegen heftiger Schmerzen in der Umgebung des Nabels — da alle andern Erscheinungen fehlten, hielt ich den Anfall für eine Neuralgie. — Von da an sah ich den Pat. nicht mehr bis am 8. Jan. 1897. 8. Jan. vormittags nach einem raschen Gange plötzlich heftige Schmerzen in der linken Schulter, Beengung auf der Brust,

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tigstes Herzklopfen. 11 Uhr 45 mein erster Besuch — kein Fieber, illend starker, erschütternder Herzstoß, Puls 100, kräftige, reine Herztöne. Abends 8 Uhr 2. Besuch. Den ganzen Tag über war das Herzklopfen erholt für die Umgebung und den Pat. à distance hörbar, manchmal i ein „eigentümlich knackendes Geräusch" — „wie wenn der Wind uchte, aufgehängte Wäsche bläst"; daher die Meinung des Pat. „daß an einer klappe etwas sei". Abends 8 Uhr höre ich in der Nähe der Herzspitze etwas r- und oberhalb von derselben ein systolisches, anstreifendes, lecken- Geräusch — wie bei Perikarditis — habe ich in meiner sorgfältig geführten ankengeschichte notiert. Zu gleicher Zeit habe ich mehrere Male auf einige Entfernung ein knackendes Geräusch gehört, „wie wenn 2 Fingernägel einander geknipst werden". Das Geräusch war auch hörbar beim An- en des Atems und änderte sich nicht bei Lagewechsel. Ein solches usch hatte ich bisher noch nie gehört. Auf der Lunge war bei sorgfältigster rsuchung nichts Abnormes zu finden.

9. I. 1897. Heute den Tag über wiederholt das auf Entfernung hörbare Ge- ch von der oben beschriebenen Qualität.

14. I. Noch Klage über große Müdigkeit, Herzklopfen bei der leichtesten Er- ng. Das anstreifende systolische Geräusch, das am 8. abends zu hören habe ich bei den täglichen Untersuchungen nie wieder wahrge- men.

17. I. Abends zwischen 6 und 6½ Uhr wieder plötzlicher Schmerz in der n Schulter, Schmerzen beim Atmen, heftigstes Herzklopfen und während Nachtessens am Tische laut hörbares, knackendes Geräusch.

19. I. Letzte Nächte gut — heute Herzaktion ruhig, Subjektivbefinden gut, e Schmerzen, kein Herzklopfen.

Im Laufe des Februar sah ich den Pat. nur 2mal in meiner Sprechstunde, das e Mal am 17. — jedesmal ganz normaler Herzbefund.

Am 3. März kommt Pat. wieder in meine Sprechstunde; er klagt über Herz- fen und Schmerzen in der Herzgegend, die sich ab und zu noch einstellen. der Untersuchung, die ich diesmal nur in stehender Position vornahm, fand normale Herzdämpfung und vollkommen reine Töne und erklärte dem Pat., ich schon so oft erklärt hatte, daß ich die Krankheit unbedingt für e Neurose halte. —

Am 6. März 1897 ging der Vater mit seinem Sohne in die Sprechstunde zu m andern Arzte A.; derselbe untersuchte, hörte am Herzen ein Geräusch, er- te die Krankheit für eine Herzbeutelentzündung und stellte dem r gegenüber eine ernste Prognose. Zur Sicherstellung seiner von der meinigen tark abweichenden Diagnose schickte der betreffende Kollege den Pat. direkt zu m dritten Arzte (B), derselbe hörte ebenfalls ein Herzgeräusch, das einer Herzbeutelentzündung herrühre, und kritisierte in höchst unkollegialer meine ungenügende Untersuchung. Die beiden Herren Kollegen haben Pat. nur im Stehen untersucht; wer aber in einem schwieriger deut- n Falle bei der Herzuntersuchung die Vorsicht unterläßt, einen Kranken ab- hselnd im Stehen und Liegen zu untersuchen, der beweist, daß er in der Herz- nostik nicht sonderlich bewandert ist, und hat kein Recht, die nach langer fältiger Beobachtung eines andern gemachte Diagnose über den Haufen zu fen, zumal wenn es sich um eine einzige, erste Untersuchung handelt. — Am chen Abend (6. III. 1897) kam der Vater in größter Aufregung zu mir in die Poliklinik, ein Sohn nach dem Befunde zweier Ärzte eine Herzbeutelentzündung habe;

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er brachte mir einen Brief des einen (A) mit, worin mir derselbe schrieb: „Herr K. Vater hat mich heute morgen wegen seines Sohnes Max konsultiert. Der Vater sagt mir, daß Sie die Krankheit für eine Herzneurose erklärt hätten. Leider kann ich mich dieser Diagnose nicht anschließen, ich glaube eher, daß es sich hier um perikardistische Geschichten (ev. Verwachsung des Perikards) handelt und bin der Meinung, daß beim Pat. Bettruhe für längere Zeit geboten ist. . . . Sie verzeihen, daß ich Ihnen bestimmte therapeutische Vorschläge mache, ich fürchte aber, daß bei ambulanter Behandlung des Pat. schlimmere Dinge sich entwickeln könnten — Ich habe dem Vater erklärt, daß ich trotz alledem an meiner Diagnose „Herzneurose" festhalte und habe noch am gleichen Abend den Pat. besucht und wieder untersucht — im Liegen und im Stehen — und habe kein Geräusch mehr gehört — auch nicht beim Vornüberbeugen des Rumpfes, und nicht bei starkem Aufdrücken des Stethoskopes.

Auf Wunsch der Eltern habe ich den jungen Mann von da an wieder täglich besucht, bei jedem Besuche sorgfältig untersucht und ich habe nichts gefunden was für Perikarditis verdächtig gewesen wäre.

7. III. und folgende Tage — Herzaktion ruhig, Herzstoß im 5. I. R. in der Parasternallinie, kräftig, eng umgrenzt, ideal reine Herztöne. Puls 56—60.

Am 12. März hat auf meinen ausdrücklichen Wunsch ein vierter Arzt (O. C.), ausgezeichneter Kenner der physikalischen Untersuchungsmethoden, den Pat. genau objektiv, ohne von dem Vorhergegangenen nur eine Silbe gehört zu haben, untersucht und in einem ausführlichen schriftlichen Befunde konstatiert, „daß das Herz bei Herrn Max M. absolut normal sei". Diesen Befund und meine ausführliche Krankengeschichte habe ich dem Kollegen A sofort zur Einsicht zugeschickt. Darauf schrieb er mir am 13. III. 1897. „Auch nach dem Berichte von C kann ich unmöglich den Fall als Herzneurose auffassen; ich halte vielmehr trotzdem an einer Affektion des Perikards fest." Natürlich habe ich es jetzt aufgegeben, noch weitere Belehrungsversuche zu machen.

Am 18. III. habe ich notiert: Heute ist sehr schön die pendelartige Bewegung des Herzens nach rechts zu beobachten — auch das spricht gegen Synechien des Herzbeutels. — Ich habe den Pat. auch röntgenisieren lassen — damals noch von Prof. Pernet im physikalischen Institut des Polytechnikums — habe absolut nichts für Perikarditis Verdächtiges gefunden.

Am 18. und 19. III. ließ ich den Pat. einen kurzen Spaziergang machen — 19. III. nach dem Spaziergange 10 Uhr 30 vormittags wieder stenokardischer Anfall — Müdigkeit in der linken Schulter, Schmerzen in der Herzgegend und unter dem linken Schulterblatte, ausstrahlend in den ganzen linken Arm, laute Stöhnen; Angstgefühle, dabei sehr starkes Herzklopfen und auf Entfernung hörbares, eigentümliches, schwer zu beschreibendes, mit dem Puls zusammenfallendes Geräusch. Bei meiner Ankunft 12 Uhr 5 Anfall vorüber — ruhige Herzaktion, reine Töne.

Am 10. IV. aus der Behandlung entlassen.

13. V. 1897 rasch vorübergehender Anfall von Schmerzen im Abdomen.

Ende Dezember 1897 akquirierte Pat. in einem neuen stenokardischen Anfall ein präcordiales Emphysem (von Dr. S. meine Diagnose bestätigt) und wurde davon vollständig geheilt (siehe Dissertation von Hermann Suter, stud. pract. von Zürich).

Mitte Oktober 1898 waren die Eltern mit ihrem Sohne auf der Rückreise von Berlin bei Erb in Heidelberg; derselbe diagnostizierte „Herzneurose".

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)ezember 1898 — noch einmal kurzdauernder Schmerzanfall auf der Brust
öne kräftig, rein. Febr. 1900 — heftige Supraorbitalneuralgie. (Influenza.)
901 nur 2malige Untersuchung, normaler Herzbefund.
. VIII. 1902 kommt von Berlin und reist mit den Eltern nach Tarasp.
. Jan. 1903 heftige Gesichtsneuralgie.
0. Jan. Untersuchung des Herzens — normaler Befund, nur Puls sehr labil,
hat schon in Berlin Schwankungen von 45 bis 130 selbst konstatiert, sehr
:r Abusus tabacci — Zigaretten.
3. 1. den ganzen Tag Schmerzen in der Herzgegend, Puls schwankt zwischen
d 70, selten ein Schlag aussetzend.
5. I. 1903 Befinden ganz gut, Puls beim Liegen 40, beim Stehen 48.
8. I. verreist wieder nach Berlin.
m Septemb. 1906 habe ich den Pat. das letzte Mal gesehen und untersucht.
Herz verhält sich ganz ruhig und ist ganz normal. Pat. ist zur Zeit als Dr.
n Paris tätig.

:h habe mich etwas lange bei dieser interessanten Krankengeschichte
ehalten, weil ich aus meiner langjährigen Tätigkeit keinen Fall
ıe, der so drastisch illustriert, wie vorsichtig man in der Beurtei-
von Herzgeräuschen sein muß. Keiner der zahlreichen seither
mir beobachteten Fälle zeigt so prägnant, welche große praktische
:utung die Unterscheidung der sog. akzidentellen Geräusche von
organischen, auf anatomischer Veränderung des Zirkulationsappa-
ı beruhenden, hat. — Jeder hat sein Steckenpferd; das meinige
von jeher die physikalische Diagnostik gewesen, ein Erbe meines
hrten, ersten Chefs Biermers, des bekannten Meisters physika-
ıer Diagnostik. Als Assistent von Biermer (1872—74) und nach-
als Sekundärarzt von Huguenin (1874—79) habe ich während
Jahren viele Kurse über physikalische Untersuchungsmethoden
ilt für Anfänger und für Vorgerücktere, und als Direktor der Poli-
ik habe ich mich von jeher mit Vorliebe mit der Pathologie des
islaufs beschäftigt; seit vielen Jahren halte ich auch in der Poli-
ik Übungen in der physikalischen Diagnostik und so bin ich all-
ılich in den Ruf gekommen, Spezialist für Herzkrankheiten zu sein
infolgedessen sind mir schon seit Jahren mehr und mehr solche
e zugekommen, wo scheinbar unerklärliche Herzgeräusche gefunden
den konnten oder gefunden worden waren. Ich habe lange nach
:r Erklärung gesucht, aber keine gefunden.

Da bin ich endlich aufmerksam geworden auf das klassische Werk
berühmten französischen Klinikers Potain. Es war im Spätherbst
Jahres 1899, als ein Volontärarzt in der Poliklinik ein 15jähriges,
h nicht menstruiertes Mädchen, das allerlei zu klagen hatte, Atem-
, Herzklopfen usw. untersuchte und an der Hörstelle der Pulmo-
is ein systolisches und diastolisches blasendes Geräusch
örte hatte, das in ihm den Verdacht auf Insuffizienz der Pulmonal-

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klappen aufkommen ließ. Er übergab mir die Patientin zu näherer Untersuchung, und da kam ich dazu, einen Herzklappenfehler, speziell die von vornherein so ganz unwahrscheinliche Schlußunfähigkeit der Pulmonalklappen mit Sicherheit auszuschließen. Bei der fast eine ganze Stunde dauernden Untersuchung fiel mir ein ganz ungewöhnlicher Wechsel der Geräusche auf — ein fortwährendes Kommen und Verschwinden und eine merkwürdige, zum Teil mit der Atmung zusammenhängende Veränderung der Geräusche, besonders nachdem das Mädchen längere Zeit auf dem Untersuchungsbette ausgeruht hatte. Ich hatte den jungen Kollegen auf diesen auffallenden Wechsel und auf das vollständige Fehlen von den Folgeerscheinungen eines Klappenfehlers aufmerksam gemacht und habe dann erfahren, daß die bei dem jungen Mädchen gehörten Geräusche offenbar die von Potain beschriebenen kardiopulmonalen [k. p.] Geräusche sein müssen, welche nicht im Herzen, sondern in der Lunge entstehen. Von dem Inhalte des Potainschen Werkes habe ich bis zum Winter 1902/03 allerdings nichts anderes gewußt, als daß Potain die bisher für unerklärlich geltenden Herzgeräusche (und überhaupt alle sog. anorganischen Geräusche) für kardiopulmonale erklärt. Ich habe seit dem Jahre 18 emsige Herzuntersuchungen gemacht und habe geglaubt, manche Entdeckung als der erste gemacht zu haben und fand dann bei genauer Einsicht in das Potainsche Werk die meisten meiner Entdeckungen bereits gemacht. Die leihweise Überlassung des Werkes, das auf der hiesigen Bibliothek nicht zu haben war und im Buchhandel vergriffen ist, verdanke ich dem Kollegen Minnich von Zürich. Zu Anfang des Jahres 1903 habe ich alle meine bis dahin gesammelten Fälle einem Doktoranden, Otto Brunner von Laupersdorf, zur Bearbeitung übergeben. Seine erste und Hauptaufgabe war die Übersetzung von Potain. Im Sommer 1903 ist die Doktordissertation erschienen und am 14. klinischen Ärztetag in Zürich (14. VII. 1903) habe ich einen Vortrag über meine damaligen Erfahrungen gehalten.

Ich hatte an jenem Tage gerade die günstige Gelegenheit 1. einen Privatpatienten, einen 20jährigen russischen Studenten vorzustellen, der im vorhergehenden Jahre von 4 russischen Militärärzten untersucht und wegen Klappenfehler militärfrei geworden war und bei dem man im Stehen ein lautes, blasendes, systolisches Geräusch an der Herzspitze hören konnte, das unmittelbar nach dem Abliegen spurlos verschwand und 2. einen poliklinischen Patienten, einen 20jährigen Weber vom Lande, der angeblich einen Herzfehler hatte und bei dem im Liegen ein lautes, systolisches Geräusch an der Mitralis zu hören war, das beim Stehen ganz verschwand. (Jeder der Untersucher konnte sich überzeugen von dem ganz prompten Erscheinen und Verschwinden des Geräusches bei Lagewechsel.)

Das Werk Potains, ein Muster aufmerksamer Forschung, ist die

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cht einer 14jährigen sorgfältigen klinischen Beobachtung, die er nbar gemeinsam mit seinem Chef de clinique H. Vaquez gemacht

Bei einer Gesamtzahl von 4300 Patienten hat Potain 380 Fälle anorganischen Geräuschen beobachtet; bei 44 Fällen wurde durch Sektion festgestellt, daß keine anatomische Veränderung der Entung der Geräusche zugrunde lag. Bei Männern wurden die anischen Geräusche in 6—9%, bei Frauen in 11,55% beobachtet. ain behandelt in eingehender Weise die verschiedenen Theorien, che bisher (1894) zur Erklärung der anorganischen Geräusche aufellt wurden, er widerlegt dieselben an Hand exakter klinischer und erimenteller Untersuchungen und kommt zu dem Resultate: Die rganischen Geräusche können weder durch Hydrämie, ämie, noch auch durch funktionelle Insuffizienz, nicht ch nervöse Störungen, veränderten Blutdruck oder durch änderte Strömungsgeschwindigkeit entstehen, sie können ht im Herzen entstehen, sie müssen außerhalb des Herzens en Ursprung nehmen, und da sie mit den perikarditischen tain) und pleuritischen Geräuschen nichts gemeinsam en, müssen sie in die Lungen verlegt werden.

Ich muß es mir leider versagen, auf die interessanten Auseinanderungen und die nähere Begründung Potains einzugehen, und verhte auch darauf, alle die Theorien zu besprechen, die seit dem re 1894 von deutschen Autoren über die sog. funktionellen, akzitellen, anorganischen, nervösen Geräusche aufgestellt worden sind. meisten derselben sind am grünen Tische ersonnen worden, nche erscheinen im Dämmerlichte von sehr schwachfüßigen Notpothesen — alle hätten das Licht der Welt gar nicht erblickt, wenn Lehre Potains bekannt gewesen wäre. Der Deutung besondere wierigkeiten machten von jeher die anorganischen Geräusche an Hörstelle der Pulmonalis, wo nach dem übereinstimmenden Urteil er Beobachter ein Lieblingssitz der akzidentellen Geräusche ist. le Versuche, die Geräusche an der Pulmonalis [resp. Hörstelle der lmonalis] zu erklären, sind vollständig gescheitert und nicht mit recht hat seinerzeit v. Ziemßen die Pulmonalgeräusche als ablut unerklärbar bezeichnet.

In der deutschen medizinischen Literatur — ich glaube, daß mir ine wichtigere Arbeit entgangen ist — finden wir fast nichts über e bedeutungsvolle Arbeit Potains, nur ganz selten und beiläufig rd, wenn von den akzidentellen Geräuschen die Rede ist, Potain iert, so finde ich z. B. im neuesten Lehrbuche über die Krankheiten s Herzens und der Blutgefäße von Romberg an einer Stelle (S. 56), o von den Herzlungengeräuschen die Rede ist, die beiläufige

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Bemerkung: „Potain geht aber entschieden zu weit, wenn er alle akzidentellen Geräusche als Herzlungengeräusche deutet.“ Dagegen ist unsern welschen Kollegen die Potainsche Lehre sehr wohlbekannt; ich habe noch vor kurzem erfahren, daß die Professoren von Lausanne, de Cérenville und sein Nachfolger Bourget, in Vorlesung und Klinik auf die Souffles cardiopulmonaires ausführlich zu sprechen kamen.

Am Schlusse seiner Arbeit stellt Potain folgende Thesen auf:

1. Es entstehen in der Lunge unter dem Einflusse der Herzbewegung gewisse lokalisierte Atmungsgeräusche, welche den Charakter und Rhythmus von Herzgeräuschen annehmen.

2. Der Mechanismus dieser Geräusche konnte in exakter Weise festgestellt werden.

3. Diese Geräusche sind richtige blasende Geräusche (souffles); sie haben eine große Ähnlichkeit mit den Geräuschen bei Klappenfehlern.

4. Dennoch kann man sie fast immer mit Sicherheit von denselben unterscheiden vermöge ganz bestimmter Merkmale.

5. Diese Merkmale sind zweierlei Art; die einen beziehen sich auf die Eigenschaften der verschiedenen Abarten der Blasegeräusche, die andern auf das gleichzeitige Vorhandensein oder Fehlen einer organischen Herzaffektion.

6. Von allen Eigenschaften, welche diese Geräusche haben, sind die wichtigsten und die zur Sicherung der Diagnose am meisten geeigneten ihr Sitz und ihr Rhythmus. Gestützt darauf, kann man die kardiopulmonalen Geräusche mit beinahe vollständiger Sicherheit erkennen.

7. Beinahe sämtliche der bisher sogenannten anorganischen Geräusche sind Kardiopulmonalgeräusche.

Nach meiner langjährigen Erfahrung schließe ich mich in den Hauptpunkten der Potainschen Lehre an, besonders aber über den Mechanismus der Entstehung der k. p. Geräusche habe ich, wie wir später genauer ausführen werden, eine ganz andere Auffassung.

Ich habe in den letzten 7 Jahren in meiner Privatpraxis 184 Fälle beobachtet (130 in der Sprechstunde und 54 in der Hauspraxis), bei welchen k. p. Geräusche gehört werden konnten. Dazu kommen noch etwa 50 ausgewählte Fälle aus der Poliklinik. Die letztere Zahl entspricht natürlich bei weitem nicht der wirklichen Häufigkeit des Vorkommens; diese ausgewählten Fälle sind nur solche, wo Herzfehler

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iagnostiziert worden waren und die mir von den Assistenzärzten entreder zur poliklinischen Demonstration oder zur genauen Diagnosetellung zugewiesen wurden. Unter den Fällen meiner Privatpraxis efinden sich nicht weniger als 31 (28 Schweizer, 2 Deutsche, 1 Russe), ie wegen Herzfehler militärfrei geworden sind. Dazu kommen noch enigstens ein halbes Dutzend poliklinischer Patienten, die ebenfalls egen Herzfehler militärfrei wurden.[1]) Bei ca. 70 weiteren Patienten einer Privatpraxis wurde von andern Ärzten die Diagnose „Herzehler" gestellt. — Ich habe mehrere Kandidaten der Medizin oder nge Ärzte untersucht, die an sich selbst einen Herzfehler diagnostiert hatten, und mehrfach habe ich Angehörige von Ärzten — meist nge Leute — zur Untersuchung bekommen, bei denen Herzfehler ngenommen worden war. Unter den obenerwähnten 70 Fällen beinden sich vereinzelt solche, welche nicht in eine Lebensversicherungsesellschaft oder nicht in eine Krankenkasse oder nicht in den Eisenahndienst aufgenommen worden waren, oder solche, die zu irgendinem andern Zweck ein Gesundheitszeugnis nötig hatten, wurden mit er Diagnose „Herzfehler" vom untersuchenden Arzte überrascht. Einmal hatte ich auch für die kantonale Frauenklinik einen Fall zu egutachten, der wegen Herzfehler zur Einleitung des Abortus empohlen worden war. Einmal bekam ich auch einen Patienten zu untersuchen, der angeblich in Nauheim glücklich von seinem Herzfehler kuriert wurde. (Ich will hier beifügen, daß nach meiner festen Überzeugung die sog. Heilungen von Klappenfehlern, über die verschiedene ernste Beobachter berichten, auf falsche Diagnosen zurückzuführen sind.)

Ich bin natürlich weit davon entfernt, genaue ziffernmäßige Angaben über die prozentuarische Häufigkeit der k. p. Geräusche und über die Details der klinischen Untersuchung machen zu wollen, wie sie Potain in vieljähriger Beobachtung auf einer stationären Klinik (und in großer konsultativer Praxis) gesammelt hat. Dazu eignet sich das hauptsächlich ambulante Material, das mir zur Verfügung stand, durchaus nicht. Mir ist es heute besonders darum zu tun, auf die praktisch hochwichtige Bedeutung der k. p. Geräusche aufmerksam zu machen und die Fehlerquellen aufzudecken, welche so ungemein häufig zu der falschen Diagnose „Herzfehler" führen. Die zahlreichen Fälle von anorganischen Geräuschen

1) Im Laufe des letzten Jahres (Okt. 1906—Okt. 1907) habe ich 8 weitere Fälle (6 Schweizer und 2 Deutsche) untersucht, welche wegen Herzfehler militärfrei wurden und ein vollständig gesundes Herz hatten. — Ich wage es nicht, zu beurteilen, welche Summe von Kraft durch die Fehldiagnose Herzfehler der Armee verloren geht.

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bei Anämischen und Chlorotischen lasse ich deshalb fast ganz unberücksichtigt — sie sind auch in meiner Statistik gar nicht mit aufgezählt und finden nur wegen besonders wichtiger Erscheinungen gelegentliche Erwähnung. Unter meinen Fällen sind solche, wo hervorragende Diagnostiker die Fehldiagnose „Herzfehler" gemacht haben, und wenn mir dieser Irrtum früher selbst öfter als seit Jahren passiert ist, befand ich mich in guter Gesellschaft. Ich bin auch überzeugt, daß manch einer, wenn er diese Zeilen liest, sich an solche Fälle erinnert, wo kürzere oder längere Zeit vorher er selbst oder ein anderer Arzt einen Herzfehler gefunden hat, den er bei späteren Untersuchungen nicht mehr finden konnte. Die individuelle Reaktion auf solche Erfahrungen ist eine recht verschiedene. Der eine geht gedankenlos an ihnen vorbei, der andere, der vielleicht etwas mehr Temperament, dafür aber weniger Sinn für Kollegialität hat, beschuldigt den Kollegen der Unfähigkeit und sagt etwa, wie es mir einmal passiert ist: „Wenn er ihr Leiden für ein nervöses erklärt, dann hat er sie nicht recht untersucht."

Historisches über die kardiopulmonalen Geräusche. Die Angabe, daß es k. p. Geräusche gäbe, äußert schon Laennec, und es ist recht interessant, wie der geniale Entdecker der Auskultation, der die neue Untersuchungmethode schon zu einer staunenswerten Vollkommenheit gebracht hatte, sich ausdrückt: „Es gibt zwei Umstände, bei welchen ein unerfahrener Beobachter an das Vorhandensein eines blasenden Geräusches (bruit de souffle) glauben könnte, ohne daß es wirklich vorhanden ist. Bei einigen Individuen überragen die Pleuren und die vorderen Lungenränder das Herz und bedecken es beinahe. Wenn man ein solches Individuum im Augenblick leicht aufgeregter Herztätigkeit untersucht, so verändert die Diastole des Herzens, indem das Herz die Lungenränder komprimiert und so die Luft daraus auspreßt, das Atmungsgeräusch in der Art, daß es mehr oder weniger dem eines Blasebalges (soufflet) oder eines weichen Holzraspels (râpe à bois douce) gleicht. Aber mit etwas Übung kann man dieses Geräusch sehr leicht von einem wirklichen Herzgeräusche unterscheiden; es ist oberflächlicher und man hört darunter den natürlichen Herzton, und wenn der Kranke einige Momente den Atem anhält, so wird es schwächer oder verschwindet ganz."

Es leuchtet ein, daß Laennec das Vorkommen eines solchen Geräusches als selten ansah. Laennec hat auch nie daran gedacht, aus dem Vorkommen dieser Geräusche einen Schluß zu ziehen in bezug auf die Entstehung der anorganischen Geräusche; denn er hielt an seiner falschen Lehre von der Entstehung derselben fest. Nach

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Laennec haben noch viele andere Beobachter ähnliche Geräusche beschrieben.

Wintrich (1854) berichtet über auskultatorische Geräusche in den Lungen, welche ohne Einfluß der In- und Exspiration hervorgerufen werden, und sagt (S. 171) wörtlich folgendes: „In der Nähe des Herzens habe ich schon ziemlich oft während der Kontraktion desselben ein sanftes, schlürfendes Atmungsgeräusch gehört wie das inspiratorische. Es ist dies auch nichts anderes als eine Luftinspiration, durch die systolische Verkleinerung des Herzens bedingt.“ — Danach ist also Wintrich der erste, welcher diese Geräusche auf eine Saugwirkung des Herzens zurückführte.

Im Jahre 1865 hat Bamberger, welchem zuerst die seltene Gelegenheit geboten wurde, die Herztätigkeit an einem 30jährigen Manne zu studieren, bei welchem eine frische Stichwunde die unmittelbare Palpation der Herzspitze gestattete, gemeinsam mit Kölliker Experimente an Kaninchen angestellt, um die Herzbewegung zu beobachten, und bei dieser Gelegenheit haben die beiden[1]) eine die beiden Herzmomente begleitende zuckende Hin- und Herbewegung des vordern linken Lungenrandes beobachtet. „Die Größe dieser Exkursion (Bamberger) mag etwa eine Linie (d. h. 3 mm) oder etwas darüber betragen, sie ist aber außerordentlich frappant und in die Augen fallend und macht etwa den Eindruck, als wäre der Lungenrand an das Herz unverrückbar befestigt und müßte jeder Bewegung desselben folgen. Ich habe öfter unter verschiedenen Umständen am linken Lungenrande in der Nähe des Herzens ein feines, dem pneumonischen ganz ähnliches Knistern gehört, das genau mit jeder Herzsystole zusammenfiel. Ich zweifle nun nicht, daß diese Erscheinung durch die oben beschriebene systolische Bewegung des Lungenrandes bedingt wird, sobald sich in den Lungenzellen etwas Flüssigkeit befindet.“

In einem im Jahre 1863 publizierten Artikel „Über unerklärliche Herzgeräusche“ tut Skoda Erwähnung eines Geräusches, welches

1) Unabhängig von Bamberger und Kölliker hat Dr. Friedrich Ernst von Zürich die gleiche Beobachtung gemacht. Im gleichen Bande des Virchowschen Archives (9. Bd. 1856 Nr. 10) in „Studien über die Herztätigkeit mit besonderer Berücksichtigung der an Herrn A. Grouxs Fissura sterni congenita gemachten Beobachtungen“ berichtet Ernst (S. 283. 284) beiläufig: „Es läßt sich dieses Verhalten der Lungenränder zu den Herzkontraktionen sehr anschaulich am Kaninchen beobachten. Wenn man in der Herzgegend die allgemeinen Decken, die M. pectorales, die M. intercostales bis auf die feine, dünne Pleura sorgfältig abpräpariert, ohne jedoch die Pleura selbst zu verletzen, so sieht man die das Herz teilweise bedeckenden Lungenränder mit jeder Systole sich mehr verschieben, mit jeder Diastole etwas zurücktreten.“

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dadurch entsteht, daß die systolische Bewegung des Herzens in intensiverem Grade allenfalls in der angrenzenden Lunge ein gewisses Quantum Luft verdrängt und diese Luft beim raschen Ausweichen ein Rauschen erzeugt, welches natürlich mit der Systole des Herzens zusammenfällt. Man könnte hierbei noch weiter gehen und auch die Entstehung eines diastolischen Geräusches für möglich erklären, dadurch nämlich, daß die Luft während der Diastole des Herzens mit einem größern Impetus in die angrenzenden Partien der Lungen einströmt.

Gerhardt in seinem Lehrbuche der Perkussion und Auskultation (Jena 1866, Tübingen 1884) berichtet über eine sehr häufige Erscheinung bei der Auskultation, die am besten als systolisches Vesikuläratmen bezeichnet wird. — „Die Lungenränder folgen der Formveränderung des Herzens, nehmen bei der Systole mehr Luft auf, um den Raum, der bei der Verkleinerung des Herzens frei wird, auszufüllen und geben so an den Grenzen des Herzens Veranlassung zu einer eigenen Form des vesikulären Atmens. Selten hört man wirklich während jeder Systole ein kurzes Geräusch von der Beschaffenheit des vesikulären Atmens, häufig während der Inspiration eine mit jeder Systole statthabende Verstärkung des Inspirationsgeräusches."

Nach Paul Niemeyer (1870) findet sich das Herz-Lungengeräusch (pulsatile respiration, Thorburn — pulsatile pulmonic crepitation, Richardson — systolisches und diastolisches Lungengeräusch, Friedreich — systolisches Vesikuläratmen, Gerhardt) am häufigsten an der linken Seite der vordern Brustwand, es entsteht nur während der In- und Exspiration und verschwindet bei angehaltenem Atem.

In den neuen Lehrbüchern über physikalische Diagnostik (Eichhorst, 4. Auflage 1896, und Sahli, 4. Auflage 1905) ist im Kapitel über die Auskultation der Lungen nur beiläufig als ein Geräusch, das in den Lungen entsteht, das systolische Vesikuläratmen erwähnt. So sagt Sahli (S. 218): „Das systolische Vesikuläratmen kommt unter noch nicht näher bekannten Bedingungen hier und da bei Kranken wie bei ganz Gesunden vor und hat keine pathologische Bedeutung. Man hört es nur in der Nähe des Herzens. In wenig ausgesprochenen Fällen äußert es sich bloß in einer systolischen Verstärkung des gewöhnlichen Vesikuläratmens, während es in anderen Fällen auch bei der Sistierung der Atmung hörbar ist. Seine Entstehung ist jedenfalls abhängig von der mit der systolischen Volumverkleinerung des Herzens (Meiokardie) verbundenen negativen Druckschwankung im Innern des Thorax. Ich glaube, daß einzelne vermeintliche akzidentelle Herzgeräusche nichts anderes sind als solches systolisches Vesikuläratmen."

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Etwas eingehender hat schon im Jahre 1899 O. Rosenbach (das Potainsche Werk war auch ihm nicht bekannt) die Herzlungengeräusche studiert. Auch er akzeptiert die von Wintrich und Bamberger zuerst gegebene Erklärung, daß diese Geräusche den durch die Volumschwankungen des Herzens bedingten Luftströmungen ihre Entstehung verdanken.

Daß gelegentlich kardiales Rasseln durch direkte mechanische Erschütterung, hervorgebracht durch die Herzbewegung, erzeugt werden kann, wenn die infiltrierte oder mit Kavernen durchsetzte Lunge in der Nähe des Herzens mit der Pleura verwachsen ist, wollen wir nicht zu erwähnen vergessen. Diese Art der kardiopneumatischen Geräusche ist aber leicht zu erkennen und hat für unsere Betrachtung keine Bedeutung.

Bevor ich nun auf den Mechanismus dieser k. p. Geräusche eingehe, wollen wir in Kürze die Beweise von Potain, daß sie wirklich im Lungengewebe entstehen, anführen:

1. Die Geräusche haben immer ihr Maximum an den Randzonen der Lunge — im Bereich der kleinen oder absoluten Herzdämpfung werden sie niemals gehört. Alle Theorien, die bisher zu ihrer Erklärung aufgestellt wurden, können widerlegt werden.

2. Man kann aber auch direkt durch das Experiment beweisen, daß die anorganischen Geräusche ihren Sitz und ihre Entstehung in den Lungen haben. Man hört diese Geräusche nicht selten bei Hunden und bei Pferden. Bei dem 4. Teil aller Hunde in der Tierarzneischule in Paris fanden sich diese Geräusche vor und die öfter gemachte nachherige Sektion ergab vollkommen normales Herz und normale Klappen. Potain hörte eines Tages bei einem Pferde oberhalb des Spitzenstoßes ein lautes Geräusch, das er nach seinem Charakter für ein kardiopulmonales hielt. Die sofort vorgenommene Sektion ergab vollständig gesundes Herz und normale Klappen. An der Stelle des Geräusches war ein zungenförmiger Lappen von Lungengewebe zwischen Herz und Thoraxwand eingeschoben. — Noch überzeugender war folgender Versuch: Bei einem Hunde sollte zu anderm Zwecke ein Experiment vorgenommen werden. Bei der Auskultation des Herzens wurde ein ausgesprochenes, anorganisches Geräusch gehört. Dr. Franck, ein Freund Potains, führte nun an der betreffenden Stelle der Brustwand einen stumpfen Haken in die Pleura ein, ohne daß Luft in die Pleurahöhle eindringen konnte. Mit Hilfe dieses Hakens konnte der Lungenrand an der Stelle, wo das Geräusch zu hören war, leicht zurückgeschoben werden und sofort verschwand das Geräusch. Ließ man dann die Lunge wieder ihren alten Platz einnehmen, so kam wieder das ganz gleiche Geräusch zum

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Vorschein. So konnte man nach Belieben das Geräusch verschwinden und zum Vorschein kommen lassen.

3. Die anorganischen Geräusche verwandeln sich oft in ausgesprochene Respirationsgeräusche und umgekehrt, je nachdem der Einfluß des Herzens oder der Atmung vorherrscht. Ein Geräusch, das so sehr der Exzitation des Herzens und den Atembewegungen unterworfen ist, kann nur in der Lunge entstehen und nichts anderes sein als verändertes Vesikuläratmen.

Einen neuen höchst wertvollen Beweis, daß die akzidentellen Herzgeräusche in der Tat an den vorderen Lungenrändern[1]) entstehen, erblicke ich darin, daß sie experimentell erzeugt werden können. Kollege Minnich von Zürich (und Weißenburg) hat mir mitgeteilt, daß bei Behandlung der Kaninchen mit minimalen Dosen von Adrenalin, wobei, wie er gefunden hat, neben der außerordentlichen Verstärkung der Herzaktion eine bedeutende Verlangsamung des Rhythmus eintritt, häufig (durchaus nicht immer) sehr starke Herzgeräusche zum Vorschein kommen. Diese Geräusche sind am stärksten wahrnehmbar auf der Höhe der Adrenalinwirkung und verschwinden allmählich mit dem Abklingen derselben. Am 16. November 1906 hat mir Kollege Minnich im hiesigen physiologischen Institut das Experiment (dabei wird die Herzgegend bis auf die Pleura costalis vollständig freigelegt) demonstriert und ich habe mich bei einem von drei Versuchen unzweifelhaft überzeugt, daß beim Sinken der Herzaktion von 240 auf 140 Schläge ein sehr deutliches, scharfes systolisches Geräusch zum Vorschein kam, das allmählich wieder ganz verschwand.

Mechanismus der kardiopulmonalen Geräusche. Da die k. p. Geräusche nach Potain nichts anderes sind als modifiziertes Vesikuläratmen, so müssen sie auch auf die gleiche Art entstehen, und, wie Laennec schon gelehrt hat, entsteht das Zellenatmen durch Reibung der in die feinen Luftwege und Alveolen einströmenden Luft. Die k. p. Geräusche sind in der großen Mehrzahl systolisch, sie entstehen durch Aspiration der Luft. „Wenn das Herz bei der Systole sich dreht und von der Thoraxinnenwand zurücktritt, so entsteht dort ein luftleerer Raum, der wie eine Saugpumpe auf das umgebende Lungengewebe einwirkt und auf dasselbe eine Aspiration ausübt. Die Luft stürzt dann mit großer Vehemenz in die Alveolen und erzeugt dadurch Geräusche, die durch den Rhythmus des Herzens mehr Herz- als Atmungsgeräuschen gleichen. Die auffallende Erscheinung, daß die k. p. Geräusche beinahe ausnahmslos durch ihre Beschaffenheit

1) Respektive an den Perikardialblättern.

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ınd ihr Timbre sich von dem normalen Atmungsgeräusch unterscheiden, ıucht Potain in folgender Weise zu erklären:

In 1 Minute erfolgen ungefähr 16—18 Respirationen und im gleichen Zeit·aume ca. 76 Herzschläge. Die Inspiration dauert 1,35 Sekunden, die Systole nur),26 Sekunden, mit andern Worten: die Systole ist von 5mal kürzerer Dauer als lie Inspiration. Bei gewöhnlicher Inspiration beträgt die eingeatmete Luft etwa /2 Liter, d. h. ungefähr 1/7 der gesamten Lungenkapazität. Aller Wahrscheinlich-ıeit nach ist das ganz anders bei der lokalisierten Inspiration, welche durch das Zurückweichen des Herzens von der Brustwand hervorgebracht wird. Durch die liastolische Erweiterung können gewisse Partien der Lunge derart zwischen Herz- ınd Brustwand komprimiert werden, daß die Luft vollständig ausgepreßt wird; ıommt nun die systolische Retraktion des Herzens, so stürzt eine 7mal größere Luftmenge in die Alveolen und da dieses Einströmen in einer 5mal kürzeren Zeit ıtattfindet, so ist die Schnelligkeit der Strömung 35mal größer als bei einer ge-wöhnlichen Inspiration. Es darf uns deshalb nicht wundernehmen, daß die k. p. Geräusche eine höhere Tonart, eine stärkere Intensität haben und daß sie wesentlich rauher erscheinen.

Diese Theorie, so bestechend sie auf den ersten Blick erscheint, ist völlig unhaltbar; sie leidet bei genauerer Prüfung an dem Fehler, daß sie sich auf Prämissen aufbaut, die falsch sind. Wenn man etwas näher darüber nachdenkt, so muß man füglich staunen, mit welcher Bereitwilligkeit die zuerst von Wintrich, Bamberger und Gerhardt aufgestellte Aspirationstheorie des sog. systolischen Vesikuläratmens von allen nachfolgenden Autoren und auch von Potain und Vaquez angenommen worden ist. Ganz unhaltbar und mit den Gesetzen der Physik in Widerspruch stehend, ist besonders auch die von Potain behauptete Annahme, daß in den Randzonen der Lunge während der Diastole des Herzens die Luft vollständig ausgepreßt werde und daß sich auf diese Weise die vom Vesikuläratmen häufig so total verschiedene Qualität der k. p. Geräusche erklären lasse. Ich selbst wurde zum prüfenden Nachdenken zunächst dadurch veranlaßt, daß ich mehrere Male Geräusche am Herzen hörte, welche die größte Ähnlichkeit mit dem zischenden Geräusche des gesprungenen Topfes hatten, so daß ich unwillkürlich an eine ähnliche Entstehung erinnert wurde. Und dann war es mir besonders unverständlich, wie durch Aspiration von Luft durch die systolische Verkleinerung ein Strömungsgeräusch auch während der Exspiration in einer der entweichenden Luft entgegengesetzten Richtung zustande kommen könne. Ebenso konnte ich die gar nicht selten vorkommenden diastolischen funktionellen Geräusche nicht durch eine Luftaspiration erklären. — Ich änderte deshalb meine frühere Ansicht und kam auf die alte, schon von Laennec akzeptierte Erklärung zurück und vertrat im Wintersemester 1903/04 bei poliklinischen Demonstrationen

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die Lehre: „Wenn das Herz während der Austreibungszeit gegen die Brustwand geschlagen wird, so wird die zwischen Herz und Brustwand gelegene Lungenpartie gepreßt, dabei wird Luft entleert und so entsteht das kardiopulmonale Geräusch." Dagegen machte mir ein Schüler, der in Physik besonders gut bewandert war, cand. med. Walter Heß (jetzt Assistenzarzt der ophthalmologischen Klinik) Opposition und belehrte mich in einem längeren schriftlichen Exposé so, daß ich meine Erklärung wesentlich modifizierte. Ich entnehme der ausführlichen physikalischen Auseinandersetzung nur einige Stellen:

„Die Lungen stellen eine Summe kleiner Blasen vor, die durch ein System von Röhren unter sich verbunden sind. Die Lungen besitzen einen hohen Grad von Elastizität; gegen ihre Begrenzungsflächen, Herz und Brustwand, sind sie leicht verschiebbar." — Es folgt eine physikalische Begründung, welche lehrt, daß bei der diastolischen Vergrößerung des Herzens die Lungen gleichmäßig komprimiert und in toto ausweichen müssen. „Es ist unmöglich, daß an den Rändern Luft ausgepreßt wird und daß dabei ein Luftstrom entsteht; ebenso verteilt sich die Luft bei der systolischen Aspiration auf das ganze Röhrensystem und Alveolargebiet, nicht nur auf die Randpartien in der Nähe des Herzens. (Nur bei einer Fixation der Lungen an der Brustwand wäre die Entstehung von lokalisierten Luftströmen denkbar.) Gegen meine Theorie macht W. Heß folgendes geltend:

„Auch in diesem Falle wird bei freibeweglichen Lungen kein Auspressen der direkt getroffenen Lungenpartien erfolgen, sondern der gepreßte Lungenteil wird in toto zurückweichen." ....

„Mit den Herzbewegungen gehen Verschiebungen der Lungenpartien einher — diese können die Quelle von Geräuschen bilden, denn wenn 2 Flächen übereinanderstreichen, muß immer ein Geräusch entstehen, nur ist es unter normalen Verhältnissen so schwach, daß es vom menschlichen Ohr nicht wahrgenommen wird. Es kann aber unter leicht denkbaren Bedingungen so anwachsen, daß es gehört wird. Ist die Länge der Lungenlamelle, welche keilförmig zwischen Herz und Brustwand eingeschoben ist, relativ groß, (was besonders oft an der Lingula der Fall ist) und ist die Herzbewegung genügend energisch, so streicht die Lunge bei der Systole rasch an Herz und Brustwand vorbei, an die es zudem noch angedrückt wird — ein stärkeres Reibegeräusch wird die Folge sein."

Daß ein Teil der k. p. Geräusche lediglich als Reibegeräusche aufzufassen sind, will ich nicht bestreiten, denn die Geräusche ähneln recht oft leichten pleuritischen oder perikarditischen Reibegeräuschen. Ich kann mich aber des Gedankens nicht erwehren, daß bei der Entstehung der k. p. Geräusche noch eine andere Komponente — Schwingungen des ein System von elastischen Blasen darstellenden Lungengewebes, verursacht durch die Erschütterung des anschlagenden Herzens — eine wichtige Rolle spielt.[1])

1) Ich komme immer mehr dazu, die Erschütterung des Lungengewebes, welche demselben bei der Quetschung des zwischen Herz und Brustwand eingeklemmten

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och ist diese spezielle Frage von geringerer Bedeutung, viel wich-
ger ist der Nachweis, daß die Geräusche zweifellos nicht durch
spiration, durch Luftströmung entstehen.[1]) Ich könnte noch
anche Gründe dafür anführen; aber dazu fehlt mir jetzt die Zeit. —
ur noch eins! Daß bei der systolischen Verkleinerung des Herzens
uft aspiriert wird, kann mit Leichtigkeit experimentell nachgewiesen
erden. Die Aspiration findet statt durch Mund oder Nase. Die
ewöhnliche Atmungsgröße beträgt 500 ccm. Die Menge der bei der
stolischen Verkleinerung des Herzens aspirierten Luft ist jedenfalls
ınz bedeutend kleiner und sicher nicht größer als die Differenz des
ährend der Systole aus- und einströmenden Blutes, und da sich
ieses Volumen auf die ganze Lunge verteilen muß, so kann un-
öglich ein hörbares Geräusch durch die Luftströmung ent-
tehen. — Die Erfahrung lehrt, daß selbst bei Atmungszügen, wenn
ieselben oberflächlich sind, Geräusche nicht wahrgenommen werden.

An dieser Stelle mußte ich in Olten (27. X. 1906) meinen Vortrag
brechen, weil die für die wissenschaftlichen Verhandlungen be-
immte Zeit abgelaufen war. — Bevor wir nun heute (23. Febr. 1907,
esellschaft der Ärzte von Zürich) dazu übergehen, die klinischen
igenschaften der k. p. Geräusche, durch die wir dieselben von den
rganischen Geräuschen unterscheiden können, näher zu studieren,
öchte ich Ihnen doch noch über einige bemerkenswerte Fälle, welche
h seither beobachtet habe, referieren. Es sind im ganzen 27 Fälle,
ber welche ich genaue Aufzeichnungen gemacht habe (5 davon sind
rauen mit Morb. Basedowii — bei fast allen andern ist von anderer
eite Herzfehler diagnostiziert worden).

1. Fall. Am 29. X. 1906 — 2 Tage nach meinem in Olten gehaltenen Vortrage
- kam in die mediz. Poliklinik der 20jährige Schreiner Emil Sl. und verlangte ein
eugnis für das österreich. Konsulat. Pat. war vor kurzem wochenlang im Spital

ungenkeiles während der Austreibungszeit des Herzens erteilt wird, als die
auptquelle der k. p. Geräusche anzusehen. Wir werden später noch einmal auf
iesen Punkt zurückkommen.

1) Wir wollen das Geräusch, das durch die Bewegung des Herzens an den
andzonen der Lungen teils durch Reibung teils durch Schwingungen des Lungen-
ewebes erzeugt wird, der Kürze halber als „Gewebegeräusch" bezeichnen. Als ein
olches Gewebegeräusch, hervorgebracht durch die Erschütterungen, welche den
ungen durch das Einströmen von Luft in die feinsten Luftwege erteilt werden,
st auch das inspiratorische vesikuläre Atmungsgeräusch aufzufassen. Die alte
Laennecsche Theorie von der Entstehung des Vesikuläratmens durch Reibung des
inspiratorischen Luftstromes an der Wandung der feinen Bronchen und Infundibula
ist, wie schon P. Niemeyer, Gerhardt u. a. gelehrt haben, nicht korrekt. Auf
gleiche Weise muß auch nach unserer Meinung die Entstehung des systol. Knister-
rasselns erklärt werden und auch in vielen Fällen — wenn nicht immer — das
Geräusch des gesprungenen Topfes.

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in Graz, er bringt von dort die Diagnose — Herzfehler und Neurasthenie — mit. Die Herzuntersuchung ergab: hart außerhalb vom Spitzenstoße, der an normaler Stelle liegt, ein lautes, systolisches Geräusch, das sehr deutlich mit einem Tone anfängt (mesosystolisches Geräusch). — Alle Schüler, aufgefordert genau darauf zu achten, was in der Gegend der Herzspitze zu hören sei, vernehmen deutlich, daß das systolische Geräusch dem Tone nachschleppt. Das Geräusch verschwindet sofort und spurlos beim Aufrichten. — Herzdämpfung normal; keine Anamnese für Klappenfehler.

2. Fall. Frau Marie L., 48 J. Zürich. I. Diagnose: progressive, perniziöse Anämie.

3. XI. 1906. Hämoglobin 18%. Rote Blutkörperchen 870000, weiße 4300.

5. XI. 1906. Gemeinsamer Besuch mit 2 Assistenten und 1 Praktikanten — tiefste Anämie, wachsbleiche Farbe mit einem Stich ins Gelbe; bedeutende Atemnot — positiver Jugularvenenpuls, deutliches systolisches Schwirren zwischen Herzspitze und Sternum, Herzdämpfung nach rechts und nach links verbreitert — lautes systolisches Geräusch an den Hörstellen der Mitralis und Trikuspidalis — das Geräusch beginnt ganz deutlich ein kurzes Weilchen vor dem systolischen Tone — beim Abheben des Ohres vom Stethoskope ist das Geräusch noch auf kurze Entfernung hörbar; sehr schwaches, systolisches, mit dem Tone beginnendes Geräusch an den Hörstellen der Aorta und Pulmonalis; diese Geräusche verschwinden ganz bei leichtem Abheben des Ohres. — Status pessimus — Dionin.

7. XI. 1906. Der gleiche Befund.

10. XI. 1906. Befinden entschieden besser. Atemnot geringer. Geräusche an M. und Tr. wie früher; an P. und A. fast kein Geräusch mehr hörbar.

14. XI. Fortschreitende Besserung — Geräusche nehmen an M. und Tr. rasch ab. — Von da an auffallende Besserung, die Herzdämpfung wurde normal und die Geräusche verschwanden allmählich ganz bei rascher bedeutender Abnahme der Anämie. Ich zweifle nicht daran, daß es sich hier um zwei Arten von Herzgeräuschen handelt — die Geräusche an der Mitralis und Trikuspidalis sind eine Folge von muskulärer Insuffizienz, verursacht durch die fettige Degeneration des Herzmuskels (positiver Venenpuls, fühlbares Schwirren, allseitige Vergrößerung der Herzdämpfung). — Heute befindet sich Pat. ganz gut. (Pat. ist mittlerweile an einem Rezidiv der perniziösen Anämie gestorben. Aug. 1907.)

3. Fall. Herr Max U., geb. 1. VIII. 1887, wird mir vom Vater mit der Diagnose „Mitralisinsuffizienz“ zugeschickt; der Klappenfehler wurde von 2 andern Ärzten und auch bei der Rekrutierung festgestellt. „Dazu kommt noch“ — so schreibt der Vater — „eine exzessive Nervosität und Hypochondrie. Erstere hat er sich bei der Vorbereitung zur Maturität, letztere durch unbefugtes Lesen meiner mediz. Literatur über Herzkrankheiten zugezogen.“

6. XI. 1906. Höhe 186 cm Brustumfang 99—103 cm. Herzstoß im 4. J. R. daumenbreit innerhalb von der Mammillarlinie. Herzdämpfung absolut normal; tadellos reine Töne — im Stehen und Liegen untersucht.

10. XI. 1906. Befinden entschieden besser, seit Pat. weiß, daß er keinen Herzfehler hat, Puls 60. — Heute ist Pat. von seinen neurasthenischen Herzbeschwerden vollständig befreit; meine Untersuchung und Diagnose hat nach Mitteilung des Vaters Wunder gewirkt.

4. Fall. 10jähriger Knabe, Sohn einer Lehrerswitwe. Vor 1 Monat wurde von

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einem hiesigen Arzte ein „Herzfehler“ konstatiert. — Bei meiner Untersuchung am 11. und 21. XII. 1906 normaler Herzbefund, keine Geräusche.

5. Fall. 17jähriger Jüngling. — Angeblich Herzfehler.

15. XI. 1906. Starker Herzchok, sehr schöne pendelartige Herzbewegung, alternierender Herzstoß; rauhes systolisches Geräusch an der Hörstelle der Pulmonalis, verstärkter 2. Pulmonalton. — Geräusch verschwindet ganz beim Aufrichten. — Im 10. Jahre Scharlach.

6. Fall. 18jährige Jungfrau. — Diagnose: Mitralklappeninsuffizienz; leichtes mesosystolisches Geräusch an der Mitralis, wesentlich lauteres und rauhes systol. Geräusch an der Hörstelle der Pulmonalis. — Eine längere genaue Untersuchung ergibt mit Sicherheit, daß die Geräusche k. p. Ursprunges sind.

7. Fall. Hermine K., Ingenieursfrau, wird von einem Prof. der Gynäkologie einer auswärtigen Universität an Prof. Wyder in Zürich gewiesen mit der Empfehlung, den künstlichen Abortus einzuleiten „wegen Herzfehler und Ankylose des linken Kniegelenks“.

Bei der 1. Entbindung April 1904 8wöchentl., sehr hohes Fieber mit Polyarthritis zuletzt im linken Knie, Herzaffektion, (Arthrit. gon.). — Dez. 1905 künstlicher Abortus „wegen des Herzens“. Pat. wird am 18. I. 1907 von Prof. W. zur Herzuntersuchung an mich gewiesen. Ich konstatierte — an der Herzspitze ein systolisches Geräusch, das nach mehreren Minuten Ruhe verschwand und beim Stehen gar nicht zu hören war.

8. Fall. Karl K., Architekt, geb. 13. XI. 1880 wurde seinerzeit in Meilen militärfrei wegen Herzfehler, rekurrierte, da er sich absolut gesund fühlte und gern Militärdienst tun wollte, wurde aber in Zürich bleibend untauglich erklärt wegen Herzfehler — absolut normaler Herzbefund.

9. Fall. (Jan. 1907.) Anna P. geb. 18. 4. 1893 von Dr. N. Mitralisinsuffizienz diagnostiziert. — Früher keine Krankheiten, 1. Menstruation 25. XII. 1906 — sehr deutlich mit Ton beginnendes systolisches Geräusch an der Herzspitze, gleichzeitig mit dem Ton beginnendes rauhes, systolisches Geräusch an der Hörstelle der P. Das Geräusch an der Spitze verschwindet sofort beim Aufrichten und das Geräusch an der Basis ändert Timbre und Intensität.

10. Fall. Frau Martine Pf., 30 J. Zürich III. — Morb. Basedowii 6. II. 1907. War früher immer gesund, Frühjahr 1906 Atemnot, heftiges Herzklopfen, spontane Heilung. Seit Neujahr 1907 angeblich fast plötzlich wieder an schweren Basedowerscheinungen erkrankt. Basedow auf den ersten Blick zu diagnostizieren. — Herz normal groß, Puls 120—130. — Ungewöhnlich lautes, einem leichten Reibegeräusch ähnliches systolisches Geräusch entlang dem ganz linken Lungenrande, entlang dem rechten Sternalrande von der 3. bis zur 6. Rippe — aber auch sehr deutlich im Bereich der ganzen absoluten Herzdämpfung — keine Stelle der Herzoberfläche ist frei von dem Geräusch. —

17. IV. 1907. Das gleiche Geräusch in gleicher Ausdehnung habe ich nachher noch mehrere Male gehört. Das Geräusch fängt in der Herzmitte und in der Gegend der Herzspitze deutlich mit einem Tone an. — 27. IX. Pat. ist seit Anfang Mai vollständig geheilt, mit der Besserung stellte sich ein „kolossaler Appetit“ ein, heute Ernährung sehr gut, vollkommenes Wohlbefinden. — Herzaktion ruhig. An der Hörstelle der Pulmonalis ist beim Liegen und beim Stehen noch ein lautes, rauhes systol. Geräusch hörbar — an allen übrigen Stellen reine Herztöne.

11. Fall. Frau P., geb. 14. X. 1869. Zürich III. — Morb. Basedowii und Ins

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mit Stenose der Mitralis. — Im Alter von 8 Jahren Scharlach, Polyarthritis – 3 Winter im Spital Münsterlingen, schon damals Herzfehler. Verheiratet seit 12 Jahren, 7 Kinder. — Präsystolisches Schwirren über der Herzspitze, exquisites präsystol. Geräusch an der Mitralis — längs dem ganzen rechten Sternalrande vom 2. I. R. bis zur 6. Rippe ein systolisches anstreifendes Geräusch.

12. Fall. Karl Bl. 3. XII. 1896. Chorea — wurde von mir bei Gelegenheit anderer Demonstrationen heute vor 4 Wochen der Gesellschaft der Ärzte von Zürich vorgestellt und da wurde von einem zur Untersuchung aufgeforderten Kollegen konstatiert, daß das laute systolische Geräusch an der Mitralis sehr deutlich dem Tone nachschleppte (daß das Geräusch mesosystolisch war, an der Hörstelle der P. war ebenfalls ein lautes, rauhes systolisches Geräusch wahrnehmbar — die Geräusche nahmen in stehender Position ganz bedeutend an Intensität ab.

13. Fall. Rosa E. geb. 9. 3. 1893 von Albisrieden. (15. II. 1907.) Früher keine Krankheiten, seit 4 Tagen Erythema nodos. Die Herzdämpfung ist scheinbar vergrößert — rechte Grenze 1 cm außerhalb vom rechten Sternalrand, die linke 1 cm nach außen von der Mammillarlinie — außerhalb und innerhalb vom Spitzenstoße ein lautes blasendes Geräusch, das dem Tone nachschleppt, lautes, rauhes systol. Geräusch an der Hörstelle der P.

14. Fall. Herr Richard H., Kaufmann von Bern, geb. 20. 4. 1872, Deutscher; anno 1892 tauglich zu allen Waffengattungen erklärt, wurde bei der Aushebung angeblich gar nicht untersucht, weil er absolut beschwerdefrei war.

Aug. 1892 in Zürich von Dr. B. wegen Lebensversicherung untersucht und wegen „Herzfehler“ nicht aufgenommen.

Am 9. XI. 1892. Einrücken in Ludwigsburg — im Anfang des Dienstes mehrere Male besonders nach Anstrengung Atemnot und wurde von 6 verschiedenen Ärzten zu verschiedenen Tageszeiten in der Revierkrankenstube — immer im Stehen – untersucht — immer Diagnose „Herzfehler“.

Vom 17. XII. 1892 bis Weihnachten im Lazarett in Ludwigsburg — wegen „Herzfehler“ zum Landsturm 2. Aufgebotes ohne Waffen versetzt. Pat. hat nach seiner eigenen Aussage die Beschwerden sehr übertrieben, weil er gern militärfrei werden wollte. Nie Scharlach, nie Gelenkrheumatismus, überhaupt nie eine ernstliche Krankheit. Arhythmie wurde schon in den Knabenjahren konstatiert. Pat. erinnert sich sehr genau, daß in der Schule die Kameraden sich gelegentlich den Puls fühlten und daß bei ihm leichte Unregelmäßigkeit zu finden war.

4. XII. 1906. Seit 3 Monaten hie und da leichte Atemnot, hie und da plötzlich ein tiefer Atemzug. Pat. ist der festen Überzeugung, daß er einen Herzfehler habe, der Gedanke hat ihn seit 1892 nie völlig verlassen. Höhe 182 cm, Körpergewicht 95 kg, stramme Haltung, tadelloses Aussehen; beim Liegen Puls 76, beim Stehen 80, selten 1 Schlag aussetzend. Herzdämpfung normal, Herztöne tadellos rein, Blutdruck 120 mm — nach der tonometr. Untersuchung Puls beim Liegen und beim Stehen ganz regelmäßig. Diagnose: leichte Herzneurose, Arhythmie, sicher kein Klappenfehler. Pat. ist höchst freudig überrascht, daß er keinen Klappenfehler hat.

Diese kleine Auswahl von Fällen, welche ich Ihnen soeben zur Kenntnis gebracht habe, hat Ihnen ohne Zweifel zur Genüge gezeigt, daß die Erkennung der anorganischen bzw. kardiopulmonalen Ge-

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räusche von der allergrößten praktischen Bedeutung ist; die Unterscheidung derselben von den Geräuschen, welche bei organischen Herzaffektionen vorkommen, ist eine der dankbarsten Aufgaben des Arztes. Niemand wird in Abrede stellen, daß die Diagnose der Herzkrankheiten heutzutage wie vor Dezennien fast ausschließlich auf der physikalischen Untersuchung beruht; ebensowenig wird man bezweifeln, daß die richtige Diagnose das einzig maßgebende für die Prognose und die Behandlung der Herzkranken ist. Es ist das unbestrittene Verdienst von Potain, daß er zuerst in klarer und scharfer Weise auf die Merkmale aufmerksam gemacht hat, welche uns ermöglichen, die akzidentellen Geräusche von den organischen zu unterscheiden. Diese Merkmale, mittelst deren wir die k. p. Geräusche erkennen und von den organischen unterscheiden können, sind Eigentümlichkeiten der Klangfarbe und der Tonhöhe, des Sitzes, des Rhythmus und der Veränderlichkeit.

1. Die Klangfarbe, der Timbre der k. p. Geräusche ist außerordentlich verschieden. Die Geräusche sind weich, leise, hauchend, schlürfend, aber manchmal sind sie auch laut, scharf und rauh; sie sind anstreifend, kratzend, schabend, ähneln den perikarditischen Geräuschen und werden auch oft mit solchen verwechselt; ja sie werden gelegentlich auch sehr laut und können ganz selten auch auf Distanz gehört werden. So wurde beispielsweise von Barié ein Geräusch auf mehrere Meter Distanz gehört, ohne daß bei der Sektion die geringste Spur von Veränderung an den Klappen gefunden werden konnte, und ich selbst habe einmal (bei dem eingangs ausführlich beschriebenen 2. Falle) das von Wintrich zuerst beschriebene, auf Entfernung hörbare „systolische Knacken" wahrgenommen. Ausnahmsweise habe ich ein zischendes Geräusch gehört — „wie wenn Dampf aus einer engen Röhre strömt". — Verhältnismäßig selten bekam ich das als systolisches Knisterrasseln von Bamberger zuerst beschriebene und oft von ihm gehörte Geräusch zu hören; immer habe ich das dem Knistern ähnliche Geräusch nur am Lungenrande gehört und zwar nur bei der Systole — niemals bei der Inspiration.

In einem Falle war das systolische Knisterrasseln besonders deutlich hörbar, aber nur genau an der Hörstelle der Aorta; das Geräusch verschwand sofort beim Aufrichten des Patienten; es ist sicher nicht — wie Bamberger gemeint hat — durch Bewegung von Sekret in den feinen Luftwegen zu erklären. Nach meiner Erfahrung nimmt besonders oft das Geräusch an der Hörstelle der Pulmonalis einen Charakter an, der an Knistern erinnert, und ich möchte vermuten, daß Potain und Vaquez die schwer genau zu bestimmende Geräuschart mit der von ihnen öfter beobachteten Eigenschaft „d'un froissement" bezeichnet haben.

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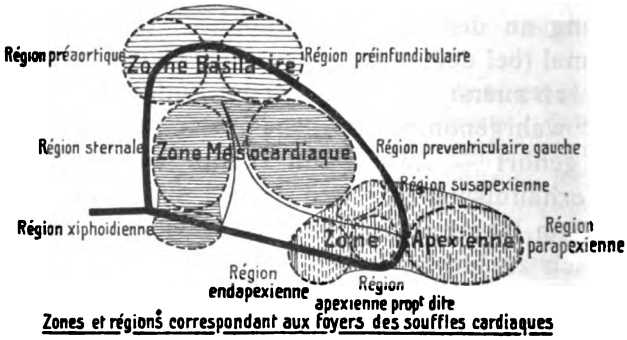
— Nicht so selten haben die Geräusche auch einen musikalischen Charakter — sie sind giemend, tönend, singend — allerdings niemals so laut wie die Stenosengeräusche der Aorta. Diese giemende, tönende Eigenschaft der k. p. Geräusche verdient eine besondere Erwähnung, da gerade diese musikalischen Geräusche vielfach als charakteristisch für Klappenfehler, Abreißung von Sehnenfäden u. dgl. gelten. – Vor allem hat der Timbre in einer großen Zahl der Fälle einen ganz oberflächlichen Charakter, wie schon Laennec ganz richtig bemerkt hat. Diese Eigenschaft ist oft sehr deutlich ausgesprochen und hat für denjenigen, der große Übung und Erfahrung im Auskultieren hat, etwas sehr Charakteristisches. Diese Eigenschaft ist aber weder konstant genug, noch so leicht zu bestimmen, daß man ihr einen sehr großen Wert zugestehen kann. Einer der bezeichnendsten Charaktere ist ein dem Vesikuläratmen identisches Timbre.

Die Tonhöhe der k. p. Geräusche ist fast nie so hoch wie die der Geräusche bei Klappenfehlern.

Die beiden Eigenschaften der Klangfarbe und der Tonhöhe haben nur mäßigen diagnostischen Wert. Ganz anders ist es mit dem Sitz und dem Rhythmus der Geräusche, welche zu den sichersten Kennzeichen der k. p. Geräusche gehören.



Zones et régions correspondant aux foyers des souffles cardiaques

Um den Sitz der Geräusche genau festzustellen, hat Potain die Herzgegend in 3 Zonen eingeteilt:

1. Zone Basilaire — Herzbasiszone,
2. Zone Mésocardiaque — mittlere Herzzone,
2. Zone Apexienne — Zone der Herzspitze.

Jede von diesen drei Zonen hat er wieder in Unterabteilungen eingeteilt.

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Die Herzbasiszone wird in 2 Unterabteilungen eingeteilt: in eine rechtsgelegene (région préaortique), welche dem Ursprunge der Aorta entspricht, und in eine linksgelegene (région préinfundibulaire), welche über dem Conus arteriosus der Pulmonalis und dem Anfangsteile der Pulmonalarterie liegt. An beiden Stellen kann man k. p. Geräusche hören; an der Hörstelle der Aorta sind sie nach Potain und Vaquez äußerst selten, an der Hörstelle der Pulmonalis sehr häufig. Nach meiner persönlichen Erfahrung sind sie an der Hörstelle der Aorta doch etwas häufiger, als die beiden französischen Autoren annehmen; dagegen stimmen alle Untersucher, welche über akzidentelle Herzgeräusche geschrieben haben, darin überein, daß diese Art von Geräuschen sehr häufig an der Hörstelle der Pulmonalis gefunden wird.

In der mittleren Herzzone hat Potain 3 Regionen unterschieden: die Gegend vor dem linken Ventrikel (région préventriculaire gauche), die Sternalregion (région sternale) und die Xiphoidesgegend (région xiphoidienne). In der ersten Region werden sehr häufig k. p. Geräusche gehört, während sie in den beiden anderen Regionen recht selten zu hören sind.

Die Zone der Herzspitze teilt Potain in 4 Abteilungen ein:

1. eine région susapexienne — direkt oberhalb der Herzspitze,
2. eine région apexienne proprement dite — eigentliche Herzspitzengegend,
3. eine région endapexienne — innerhalb von der Herzspitze, und
4. eine région parapexienne — außerhalb von der Herzspitze.

An einer von diesen 4 Regionen (Potain), in der Gegend der eigentlichen Herzspitze, bekommt man fast nie k. p. Geräusche zu hören; das Geräusch der Mitralisinsuffizienz hat immer genau an dieser Stelle seine maximale Intensität. Im Gegensatz dazu sind die Geräusche, welche ihr Punctum maximum innerhalb oder außerhalb von der Herzspitze haben, immer anorganisch. Wenn das Geräusch seinen Sitz in der mittleren Herzzone über dem linken Ventrikel oder in der Spitzenzone innerhalb oder außerhalb von der Herzspitze hat — so lautet die Lehre von Potain — dann ist der Sitz des Geräusches allein schon pathognomonisch. An diesen Stellen wird nach Potain mehr als die Hälfte aller k. p. Geräusche beobachtet. Fast jedes Geräusch (Potain), welches seine maximale Intensität an der Hörstelle der Aorta oder direkt an der Herzspitze hat, ist organisch.

Bei meinen eigenen Untersuchungen, welche — wie ich schon früher mitgeteilt habe — hauptsächlich an ambulanten Kranken

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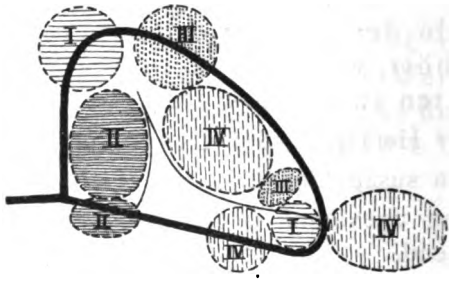
macht worden sind, war es mir nicht möglich, so genau auf den Sitz der k. p. Geräusche zu achten, wie Potain und Vaquez es getan haben. Bei einer größeren Zahl der von mir beobachteten Fälle habe ich die Art der von andern Ärzten wahrgenommenen Herzgeräusche lediglich daran erkannt, daß ich bei meiner Untersuchung überhaupt kein Geräusch gehört habe. Insbesondere habe ich der genauen Lokalisation der Geräusche in der Spitzenregion weniger Aufmerksamkeit geschenkt als Potain. Ich verzichte deshalb ganz darauf, wie Potain über die Häufigkeit des Vorkommens der k. p. Geräusche an den verschiedenen Stellen des Herzens zahlenmäßige Angaben zu machen — meine eigenen Erhebungen sind, trotzdem auch ich tabellarisch darüber Protokoll geführt habe, zu lückenhaft. Nur das möchte ich mit besonderem Nachdrucke erwähnen, daß nach meiner eigenen Erfahrung die Geräusche am allerhäufigsten im 2. I. R. links vom



Sternum, d. h. an der Hörstelle der Pulmonalis, gehört werden und daß demnächst die Herzspitzenregion ein Lieblingssitz der k. p. Geräusche ist. — Zudem ist die Bestimmung des Sitzes der maximalen Intensität der Geräusche nicht immer leicht, denn die Geräusche haben oft mehrfache maximale Stellen; besonders oft werden sie gleichzeitig an der Hörstelle der Pulmonalis und in der Herzspitzenregion gehört, ohne daß von einer Fortleitung von der einen zur anderen Stelle die Rede sein kann.

Endlich gibt es noch eine Gegend — Potain —, wo man niemals anorganische (k. p.) Geräusche hört und wo keine vorkommen können; in der mittleren Herzregion, da wo keine

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unge zwischen Herz- und Brustwand eingelagert ist, im ereich der absoluten oder kleinen Herzdämpfung. Dieser ehre, welche Potain bekanntlich als den ersten Beweis dafür anhrt, daß die k. p. Geräusche tatsächlich im Lungengewebe entstehen, üssen wir auf das nachdrücklichste widersprechen. Ich habe — wie ich glaube — den sicheren Beweis erbracht, daß die otainsche (von Wintrich begründete) Erklärung von der Entehung der k. p. Geräusche eine physikalische Unmöglichkeit st; daß die Geräusche in den Randzonen der Lunge unmöglich durch spiration von Luft entstehen können, daß sie vielmehr teils durch eibung der Perikardialblätter aneinander, teils durch Quetschung zw. Schwingungen des Lungengewebes in den Randzonen der Lungen rzeugt werden. Die Perikardialblätter liegen fortwährend mit spiegelatten Flächen dicht aneinander; während der Herzrevolution, mit der orm- und Lageveränderung, welche damit verbunden ist, findet eine erschiebung des innern Blattes des Herzbeutels dem äußern gegenber statt. Dabei muß ein Geräusch entstehen, das aber meist ür jedes menschliche Ohr unhörbar ist. Wenn ein besonderer Druck stattfindet, wenn die Reibung eine besonders ntensive ist, entsteht ein hörbares Geräusch — und zwar nicht nur an den Randzonen der Lunge, da wo die Lunge zwischen Herz und Brustwand eingekeilt ist, sondern auch — allerdings viel seltener — im Bereiche der absoluten Herzdämpfung. Sehr selten hört man im ganzen Umfange der absoluten Herzdämpfung ein anstreifendes, reibendes Geräusch (z. B. im früher zitierten Fall 10).

Dagegen gar nicht so selten, wie vielfach angenommen wird, hört man diese Geräusche an einer sehr eng umschriebenen Stelle links unten neben dem Sternum beim Ansatz der 5. oder 6. Rippe. Die Geräusche an dieser Stelle sind mir schon aus der Biermerschen Klinik bekannt (1870—1874). Die erste eingehende Schilderung derselben findet sich in der klassischen Arbeit von Johannes Seitz — Zur Lehre von der Überanstrengung des Herzens. 1874 —, der die Geräusche schon ganz richtig durch Anstreifen des Herzens an seine Umgebung erklärt hat. Die Klangfarbe der Geräusche an dieser Stelle ist leicht schabend, leckend, kratzend, anstreifend; sie sind meist systolisch, kurz und dem Tone angehängt. Mehrere Male habe ich aber bisher auch ein diastolisches, kurzes Anstreifen gehört.[1]) Auch Johannes Seitz hat das diastolische Anstreifen schon beschrieben. Einmal habe ich auch an der gleichen Stelle im Bereiche der absoluten Herzdämpfung ein

1) Das diastolische kann sogar stärker und von etwas längerer Dauer als das systolische sein.

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deutlich tönendes, giemendes, systolisches Geräusch gehört (jüngere Frau mit Thrombose der Pulmonalarterien, 1907), ohne daß nachher bei der Sektion etwas am Herzbeutel oder im Innern des Herzens gefunden werden konnte. — Es können also durch einfaches Gleiten der glatten Perikardialblätter übereinander tönende Geräusche erzeugt werden! — Man kann den Charakter dieses akzidentellen perikardialen Geräusches ganz täuschend reproduzieren, wenn man die linke Hand auf das eigene linke Ohr auflegt und mit dem Perkussionsfinger der rechten Hand auf den Rücken der linken Hand eine leicht klopfende und gleichzeitig kurze, schleifende Bewegung ausführt.

Wer immer sich die Mühe nimmt, bei der Auskultation des Herzens besonders bei Herzschwäche oder bei Herzdilationen infolge chronischer Muskelerkrankung auch die eben genannte etwas stiefmütterlich behandelte Stelle noch wenige Augenblicke zu behorchen, und wer es namentlich nicht versäumt, diese Stelle auch im Stehen zu untersuchen, der ist überrascht, wie oft dieses kurze leckende, anstreifende, leicht schabende Geräusch gehört werden kann. Vielfach hat man diese „akzidentellen Reibegeräusche" oder pseudoperikardialen Geräusche auf abnorme Trockenheit des Herzbeutels oder auf Sehnenflecke zurückgeführt.[1]) Noch häufiger aber wird fälschlich Herzbeutelentzündung diagnostiziert. Leider können wir auf diese praktisch wichtigen Dinge nicht näher eingehen, weil sie uns zu weit ab von unserm Thema führen würden. — Es leuchtet ein, daß wegen der gleichartigen Entstehung der sog. kardiopulmonalen oder Herzlungengeräusche und der pseudoperikardialen Geräusche deshalb die Bezeichnung kardiopulmonale oder Herzlungengeräusche nicht korrekt ist; es wäre darum — wie ich glaube — besser, diese Bezeichnung ganz fallen zu lassen, und einfach entweder von akzidentellen oder falschen Herzgeräuschen zu reden im Gegensatz zu den wahren Geräuschen, welche auf einer anatomischen Veränderung des Herzens beruhen.

3. Die Art der Verbreitung der Geräusche ist bei den organischen und akzidentellen Schallerscheinungen eine recht verschiedene. Die Geräusche, welche bei den Ventilfehlern entstehen, verbreiten sich im allgemeinen mehr oder weniger vom Entstehungsherde und zwar in einer Richtung, welche einem jeden Klappenfehler eigen ist. Die organischen Geräusche pflanzen sich z. B. sehr oft gegen die Achselhöhle oder nach der Klavikula fort; sehr oft werden sie auch am

1) Jürgensen hat das falsche perikarditische Reiben für ein Herzmuskelgeräusch erklärt und gar nicht so selten bei vielen Infektionskrankheiten gehört.

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ken, besonders links von der Wirbelsäule wahrgenommen oder sie nzen sich weiter in die großen Gefäße fort. Die k. p. Geräusche l öfter ganz zirkumskript, werden nur an einer ganz eng umnzten Stelle, welche mit dem Trichter des Stethoskopes bedeckt rden kann, gehört; sie pflanzen sich meist sehr wenig oder nicht fort. Niemals — das muß nochmals mit größter Entiedenheit hervorgehoben werden — sind die valvulären Geräusche auf en so engen Raum begrenzt. Das ist für den erfahrenen Beobachter allergrößtem diagnostischen Wert — ein besonders zuverlässiges ysikalisches Zeichen. Hat in solchen Fällen, was öfter gerade am Urunge der großen Gefäße zutrifft, der Klangcharakter der Geräusche nlichkeit mit den häufig engumschriebenen organischen perikardichen Geräuschen, dann wird leicht die falsche Diagnose „Perikaris" gemacht. Allerdings können sich die k. p. Geräusche oft an hreren Stellen, ja sogar entlang dem ganzen linken oder — seltener dem rechten Lungenrande entwickeln, ohne daß es sich um eine gentliche Propagation handelt; da muß man dann genau auf den ythmus achten.

4. Von viel größerem diagnostischen Werte als die Bemmung des Sitzes ist für den praktischen Arzt die genaue Beobchtung des Rhythmus der Geräusche. Unter Rhythmus der Geusche verstehen wir die genauen Beziehungen derselben zu den hasen der Herzbewegung und zu den normalen Tönen.

Die k. p. (akzidentellen) Geräusche sind meistens systolisch. ie bieten bei scharfer Beobachtung einige Eigentümlichkeiten, die zu rkennen von der größten Wichtigkeit ist. Zur genaueren Differenierung derselben von den organischen Geräuschen unterscheidet Potain holosystolische (ὅλος = ganz) Geräusche, d. h. solche, welche ie ganze Systole ausfüllen, und merosystolische (μέρος = Teil) Geäusche, d. h. solche, welche nur einen Teil der Systole einnehmen. Unter den merosystolischen Geräuschen unterscheidet Potain:

a) protosystolische (πρῶτος = der erste), die ganz im Anfange der Systole entstehen und rasch aufhören,
b) mesosystolische (μέσος = Mitte), welche in der Mitte der Systole beginnen und endigen, bevor man den zweiten Ton hört, und
c) telosystolische (τέλος = Ende), welche nur gegen das Ende der Systole gehört werden.

Bei den Klappenfehlern sind die Geräusche — Potain — immer holosystolisch, sie füllen die ganze Systole aus. Die anorganischen Geräusche sind fast immer merosystolisch, sie nehmen nur selten die ganze Systole ein. Nach Potain ist der

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mesosystolische Rhythmus der k. p. Geräusche weitaus der häufigste; er ist nach Potain absolut charakteristisch für die k. p. Geräusche. —

Damit stimmt meine Erfahrung im großen und ganzen überein und wenn ich nicht so ganz feine Unterschiede im Rhythmus der Geräusche wie Potain gemacht habe und speziell auf die Unterscheidung von telosystolischen Geräuschen, welche innerhalb von der Herzspitze noch mehr verspätet als an den andern Stellen der Spitzenregion entstehen sollen, keinen Wert lege, so möchte ich doch ein ganz besonderes Gewicht darauf legen, daß namentlich dann, wenn die Geräusche ihren Sitz über dem linken Ventrikel oder in der Gegend der Herzspitze haben, das k. p. Geräusch sehr deutlich mit einem Tone anfängt, während die Geräusche an der Herzbasis, also hauptsächlich an der Hörstelle der Pulmonalis, genau mit dem systolischen Tone einsetzen, protosystolisch sind. — Die Schallerscheinungen, „welche sich an den ersten Ton anschließen", „welche dem Tone nachschleppen oder angehängt sind" — wie diese akustische Erscheinung von den verschiedensten deutschen Autoren beschrieben wird — das perisystolische Geräusch (Bamberger) und das prädiastolische Geräusch (Sahli) sind sicher keine Insuffizienzgeräusche. Wir wollen die von Potain gewählte Bezeichnung „mesosystolisches Geräusch" beibehalten. Wir werden später noch hören, daß das Geräusch fast mathematisch genau in der Mitte des systolischen Tones anfängt, denselben allerdings aber oft überdauert. So ist also das Verhalten eines Mitralklappengeräusches zum ersten Tone von größter Bedeutung für die Diagnose, und wer es durch Übung erlernt hat, streng auf den Rhythmus eines systolischen Geräusches in der Herzspitzenregion zu achten, der wird leicht diagnostische Irrtümer vermeiden.

Recht oft ist der mesosystolische Rhythmus so evident, daß auch der weniger Geübte, wenn er einfach aufgefordert wird, ganz genau die Aufmerksamkeit auf den Rhythmus zu konzentrieren, mit Leichtigkeit hört, daß das Geräusch erst ein kurzes Weilchen — ich will hier schon bemerken, daß diese Zeit selbstverständlich der Verschlußzeit entspricht — nach dem Beginn des ersten Tones auftritt. Je größer die Übung des Untersuchers ist, desto rascher erkennt er das besonders zuverlässige Auskultationsphänomen. Ist die Entscheidung, ob ein Ton neben dem Geräusche hörbar ist und in welchem Momente der Ton genau beginnt, schwierig zu treffen, so kann man sich mit Vorteil des bekannten Kniffes bedienen und das Ohr ganz wenig vom Stethoskope abheben — der Ton tritt dann oft deutlicher hervor und der Rhythmus wird besser erkannt.

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Die von Potain aufgestellte Regel, daß ein mesosystoches Geräusch sicher ein anorganisches sei, erfährt nur e Ausnahme. v. Noorden hat darauf aufmerksam gemacht, daß Stenose der Aorta nicht selten das systolische Geräusch mit einem he anfängt. Ich selbst habe schon seit vielen Jahren in den Fällen Aortenstenose, bei denen die maximale Intensität des Geräusches der Herzspitze war, weshalb anderwärts Insuffizienz der Mitralklappe gnostiziert wurde, die Diagnose Stenose der Aorta besonders desb stellen können, weil das Geräusch sehr deutlich — sogar beders deutlich, der verlängerten Verschlußzeit entsprechend, mit em Tone angefangen hat.

Aber auch die diastolischen akzidentellen Herzgeräusche sind ht solche Raritäten, wie von vielen Autoren behauptet wird. Wohl erste, der das Vorkommen von einem diastolischen akzidentellen rzgeräusch bei einem Falle von höchster Oligämie nach kopiösen rmblutungen — Sektion: höchste Anämie, Verfettung des Herzens beschrieben hat, ist Friedreich (1862). Nach ihm haben King 871) und Strümpell (1876) je einen Fall von tiefer Anämie behrieben, wo ebenfalls ein diastolisches Herzgeräusch zu hören war. h selbst habe im Jahre 1877 in meiner Monographie „Die progresve, perniziöse Anämie" (62 Fälle) 7 Fälle publiziert, bei denen in hr vorgerücktem Stadium der Krankheit, meist bei gleichzeitig vorndenem Venenpuls und nachweisbarer Dilatation der Herzhöhlen Herzverfettung) ein diastolisches Geräusch am Herzen zum Vorschein am. Eichhorst (1878) hat in 3 von 7 Fällen bei perniziöser Anämie benfalls ein diastolisches akzidentelles Geräusch beobachtet. — Potain at 20mal diastolische Geräusche gefunden und zwar bei den verchiedensten Krankheiten (mit Ausschluß der perniziösen Anämie, die nscheinend von Potain nie beobachtet wurde). — Unter den ca. 80 Fällen, welche ich dieser Arbeit zugrunde gelegt habe, habe ich mal diastolische Geräusche beobachtet (also im ganzen 15 Fälle mit iastolischem Geräusche — nicht mitgerechnet die Fälle von diastoischem akzidentellen Reibegeräusch links unten neben dem Sternum)[1]).

Ich will heute nicht näher eingehen auf diese Fälle, nur das eine möchte ich feststellen, daß auch die diastolischen akzidentellen Geräusche unzweifelhaft in gleicher Weise entstehen wie

1) Die diastolischen Geräusche sind ebenfalls meist dem Tone angehängt (mesodiastolisch). Ihr Sitz ist entweder die Herzbasis, nach Potain hauptsächlich an der Hörstelle der Aorta oder an der Herzspitze, nach meiner eigenen Erfahrung daselbst sogar häufiger.

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die systolischen — durch Reibung der Perikardialblätter aneinander bzw. durch Quetschung der Randpartien der Lungen. Während der Diastole gleitet das Herz auf dem gleichen Wege, den es während der Austreibungszeit gemacht hat, zurück und nimmt genau seine ursprüngliche Stelle wieder ein. Alle andern Erklärungsversuche, wie z. B., daß die diastolischen Geräusche fortgeleitete Venengeräusche seien (Sahli, Krehl u. a.), halten vor einer strengen Kritik nicht stand und sind an Hand der einzelnen Beobachtungen mit Leichtigkeit zu widerlegen.[1])

5. Die k. p. Geräusche sind recht häufig durch eine außerordentliche Veränderlichkeit ausgezeichnet; sie können erscheinen, verschwinden, Ort, Rhythmus und Klangfarbe können von einem Tag auf den andern, ja von einem Augenblicke zum andern wechseln. So kommt es oft vor, daß der eine Untersucher ein lautes Herzgeräusch wahrnimmt, das ein zweiter kurze Zeit nachher nicht mehr hören kann. Die k. p. Geräusche sind auch veränderlich bezüglich ihres Sitzes; ein Geräusch, das sich zuerst im 2. I. R. links hörbar macht, findet sich kurze Zeit später an der Spitze oder umgekehrt. Dieser auffallende Wechsel ist z. B. bei der Chlorose schon lange bekannt, aber nur vielfach wieder vergessen worden.

Der Wechsel der k. p. Geräusche findet entweder spontan statt oder infolge von veränderter Herztätigkeit oder unter dem Einflusse von Änderung der Atmung oder bei Wechsel der Lage des Patienten.

a) Es ist schon lange bekannt, daß im Beginne der Krankenuntersuchung die Herzaktion oft aufgeregt ist. Schon Celsus hat den Rat erteilt, den Puls nicht am Anfange der Untersuchung zu zählen, da er dann leicht frequenter ist, und besonders Bamberger hat nachdrücklich darauf aufmerksam gemacht, wie wichtig für die Vornahme der Herzuntersuchung körperliche und psychische Ruhe des zu Untersuchenden sei, da sonst selbst der Geübte den gröbsten Täuschungen ausgesetzt sei. Gar nicht selten hört man, wenn die Untersuchung des Herzens mit der Auskulation angefangen wird, ein lautes systolisches Herzgeräusch, das nach kürzerer oder längerer Ruhe auf dem Untersuchungsbette vollständig verschwindet. Wiederholt habe ich ein Geräusch nur ganz kurze Zeit, kaum eine Viertelsminute lang, gehört. Meine Schüler erinnere ich deshalb öfter an die Regel, die

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1) Wer sich für dieses spezielle Kapitel näher interessiert, findet in einer kürzlich erschienenen Zürcher Dissertation die von einem meiner Schüler (Charles Favre, Über diastolische, akzidentelle Herzgeräusche. 1907) die genaue Schilderung der Potainschen, meiner und der meisten bisher veröffentlichten Fälle.

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erzuntersuchung nicht mit der Auskultation, sondern mit der Inspek-
on und Perkussion zu beginnen. Da man gewöhnlich eine Hörstelle
ur wenige Sekunden behorcht, macht man sehr leicht falsche dia-
nostische Schlüsse, wenn ein Geräusch am Herzen gehört wird.

Umgekehrt hat man die Behauptung aufgestellt — und in der
prechstunde des Arztes wird noch sehr häufig auf diese Art die
unktion des Herzens geprüft —, daß manche organischen Herzgeräusche
st durch Herumlaufen im Zimmer, durch einige Turnübungen oder
urch öfteres tiefes Einatmen u. dgl. hörbar gemacht werden können.
as ist ein großer Irrtum! Alle Herzgeräusche, die in der Ruhe ganz
hlen und erst bei Anregung der Herztätigkeit zum Vorschein kommen,
nd sicher akzidentelle. Auch die leichten perikardialen Reibege-
iusche, welche erst auf diese Weise bemerklich werden, sind keine
chten Reibegeräusche. Ja selbst die schwachen Geräusche, welche
urch Bewegung eine bedeutende Verstärkung erfahren, sind meist
eine organischen Geräusche.

b) Laennec, der berühmte Erfinder der Auskultation, der bekannt-
ch auch die anorganischen Geräusche schon genau kannte, hat geglaubt,
aß ein Herzgeräusch, welches seinen Sitz in der Lunge hat, ver-
chwinden müsse, wenn die Atmung unterbrochen wird. Viele Autoren
ind noch heute der Meinung, daß man so am leichtesten und ganz
asch die Herzlungengeräusche erkennen und diagnostische Irrtümer
ermeiden könne. Das ist aber noch lange nicht immer der Fall.
Hier und da verwandelt sich das k. p. Geräusch bei forcierter Ein-
tmung in ein sakkadiertes Geräusch. — Bei tiefer Einatmung kann
das k. p. Geräusch beträchtlich an Intensität abnehmen oder selbst
ganz verschwinden und bei der Exspiration wird es bedeutend stärker.
Auch das trifft aber lange nicht in allen Fällen zu. Hier und da
kommt es sogar nur auf der Höhe der Inspiration oder nur bei der
Exspiration zustande. Niemals erfährt ein im Innern des Her-
zens entstehendes Geräusch eine so erhebliche Änderung
der Stärke während der Ein- und Ausatmung. Die Abschwä-
chung der sicher organischen Geräusche, welche durch die Vorlage-
rung der Lunge bedingt wird, ist nach meiner Erfahrung niemals
beträchtlich.

c) Besonders charakteristisch und absolut beweisend für
die Diagnose ist der Wechsel der anorganischen Geräusche
bei Änderung der Lage des Kranken. Von einigen Autoren wurde
schon früh darauf hingewiesen, daß die anorganischen Geräusche bei
Lagewechsel einer Veränderung unterworfen sind, wie sie bei den
organischen Geräuschen nicht in gleichem Maße vorkommt. Herz-

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geräusche, die nur im Liegen oder nur im Sitzen oder Stehen hörbar sind, sind niemals Klappengeräusche. Recht häufig ist dieser Wechsel ein ganz auffälliger; ein Geräusch, das im Liegen hörbar ist, kann spurlos verschwinden beim Aufstehen oder umgekehrt. Geräusche, die nur hier und da einmal in der rechten oder linken Seitenlage gehört werden, sind niemals organische Geräusche. Auch Geräusche, die bei Lagewechsel beträchtlich zu- oder abnehmen, sind sicher k. p. Geräusche. Es muß deshalb mit gebührendem Nachdrucke und wiederholt jedem, der eine Herzuntersuchung zu machen hat, der Rat erteilt werden, die Untersuchung in verschiedenen Positionen — im Liegen und im Stehen — vorzunehmen, wenn ein Geräusch am Herzen gehört wird, und es ist selbstverständlich ganz gleich, ob die Untersuchung zuerst am Stehenden oder am Liegenden vorgenommen wird. Mir macht es bestimmt den Eindruck, daß besonders häufig im Stehen Herzgeräusche gehört werden, die akzidenteller Natur sind. Ich selbst untersuche das Herz fast immer zuerst in liegender Position des Kranken und ich halte es geradezu für einen Kunstfehler, wenn — wie das so sehr häufig geschieht — die Untersuchung des Herzens nur im Stehen vorgenommen wird. — Wenn aber ein Geräusch bei Lagewechsel sich nicht ändert, so ist es nicht notwendigerweise ein organisches Geräusch; denn viele anorganischen Geräusche ändern sich kaum bei Lagewechsel. — Die organischen Geräusche sind lange nicht in dem Maße einer Veränderung fähig; sie sind im allgemeinen fix und verschwinden kaum mehr. Wenn vielleicht eine Ausnahme zu machen ist für die Mitralstenose und die Trikuspidalisinsuffizienz, so dürfte die Diagnose meist keine ernstlichen Schwierigkeiten bereiten, denn der erste dieser Herzfehler ist durch eine Reihe anderer physikalischer Erscheinungen (perkussorischer und auskultatorischer) ausgezeichnet und der zweite ist am positiven Venenpuls leicht zu erkennen.

6. Endlich ist für die Differentialdiagnose der Geräusche noch die Palpation zu verwerten. Akzidentelle Geräusche werden niemals durch das Gefühl wahrgenommen. Das Schwirren, welches manchmal bei tiefen Anämien (z. B. bei der perniziösen Anämie) nachgewiesen werden kann, ist der fühlbare Ausdruck der durch die Verfettung des Herzmuskels bedingten muskulären Insuffizienz.

Die Entstehung des Herzstoßes und der Herztöne. — Zum Verständnis der physikalischen Phänomene, welche wir im vorhergehenden geschildert haben, sind einige Bemerkungen und physiologische Kenntnisse notwendig, und ich muß zunächst meine Ansicht über die Entstehung des Herzstoßes und über die Entstehung der Herztöne, die von den gangbaren Erklärungen in einigen nicht un-

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esentlichen Punkten abweicht, feststellen. Die Literatur über den erzstoß und über die Entstehung der Herztöne ist eine außerordentlich große und es kann heute selbstverständlich nicht meine Aufgabe in, auf dieselbe näher einzugehen. Ich berücksichtige jetzt nur die erneuesten Lehrbücher bzw. die neuesten Auflagen derselben (Sahli, ntersuchungsmethoden. 1905. Romberg, Herzkrankheiten. 1906. ermann, Physiologie. 1905. Tigerstedt, Physiologie. 1907).

Bekanntlich hat Martius, der nach der Markiermethode (Marey) e Herztöne nach dem Gehör auf der Herzstoßkurve mittels eines ektrischen Signals verzeichnet hat, die Lehre aufgestellt, daß die nze Erscheinung des Herzstoßes in die Verschlußzeit fällt, h. in die Zeit, in welcher noch alle Klappen geschlossen sind.

In seiner Abhandlung „Über den Herzstoß des gesunden und kranken Menhen" (1894) sagt Martius: „Wir können den Vorgang des Herzstoßes ohne weire Hilfsmittel durch reine Betrachtung noch weiter analysieren. Wenn man einen t ausgebildeten Spitzenstoß eines jugendlichen Individuums aufmerksam befühlt, kann man meist deutlich 3 Phasen des Vorganga unterscheiden: 1. ein Vorgeölbtsein der Brustwand, 2. ein mehr oder weniger brüskes Zurücksinken derselben, eine Pause, während welcher nichts von Bewegung gefühlt wird."

Dieser Lehre habe ich seinerzeit in einem Vortrage „Eine neue Theorie des erzstoßes (1896)" Opposition gemacht und habe gezeigt, daß wir — hie und da — n Vorgange der Herzbewegung deutlich 4 Phasen unterscheiden können: 1. eine eine hügelige Hervorwölbung, 2. eine wellenförmige, pendelartige Bewegung nach echts, 3. 2—3 cm nach innen von der systolischen Hervorwölbung eine zweite etwas eniger starke und weniger umfangreiche Hervorwölbung, welche nach der Auskultation ganz deutlich mit dem 2. Tone zusammenfällt, 4. eine schattenartige, ückläufige blitzschnelle Bewegung von der Stelle des 2. Stoßes zur Stelle des 1. Das — wie ich glaube — von mir zuerst beschriebene Phänomen des doppelten Herzstoßes habe ich als Ictus cordis alternans bezeichnet.) Die erste Hervorwölbung führte ich — wie Martius — auf die Verschlußzeit zurück.

Ich hatte damals — wie Martius, R. Geigel und zahlreiche ndere — noch nicht genügend bedacht, daß wir mit dem einfachen Gehör überhaupt gar nicht imstande sind, den Anfang eines Herztones zeitlich genau zu markieren. — Untersuchungen von Schmidt, Hürthle, Hilbert, Hochhaus u. a. haben außerdem gezeigt, daß die Periode der Vorwölbung länger dauert als die Verschlußzeit, und daß der Karotispuls beginnt, bevor das Ansteigen des Herzstoßes den Gipfel erreicht hat. Die meisten modernen Kliniker haben deshalb die Martiussche Lehre, daß der Herzstoß eine Funktion der Verschlußzeit sei, verlassen und geben nur zu, daß an der Bildung des Herzstoßes die Formveränderung des Herzens während der Verschlußzeit, die plötzlich eintretende Härte desselben den größten Anteil haben. Auch das möchte ich bezweifeln und bin der Meinung, daß der Herzstoß hauptsächlich in den Anfang der Austreibungszeit fällt und wesentlich abhängig ist von der Be-

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wegung des Herzens, welche dasselbe durch die Geradestreckung der Aorta, durch Spreizung des Aortenbogens beim Ausströmen des Blutes erfährt. Durch eine einfache Fragestellung bin ich dazu gekommen, die geläufige Lehre von der Entstehung des Herzstoßes in diesem Sinne zu korrigieren. Ich habe mich gefragt: Wie groß kann die Formveränderung des Herzens sein, die während der Verschlußzeit entsteht, während welcher die Wandung des blutgefüllten Herzens sich um den inkompressiblen Inhalt zusammenzieht und den Druck so steigert, daß die Taschenklappen eröffnet werden? Darauf mußte ich antworten: Die Formveränderung des Herzens während der Verschlußzeit kann nur eine geringe sein und jedenfalls nicht so groß, daß sie den sichtbaren Herzstoß erzeugen kann.

Durch weitere Überlegung bin ich dazu gekommen, zu sagen:

1. Wenn der erste Ton an der Spitze wirklich ein Klappenton ist und durch Spannung der Vorhofsklappen entsteht, dann kann er nur so lange dauern wie die Verschlußzeit.

2. Der 1. Ton an der Herzbasis, der nach der bisher allgemein akzeptierten Lehre von Bamberger durch plötzliche Spannung der Arterienwand im Wurzelgebiete der großen Gefäße entsteht, muß etwas später eintreten als der 1. Ton an den Atrioventrikularklappen.

Ich habe daraufhin gesucht und in Pflügers Archiv für Physiologie (1894) eine oft zitierte, aber offenbar wenig beachtete Arbeit gefunden, welche meine aprioristischen Schlußfolgerungen glänzend bestätigt hat.

Einthoven und Geluk haben durch einen sehr ingeniösen Apparat unter Anwendung eines äußerst empfindlichen Mikrophons die Herztöne aufgenommen und zwar haben sie mittels eines Kapillarelektrometers die Stromschwankungen, welche durch die Erregung eines stromdurchflossenen Mikrophons hervorgerufen werden, photographisch registriert — unter gleichzeitiger Anwendung des Kardiographen. Ich habe mich durch Rücksprache mit Kollegen Höber vergewissert, daß die Versuchsanordnung von Einthoven von größter Zuverlässigkeit ist. Mit einer bisher unerreichten Genauigkeit bis auf einige Zehntausendstel einer Sekunde kann der Anfang jedes Tones bestimmt werden.

„Die Kurve C stellt das menschliche Kardiogramm dar, S ist die Zeitlinie, deren jede Abteilung 0,02 Sekunden entspricht. Die breiten Streifen 1 und 2 bedeuten den 1. und 2. Herzton. Der 1. Ton tritt eher an der Herzspitze als im 2. Interkostalraum hervor; bei a fängt der 1. Spitzenton an, bei b der 1. Aorta- und Pulmonalton. (Weder vom 1. noch vom 2. kann der genaue Zeitpunkt angegeben werden, worauf sie endigen; dieser Zeitpunkt muß irgendwo in den schraffierten Stücken cd und fg gefunden werden.) An der Herzspitze ist der 1. Ton schon vorhanden — bei aa_1 — vor dem Anfange des Kardiogrammes. Dieses Ergebnis könnte auf verschiedene Weise gedeutet werden, wir gehen jedoch

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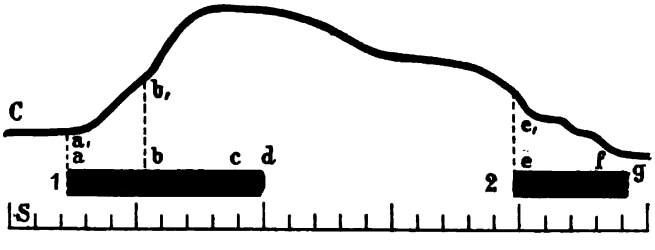
cht näher darauf ein, weil das Zeitintervall nur klein ist. Eine größere Bedeutung hat die Tatsache, daß im 2. I. R. der 1. Ton in dem Augenblick fängt, wo ungefähr die Hälfte des anakroten Teiles des Kardiogramms vollendet ist (bb_1).

„Wenn wir uns die Frage stellen, welchem Umstande es zugeschrieben werden ß, daß der 1. Ton als Aorta- oder Pulmonalton so viel später als der 1. Spitzen- anfängt, so erblicken wir zwei Möglichkeiten. Entweder man hört im 2. Interstalraume als 1. Ton einen andern Schall als an der Herzspitze — und zwar rvorgerufen durch die plötzliche Spannung der Arterienmembran oder die Semilunarklappen verhindern — solange sie noch geschlossen sind — Fortpflanzung des Tones von den Ventrikeln nach den Arterien. In beiden llen wird der 1. Ton im 2. I. R. anfangen müssen in dem Augenblicke, wo die milunarklappen sich öffnen, und daraus folgt, daß der Anfang dieses Tones n ausgezeichnetes Mittel darstellt, diesen Zeitpunkt zu bestimmen ehe das Kardiogramm b_1).

Der Anfang des 2. Tones liegt im Kardiogramm bei e_1 und gibt hier den Zeitnkt an, wo die Semilunarklappen geschlossen werden. Die ganze Herzsystole uß also in 2 Perioden geteilt werden, die 1. a_1b_1 — durchschnittlich 061 Sekunden andauernd — worin der Druck in den Ventrikeln sich erhöht, er doch noch ungenügend bleibt, das Blut in die Arterien zu treiben — die 2. e_1, die Austreibungsperiode des Blutes, anfangend mit der Öffnung, ndigend mit der Schließung der Semilunarklappen.“



Die genaue Prüfung des Einthovenschen Kardiogrammes ergibt n der Tat:

1. Erst am äußersten Ende der Systole sinkt das Plateau der Kurve, as sich gegen die Abszisse neigt, kaum merklich unter die Stelle, elche im aufsteigenden Schenkel während der Verschlußzeit erreicht vorden ist; daraus ergibt sich, daß die Ursache des normalen Herzstoßes zur Hauptsache in der Bewegung zu suchen ist, elche das Herz beim Ausströmen des Blutes erfährt.

2. Die Austreibungsperiode dauert fast 5mal länger als lie Verschlußzeit.

3. Der systolische Ton an der Herzspitze, der auch für das Ohr erkennbar länger ist als der 1. Ton an den großen Gefäßen, nimmt nicht ganz die Hälfte der Systole ein; er klingt ganz genau

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so viel länger als der 1. Ton an der Herzbasis, als die Verschlußzeit dauert.

4. Der 1. Ton an den großen Gefäßen dauert genau so lange wie der 2. Herzton.

Die Pause zwischen den beiden Herztönen dauert etwas länger als der systolische Ton. Es ist deshalb ganz falsch, wenn in einem bekannten Lehrbuche der Physiologie die Lehre verbreitet wird — „der 1. Herzton hält so lange an wie die Systole der Kammern". Wäre dem so, so würden wir ja gar keine Pause zwischen den beiden Tönen hören, die beiden Töne müßten in einen verschmelzen. Aus dem gleichen Grunde kann aber auch der 1. Herzton unmöglich ausschließlich ein Muskelton oder ein Spannungston sein, der während der ganzen Systole durch Spannung der ganzen Umwandung des Ventrikels erzeugt wird. Die Frage, ob nicht der Herzmuskelton als Komponente des 1. Herztones anzunehmen sei, ist seit den Experimenten von Ludwig und Dogiel (1868), welche gezeigt haben, daß man den ersten Ton auch am herausgeschnittenen, blutleeren Herzen hört, oft und viel diskutiert worden und der Streit ist auch heute noch nicht entschieden. Ich muß es mir versagen, hier auf die Streitfrage näher einzugehen. Nur das muß erwähnt werden: Blutdruckversuche am schlagenden Herzen haben gezeigt, daß die Druckzunahme, welche ja nur von der Zuckung abhängt, bis gegen das Ende der Systole anhält, oft kurz vor dem 2. Tone erst ihren Höhepunkt erreicht. Desgleichen ergeben die Aktionsströme des menschlichen Herzens, wie noch jüngst von Einthoven durch photographische Registrierung der Ausschläge seines Saitengalvanometers nachgewiesen worden ist, daß die Kammersystole einer einfachen Muskelzuckung gleichzustellen ist und nicht als eine summierte Zuckung aufgefaßt werden kann. Zudem sind nach meiner Meinung die physikalischen Bedingungen für das Zustandekommen eines Tones am herausgeschnittenen blutleeren Herzen ganz andere als an dem blutgefüllten lebenden Organe. Ich meinerseits bezweifle ganz entschieden, daß dem Herzmuskelton — wenn überhaupt ein solcher entsteht — ein nennenswerter Anteil an der Bildung des 1. Herztones zugeschrieben werden kann. Die klinische Erfahrung spricht gar nicht dafür und es ist durchaus nicht nötig, die alte bewährte Lehre, daß die Herztöne[1]) nur durch Spannung von elastischen Membranen entstehen, zu ver-

1) Daß der 1. Spitzenton aus 2 Komponenten besteht, ist übrigens schon lange nachgewiesen worden. Wintrich hat vermittelst passender Resonatoren beide Töne voneinander unterscheiden können: den helleren, kürzeren Klappenton, sowie das „tiefere, längere Muskelgeräusch".

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sen. Wenn während der Verschlußzeit der Ventrikel plötzlich die ıze Umwandung in Spannung versetzt wird, ist es begreiflich, daß bei die zarten elastischen Vorhofklappen weit geeigneter zur Tondung sind als die dicke Muskulatur — das lehrt schon die Enthung des 2. Tones, der nach der unbestrittenen Annahme aller nur rch Spannung, durch den plötzlichen Schluß der Semilunarklappen rvorgebracht wird. In gleicher Weise ist auch der rasch auf die ır elastische Aortenwandung ausgeübte Druck, bedingt durch das h Öffnen der Semilunarklappen hervorstürzende Blut, viel eher die sache eines Tones als der bei der Herzkontraktion langsamer auftende, auf die viel weniger elastische Herzwandung erfolgende gendruck des Blutes.

Der 1. Ton über den Kammern baut sich demnach aus 2 standteilen auf: aus einem Tone, der durch Anspannung r Vorhofsklappen (plus Semilunarklappen), und aus einem ne, der durch Schwingungen der elastischen Membran im urzelgebiete der großen Gefäße erzeugt wird. In der Polinik pflege ich der Kürze halber die erste etwas kürzere Komponte des 1. Herztones als „Verschlußton" und den 2. etwas längeren il als „Austreibungston" zu bezeichnen. — Der Austreibungs- oder efäßton wird natürlich, da die Türe zwischen der Kammer und rta offen ist, noch besser nach der Herzspitze fortgeleitet als der Ton, der durch Schluß der Semilunarklappen entsteht und bei verhlossener Türe nach der Hörstelle der Mitralis fortgeleitet wird. — er allgemeine Brauch, den 1. Ton an der Herzspitze oder rechts ıten am Sternum als Mitral- oder Trikuspidalklappenton zu bezeichen, ist deshalb ganz unberechtigt und es wäre viel korrekter und eniger irreleitend, vom systolischen Ton der linken oder der rechten ammer zu sprechen.

Ich rekapituliere:

1. Der Herzstoß entsteht hauptsächlich im Anfange der Austreibungsit bei der Bewegung des Herzens.
2. Es entstehen über dem Herzen sechs verschiedene Töne:
   a) Vier systolische — je einer über den Ventrikeln und je einer am Ursprunge der großen Gefäße. Die systolischen Töne fangen nicht im gleichen Momente an. Der systolische Ton an den großen Gefäßen erklingt etwas später als der systolische über den Kammern; der systolische Ton an der Spitze setzt sich aus zwei Teilen zusammen — aus dem eigentlichen Klappentone und aus dem fortgeleiteten Gefäßton.
   b) Die zwei diastolischen Töne entstehen über den Semilunarklappen der Aorta und Pulmonalis.

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Auf Grund dieser einfachen und klaren, durch physiologische Tatsachen erhärteten Lehre von der Entstehung des Spitzenstoßes und der Herztöne, welche ich schon seit mehreren Jahren meinen Schülern vortrage, und welche in dieser präzisen Form meines Wissens auf keiner Klinik gelehrt wird, können wir nun die physikalischen Erscheinungen, welche wir früher geschildert haben, leicht verstehen. Daß die akzidentellen Geräusche in der Spitzenregion und über dem linken Ventrikel den so sehr charakteristischen mesosystolischen Rhythmus haben, das macht nun dem Verständnis keine Mühe mehr. – Nur beiläufig will ich noch bemerken, daß auch verschiedene andere physikalische Erscheinungen, welche sich am Krankenbette zeigen und deren Erklärung bisher großen Schwierigkeiten begegnete (gespaltener — doppelter 1. Herzton — manche Fälle von Galopprhythmus — verstärkter, paukender systolischer Ton bei Mitralstenose — lauter Herzton bei hochgradiger Herzschwäche usw.), viel leichter zu erklären sind, wenn wir uns an die Tatsache erinnern, daß der 1. Ton an der Hörstelle der Mitralis (bzw. Trikuspidalis) aus zwei Tönen – dem Mitralklappen- und dem Arterientone — besteht. Doch darauf können wir jetzt nicht eingehen.

Ätiologie und Vorkommen. Wenn mir — wie das schon öfter vorgekommen ist — der wohlbegründete Einwand gemacht wird, warum nicht immer und bei allen Menschen k. p. (akzidentelle) Geräusche gehört werden, da doch die Bedingungen zur Entstehung derselben scheinbar immer vorhanden sind, dann muß ich immer wieder daran erinnern, daß nach unsern frühern physikalischen Erörterungen in der Tat immer ein Geräusch entstehen muß, wenn bei der Herzrevolution eine Verschiebung des innern Blattes des Herzbeutels dem äußern gegenüber stattfindet und das Herz an den Randzonen der Lunge vorbeigleitet. Das Geräusch ist nur unter gewöhnlichen Verhältnissen so schwach, daß es vom menschlichen Ohr nicht wahrgenommen werden kann. Wenn der Druck der übereinander gleitenden Flächen ein größerer wird und wenn die Geschwindigkeit der Bewegung steigt, dann kann das Geräusch so anwachsen, daß es gehört wird. —

Die Erfahrung lehrt nun, daß an den Stellen und bei den Individuen, wo eine dünne Schicht von Lungengewebe zwischen Herz und Brustwand sich einschiebt, die Geräusche leichter entstehen — daher besonders in der Gegend der Lingula des obern Lappens und da, wo der Conus arter. der Pulmonalis der Lunge bzw. der Brustwand am dichtesten anliegt. Die Dicke der Lungenränder und ihr Verlauf ist großen individuellen Schwankungen unterworfen; so kann der linke Lungenrand, wie genaue anatomische Untersuchungen (Anatom. Institut

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rich) ergeben haben, bis an den rechten Sternalrand und der rechte ngenrand bis nach links über das Sternum reichen. Je dünner die erlagernde Lungenschichte ist, desto leichter entsteht ein Geräusch. enn die Randpartien der Lunge bei einer tiefen Inspiration sich rk blähen, so kommt nicht so leicht ein Geräusch zustande oder Geräusch kommt erst bei der Exspiration zum Vorschein. Jeden- s ist der Verlauf und die Dicke der Lungenränder von wesent- ner Bedeutung für den Wechsel der Geräusche bei Lageverände- ng. Mit Zurückdrängung der Lungenränder, stärkerem Anliegen Herzens an die Brustwand muß es zusammenhängen, daß die identellen Geräusche besonders häufig nur in stehender Posi- n gehört werden — dabei wirkt natürlich wesentlich verstärkend die Geräuscherzeugung mit die bei Nervösen so sehr häufig beob- tete Labilität des Pulses.

Der Grad der Atmungstätigkeit ist ebenfalls von Bedeutung. enn die Herztätigkeit sich beschleunigt und der Atmungsrhythmus eichzeitig ein bedeutend rascheres Tempo annimmt — wie bei arken körperlichen Anstrengungen — dann kommen k. p. Geräusche lten zustande. Potain und Vaquez haben Untersuchungen bei rnern angestellt und haben bei intensiver körperlicher Arbeit keine eräusche beobachtet. Ich selbst habe schon im Jahre 1891 — da- ls, um die Frage der akuten Herzdilatation zu studieren — bei rnern, welche sehr anstrengende Übungen — Schwingen im Knick- tz, ein- und zweihändiges Heben von 50 kg schweren Steinen vom den bis zur Hochhebhalte — bis zur Erschöpfung ausführten, wohl deutende Steigerung der Puls- und Atemfrequenz, aber in keinem nzigen Falle eine Dilatation des Herzens oder ein Herzgeräusch ftreten sehen.

Ganz anders verhält es sich, wenn die Herzfrequenz sich er- öht und der Herzschlag gleichzeitig verstärkt wird, während r Atmungsrhythmus der gleiche bleibt — da entstehen k. p. Ge- äusche sehr gern. Beschleunigung der Herztätigkeit mit gleich- itiger Verstärkung des Herzschlages beobachtet man in unserm ervösen Zeitalter außerordentlich häufig.

Psychische Erscheinungen, leichte körperliche Bewegungen und die erschiedensten andern Momente können die abnorm kräftige Herz- ktion auslösen. Leichte Gemütsbewegungen sind ohne Zweifel die rsache, daß im Sprechzimmer des Arztes, bei der militärischen Unter- uchung, bei den Untersuchungen für Lebensversicherungen[1]) u. dgl.

1) Die amerikanischen Ärzte Collom und Prince Morton (zit. nach Po- ain und Vaquez) haben bei Untersuchungen für Lebensversicherungen der erste

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oft k. p. Geräusche gehört werden. Nicht mit Unrecht hat Vaquez die Geräusche deshalb als bruits de consultation bezeichnet. — Nochmals und mit besonderem Nachdrucke muß hervorgehoben werden, daß die schon nach leichten Turnübungen öfter auftretenden Herzgeräusche nur auf die beschleunigte und verstärkte Herztätigkeit zurückzuführen sind. Oft genug habe ich beobachtet, daß bei einfachem Wechsel aus der liegenden in die stehende Position plötzlich die Pulsfrequenz um 20, 40, ja sogar 60 Schläge gestiegen und dabei ein lautes Herzgeräusch zum Vorschein gekommen ist, das bei Eintritt psychischer Ruhe wieder vollständig verschwand. Man findet diese abnorm kräftige Herzaktion und gleichzeitige Beschleunigung der Herztätigkeit und Herzgeräusche vor allem bei Morbus Basedowii (bei dem die Geräusche fast nie fehlen), bei nervösen, hysterischen und neurasthenischen Patienten, bei den verschiedensten Herz- und Gefäßneurosen, bei Chorea, in der ersten Hälfte der Schwangerschaft. Man hat bei diesen Affektionen die Geräusche vielfach als „nervöse" bezeichnet und einzelne haben auch unglaubliche Theorien für die Entstehung vom Zentralnervensystem aus ersonnen. Man findet diese abnorme, geräuscherzeugende Erregbarkeit des Herzens auch recht oft im Alter der Pubertät, ohne daß eine besondere Nervosität nachzuweisen ist — oft genug tritt die verstärkte und beschleunigte Herzaktion auch ohne jede bekannte Ursache ein.

Auch bei der Chlorose und bei vielen Fällen von Anämie ist offenbar die durch ihren Einfluß auf das Zentralnervensystem erzeugte Exzitation des Herzens die Ursache der Herzgeräusche. Sorgfältige Untersuchungen von Potain und Vaquez haben schon ergeben, daß das Auftreten der sog. „anämischen" Geräusche gar nicht abhängig ist von dem Grade der Chlorose und der Verminderung der roten Blutkörperchen. Zahlreiche andere Gründe sprechen auch dagegen, daß die Geräusche bei Chlorose und Anämie auf einer funktionellen Klappeninsuffizienz beruhen. Doch darauf will ich jetzt nicht näher eingehen.

In gleichem Sinne wie die psychische Emotion wirkt auch das Fieber herzerregend — alle sog. febrilen Geräusche sind nichts anderes als akzidentelle Herzgeräusche. Man hört k. p. Geräusche gar nicht selten am Anfange und im Verlaufe von fieberhaften Krankheiten, besonders wenn nicht gleichzeitig die Atmung beschleunigt ist, wie z. B. bei der Pneumonie, bei der man die Geräusche selten hört. — Nach Potain und Vaquez, welche die Häufigkeit der k. p. Geräusche

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27 mal unter 200 Fällen (d. h. in $^1/_7$) und der zweite sogar bei 35 % der Untersuchten anorganische Herzgeräusche gehört.

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den verschiedenen Krankheiten in einer Kurve graphisch dargestellt
ben, findet man die Geräusche — außer bei M. Basedowii und
lorose — am häufigsten bei Masern, Scharlach und beim akuten
lenkrheumatismus. Damit stimmt auch meine Erfahrung überein.
Auch die Herzgröße übt einen Einfluß auf das Zustande-
mmen der k. p. Geräusche. Bei normal großem Herzen ent-
hen die Geräusche viel häufiger als beim vergrößerten Organe.
n général les petits cœurs soufflent et les grands cœurs sont silen-
ux" (Vaquez). Daß das den französischen Autoren Potain und
quez offenbar nicht bekannte pseudoperikardiale Reiben besonders
ne bei dilatiertem Herzen entsteht, ist schon erwähnt; aber auch
eigentlichen kardiopulmonalen Geräusche kann man nicht so selten,
e die beiden französischen Autoren angeben, selbst bei ganz be-
utender Dilatation des linken Ventrikels in der Spitzenregion 3 bis
cm außerhalb von der Mammillarlinie zu hören bekommen. Diese
räusche werden ganz allgemein als organische aufgefaßt — bedingt durch
ative Insuffizienz der Mitralklappen. Darüber später noch ein Wort.
Von wesentlicher Bedeutung für das Zustandekommen der
p. Geräusche ist auch der Stand des Zwerchfells. Bei Hoch-
nwangeren und bei vielen andern Menschen mit stärkerer Auf-
ibung des Unterleibs wird das Herz viel ausgedehnter der Brust-
nd angelagert und an das äußere Blatt des Herzbeutels angedrückt;
durch wird die Geräuschbildung begreiflicherweise sehr begünstigt.
ist auch meines Erachtens das Geräusch zu erklären, welches ge-
gentlich willkürlich durch starkes Pressen bei ganz Gesunden hervor-
rufen werden kann (Gerhardt). Daß im Alter von 10—14—16 Jahren
e Geräusche so häufig gehört werden, ist sicher zum Teil auf den
diesem Alter beobachteten höheren Stand des Diaphragmas, auf die
nfangreichere Entblößung und das stärkere Anliegen des Herzens
die Brustwand zurückzuführen. Vermehrter Druck der übereinander-
gleitenden Flächen und die größere Wandständigkeit des Herzens ist
ch, wie bereits hervorgehoben, sicher der Hauptgrund, daß die
eräusche viel häufiger in stehender als in liegender Position gehört
erden. Durch stärkeres Andrücken der Gleitflächen werden auch
hne allen Zweifel die Geräusche erzeugt, welche man durch starkes
ufdrücken des Stethoskopes zur Wahrnehmung bringen kann.

Das Alter spielt bei der Entwicklung der k. p. Geräusche
ine große Rolle. Nach Potain, der auf seiner Klinik nur Kranke,
ie das 15. Altersjahr überschritten hatten, beobachtete, kommen die k. p.
Geräusche am häufigsten im Alter von 15—30 Jahren vor. Ich kann
ies nach meiner Erfahrung bestätigen und kann ergänzend hinzufügen,
aß die Geräusche auch im Kindesalter recht oft zur Beobachtung

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kommen. In der medizinischen Poliklinik, wo alljährlich mehrere Tausend Kinder (bis 5000 auf eine Gesamtzahl von 15000 Patienten) behandelt werden, habe ich in den letzten 8 Jahren, in welchen ich mich besonders intensiv mit dem Studium der Herzgeräusche beschäftigte, k. p. Geräusche besonders oft bei Kindern vom 10. bis 14. Altersjahre beobachtet. — Hochsinger, Soltmann u. a. leugnen, daß akzidentelle Herzgeräusche in den ersten 3 Lebensjahren vorkommen. Andere Beobachter (Rheiner, 1903, Cornelia de Lange, 1907, usw.) erheben dagegen Widerspruch; ich selbst habe schon seit Jahren ab und zu einmal im frühesten Kindesalter Herzgeräusche beobachtet, für die keine plausible Ursache zu finden war, und ausnahmsweise habe ich auch durch die Sektion das Fehlen einer anatomischen Grundlage für die Bildung eines Herzgeräusches nachweisen können.

Vor kurzem hat Lüthje an einem großen Beobachtungsmaterial nachgewiesen, daß im Kindesalter vom 6.—16. Altersjahre akzidentelle Geräusche an der Pulmonalis sehr häufig gefunden werden, „daß sie fast als Regel gelten können". Lüthje hat 854 Schulkinder untersucht und fand 612mal (71,6%) ein systolisches Geräusch (509mal schon in der Ruhe, d. h. gleich nach dem Entkleiden, 103mal erst im Anschluß an einige Turnübungen). Lüthje fand, daß die Geräusche im Exspirium unverhältnismäßig an Stärke zunehmen, während sie im tiefsten Inspirium unverhältnismäßig abnehmen oder ganz verschwinden. Er nimmt an, daß die akzidentellen Geräusche am ungezwungensten ihre Erklärung als „Pulmonalstenosen-Geräusche" finden — bedingt durch die Raumverhältnisse an der Stelle, wo die Pulmonalis liegt, und durch den Wechsel dieser Raumverhältnisse im In- und Exspirium. Leider muß ich es mir versagen, hier näher auf die Arbeit von Lüthje einzugehen; wer dieselbe aber vorurteilslos bis in ihre Einzelheiten verfolgt und meine Auseinandersetzungen über die Entstehung der k. p. (akzidentellen) Geräusche einer nochmaligen Prüfung unterzieht, der wird zugeben müssen, daß die kardiopulmonale Entstehungstheorie der Geräusche weit einfacher und zutreffender ist. Lüthjes Erklärung der Geräusche als Pulmonalstenosengeräusche ist eine physikalische Unmöglichkeit.

Physikalisch undenkbar ist es auch, daß die akzid. Geräusche an der Hörstelle der Pulmonalis in der Arterie selbst durch die vermehrte Strömungsgeschwindigkeit entstehen. Es ist nicht einzusehen, wie durch Erhöhung der Blutströmungsgeschwindigkeit im Sinne der Weberschen Versuche endokardiale Geräusche nur an der Pulmonalis entstehen sollen und nicht auch oder sogar eher an der Aorta, wo nach der übereinstimmenden Angabe aller Untersucher akzid. Geräusche nur selten gehört werden. Ganz unmöglich ist es auch, die akzid. Geräusche in der Herzspitzenregion, wo sie bei Erwachsenen mindestens so häufig wie an der Hör-

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le der Pulmonalis gefunden werden, aus der vermehrten Strömungsgeschwin-
keit zu erklären. Zugunsten der kardiopulmonalen Entstehung bzw. der Reibungs-
orie aller akzid. Geräusche spricht selbstverständlich die Möglichkeit, die Ge-
sche an allen Hörstellen einheitlich zu erklären. In der Verstärkung und
chleunigung des Herzschlages, in der psychischen Erregung, welche auch nach
thje eine besondere Rolle für die Entstehung „des Pulmonalstenosen-Geräusches"
elt, haben wir — wie das früher ausführlich auseinandergesetzt worden ist —
e vollständig ausreichende Erklärung für die wesentliche Verstärkung der ge-
scherzeugenden Verschiebung zwischen Herz und Lungen (bzw. innerem und
erem Blatte des Herzbeutels).

Im Frühjahr und Sommer 1907 habe ich die von Lüthje gefundenen
tersuchungsergebnisse einer Prüfung unterworfen und habe zum
l unter Mithilfe von langjährigen Assistenzärzten, die im Auskul-
ren sehr bewandert sind, im Verlaufe von wenigen Monaten 300
unde Kinder (250 im Ambulatorium der medizinischen Poliklinik
d 50 in der Privatpraxis) untersucht und habe nur 85mal, d. h.
28% aller untersuchten Fälle, Herzgeräusche gefunden. — Ich habe
die kleine Statistik noch 89 Kinder im Alter von 0—6 Jahren aufge-
nmen und habe gefunden:

Alter	von	0— 3	Jahren	bei	39	Kindern	1	mal,	d. h. in	2,5%[1])
„	„	4— 6	„	„	50	„	8	„	„ „ „	16%
„	„	7—10	„	„	89	„	26	„	„ „ „	28%
„	„	11—16	„	„	122	„	50	„	„ „ „	40%

rzgeräusche — in der überwiegenden Mehrzahl der Fälle nur „an
r Hörstelle der Pulmonalis". Ich habe aber auch gar nicht
ten ebenfalls ein Geräusch in der Herzspitzenregion gefunden, und
nnte, wie das schon früher ausdrücklich hervorgehoben worden ist,
t Sicherheit nachweisen, daß das Geräusch in der Gegend der Herz-
tze nicht von der Pulmonalis einfach fortgeleitet war. Auffallend
ufig hat das Geräusch in der Gegend des Conus art. der Pulmo-
is, was ebenfalls schon früher betont worden ist, einen wesentlich
heren, kratzenden Charakter. — Ich habe also sehr viel seltener
Lüthje (Lüthje im Alter von 7—16 Jahren bei 71,6% — ich nur
36%) bei gesunden Kindern Herzgeräusche nachweisen können.
ese große Differenz in den Prozentzahlen ist unzweifelhaft so zu
klären, daß Lüthje seine Massenuntersuchungen nur an den
ehenden Kindern und bei größerer psychischer Erregung
rgenommen hat, während alle meine Untersuchungen bei größerer
rperlicher und psychischer Beruhigung — in Rückenlage auf
nem Untersuchungsbette — gemacht worden sind.[2])

1) Cornelia de Lange hat bei 1800 Kindern unter 4 Jahren nur 29mal (in
%) akzid. Herzgeräusche gefunden.

2) Lüthje fand bei den 854 von ihm untersuchten Kindern 703mal eine

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Diagnose. Für die Praxis ist es von allergrößter Bedeutung, die akzidentellen Herzgeräusche von den sog. organischen unterscheiden zu können. Recht oft ist die Differentialdiagnose zwischen den beiden Geräuscharten nicht nur per exclusionem zu machen; denn wer durch stete Übung die nötige Geschicklichkeit sich erworben und ein in allen akustischen Einzelheiten erfahrenes Ohr hat, und wer es erlernt hat, seine Aufmerksamkeit scharf auf den Sitz, Rhythmus, Schallcharakter und die Art der Ausbreitung der Geräusche zu richten, und wer es nie unterläßt, das Herz in verschiedener Stellung des Patienten zu behorchen, dem wird es recht oft möglich, schon bei der ersten sorgfältigen Untersuchung eine bestimmte Diagnose zu stellen.

Langjährige Erfahrung hat mich gelehrt, daß hauptsächlich drei organische Herzaffektionen öfter fälschlich diagnostiziert werden, wo es sich um einfache akzidentelle Herzgeräusche handelt — Endokarditis, Perikarditis und vor allem Schlußunfähigkeit der Mitralklappen. Über die beiden ersteren will ich mich kurz fassen, auf die so auffallend häufig irrtümlich angenommene Insuffizienz der Mitralis muß ich notwendig noch etwas näher eingehen.

Die Diagnose Endokarditis wird viel zu oft gemacht. Es kann nicht genug zur Vorsicht gemahnt werden, nicht sofort an eine Endokarditis zu denken, wenn bei Scharlach, akutem Gelenkrheumatismus und anderen fieberhaften Infektionskrankheiten schon in den ersten Tagen der Erkrankung Geräusche am Herzen (Hörstelle der Pulmonalis oder Spitzenregion) auftreten. Diese Geräusche können sicher nicht durch eine Veränderung der Klappen bedingt sein — lehrt ja doch die Erfahrung, daß geringfügige und sogar tiefergreifende Veränderungen an den Klappen bei der benignen Endokarditis das Spiel

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Akzentuation des 2. Pulmonaltons (also in 82%). Ich habe die Verstärkung des 2. Tones an der Hörstelle der Pulmonalis, die mir schon seit vielen Jahren (siehe den eingangs erwähnten 1. Fall vom Jahre 1890) öfter auffiel, nur in 20% der Fälle notiert. Es ist einleuchtend, daß bei der Diagnose „Verstärkung des 2. Pulmonaltones" dem subjektiven Ermessen ein großer Spielraum gelassen ist, hat ja doch bekanntlich Vierordt bei genauen Messungen gefunden, daß am gesunden Menschen der 2. Pulmonalton etwas lauter klingt als der 2. Aortenton. Jedenfalls habe ich schon seit langer Zeit diesem physikalischen Zeichen besonders im jugendlichen Alter von 10—18 Jahren kein Gewicht beigelegt und mit der ausdrücklichen Betonung, daß die Verstärkung des 2. Tones „an der Hörstelle der Pulmonalis" wahrgenommen wird, möchte ich andeuten, daß nach meiner Auffassung die Verstärkung des 2. Tones gar nicht notwendigerweise von der Pulmonalis stammt, sondern daß vielmehr die Verstärkung des 2. Tones an der genannten Hörstelle einfach durch Summation der beiden 2. Töne (Aorta und Pulmonalis) entsteht. Mit dieser Erklärung ist — wie mir scheint — „das physikalische Rätsel" am einfachsten zu lösen.

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Klappen nicht hindern und daß gewöhnlich erst nach längerem
tehen der Entzündung, besonders bei dem so häufigen Sitze an
Mitralsegeln, die Klappenfunktion gestört wird. — Bekanntlich
d auch nicht allzuselten maligne Endokarditis diagnostiziert, wo
der nachfolgenden Sektion keine anatomische Veränderung an den
ppen nachgewiesen werden kann.

Das gleiche gilt von der Perikarditis, die ebenfalls viel zu häufig
enommen wird.[1]) Reibegeräusche, die an ganz umschriebenen
len, besonders oft am Ursprunge der Lungenarterie wahrnehmbar
l, die verschwinden oder kommen, wenn der Kranke seine Lage
ert, Geräusche, die nur in aufrechtem Stehen oder nur in der
en oder anderen Seitenlage gehört werden, Geräusche, die erst
n Aufdrücken des Stethoskopes zum Vorschein kommen, sind fast
Perikardialgeräusche.

Ganz auffallend häufig wird die Fehldiagnose „Mitralinsuffizienz"
nacht. Mehr als in der Hälfte der Fälle, in der dieser Klappen-
ler diagnostiziert worden ist, konnte ich in den letzten 8 Jahren
Bestimmtheit nachweisen, daß die Diagnose falsch war. — Es
cheint mir zweckmäßig, an dieser Stelle zunächst einige physiolo-
che Bemerkungen vorauszuschicken. Die Physiologie lehrt uns,
die Entleerung der linken Kammer durch eine Verkleinerung ihrer
hle im queren Durchmesser erfolgt, während der Längsdurchmesser
verändert bleibt. Die rechte Kammer entleert ihren Inhalt durch
rkürzung ihrer Länge und durch Heranziehung der Außenwand des
hten Ventrikels an die Kammerscheidewand. Als wesentlich mit-
rkender Mechanismus kommt hinzu, daß der Umfang der Herzbasis
d der Atrioventrikularöffnungen infolge von Kontraktion des von
ehl u. a. nachgewiesenen, das Ostium umgebenden Muskelwulstes
hohem Maße schmäler wird. Ein Umschlagen der Klappen in den
rhof und ein Aufblähen der Segel nach dem Vorhofe hin wird
rch die Sehnenfäden verhindert, welche sich nicht nur an den freien
ndern, sondern auch an den Seitenflächen der Klappensegel an-
zen. Die Vorhofsflächen der Klappensegel werden fast in ihrer
nzen Ausdehnung dicht aneinander gepreßt, und so kommt ein außer-
dentlich sicherer Verschluß der Ostien zustande, besonders des
ken. Erinnern wir uns ferner an die wunderbare Leistungsfähigkeit des
rzens, an seine Fähigkeit, sich sofort an die größten Anforderungen

1) So handelt es sich nach meiner Meinung bei der Angabe von Buttersack
iert nach Eichhorst 1904), daß 13,3% Schüler und 9% Rekruten im 2. linken
erkostalraume „perikarditisches Reibegeräusch" dargeboten hätten, sicher um
e Verwechslung mit dem an dieser Stelle besonders häufig hörbaren k. p. Ge-
sche.

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anzupassen — „das Herz ist der vollendetste Motor, den die Welt kennt", sagt Krehl so treffend —, erinnern wir uns daran, daß im Tierversuche die Füllungen des Herzens um das 6fache gesteigert werden können, ohne daß das Herz versagt, dann werden wir nicht glauben, daß geringfügige Störungen imstande sein können, diese wunderbare Anpassungsfähigkeit zu stören. Niemals ist es möglich, daß beim ganz gesunden Menschen die verstärkte Herztätigkeit den Schluß der Mitralis verhindern und eine vorübergehende Insuffizienz herbeiführen kann. Es ist auch ganz undenkbar, daß die leichte Mehrarbeit, welche einige Turnübungen verursachen, den Schluß der Klappen durchbrechen und die Klappen, wenn auch nur vorübergehend, schlußunfähig machen können. Wissen wir ja doch, daß das Herz in gleicher Weise die leichte Arbeit des Kreislaufs beim ruhenden Menschen besorgt wie bei demjenigen, der die schwerste Arbeit verrichtet.

Wir unterscheiden 3 Arten von Schlußunfähigkeit der Mitralklappen — die relative, die muskuläre und die endokarditische.

Unter relativer Insuffizienz versteht man die Schlußunfähigkeit der Klappen, die durch Dilatation des Insertionsringes der Klappen ohne irgendwelche organische Veränderung zustande kommt. Das Ostium wird mechanisch so weit gedehnt, daß die normal großen Klappen nicht mehr zur Deckung genügen, zum Verschluß nicht mehr ausreichen. Das Vorkommen solcher Insuffizienzen ist schon lange bekannt; sie sind aber verhältnismäßig selten, nur an der Trikuspidalis (bei Mitralklappenfehlern) und an der Aorta bei übermäßiger Dehnung des Bulbus der Aorta (Aneurysma) etwas häufiger.

Die relative Insuffizienz der Mitralis ist jedenfalls äußerst selten; ihr Vorkommen ist schon von Friedreich im Gefolge von Aorteninsuffizienz beobachtet und durch genaue Messungen festgestellt worden. Vielfach hat man auch angenommen, daß die systolischen Geräusche, welche bei hochgradiger Dilatation des linken Ventrikels, z. B. bei der idiopathischen Herzerweiterung, in der Spitzenregion gehört werden, durch Ausweitung des Mitralostiums entstehen. Diese Art von Geräuschen ist mir schon seit meiner Assistentenzeit bekannt. Seitdem ich gelernt habe, die akzidentellen Geräusche von den echten Insuffizienzgeräuschen zu unterscheiden, weiß ich, daß selbst bei der hochgradigsten Dilatation des linken Ventrikels eine relative Insuffizienz fast nie entsteht. Ein einfacher Lagewechsel, eine geringe Drehung des Rumpfes nach rechts genügt oft, das Geräusch sofort zum Verschwinden zu bringen, und neulich habe ich beispielsweise beobachtet, daß das vermeintliche Insuffizienzgeräusch kurze Zeit vor dem Tode verschwand, als ein linksseitiger Hydrothorax die Lunge vom stark erweiterten Herzen abgedrängt hatte. — Potain

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es durch experimentelle Untersuchungen am herausgenommenen rzen nach, daß die Trikuspidalklappen bei einer mäßigen Ausdehng des rechten Ventrikels insuffizient werden, während die Mitralppen mit merkwürdiger Ausdauer schließen, wenn der Druck im ken Ventrikel ganz bedeutend gesteigert und der Ventrikel ganz eblich dilatiert wird.

Von der relativen Insuffizienz, bei der das pathologisch erweiterte tium wegen seiner Größe nicht mehr von den Segeln geschlossen rden kann, ist als eine besondere Art der Schlußunfähigkeit des tralostiums zu trennen die „muskuläre". Wenn wir uns nochls vergegenwärtigen, daß für den prompten Verschluß der Mitralppe nicht nur eine Verengerung des Insertionsringes durch die gförmigen Muskelzüge, sondern auch eine normale Funktion der pillarmuskeln erforderlich ist, so erscheint das Vorkommen einer chen Insuffizienz als sehr verständlich. Ungenügende, mangelhafte ntraktion des Herzmuskels infolge ungenügender Speisung mit artellem Blute und Erkrankungen des Herzmuskels durch akute oder ronische Myokarditis werden vielfach als Ursache für die muskuläre uffizienz betrachtet. Besonders in Deutschland und England wird ch viel von „funktioneller" Insuffizienz der Mitralklappen gesprochen; n vielen Autoren werden die akzidentellen Geräusche bei Chlorose d Anämie so erklärt. Demgegenüber müssen wir nochmals hervorben, daß bei den Anämien das Vorkommen der Geräusche gar nicht hängig ist von dem Grade der Anämie, daß es ganz leichte Formen ot mit sehr starken Geräuschen und sehr schwere Anämien, wo die eräusche bis zum letalen Ausgange vollständig vermißt werden erniziöse Anämie). Unverständlich ist auch, warum diese muskuläre suffizienz so selten an der Trikuspidalis beobachtet wird, bei der kanntlich Schlußunfähigkeit ohne Veränderung an den Klappen viel ichter zustande kommt. Insuffizienz dieser Klappe wird nur dann ei tiefer Anämie beobachtet, wenn die Herzmuskulatur stark fettig generiert ist. In solchem Falle kommt es auch gewöhnlich zu einer uskulären Insuffizienz der Mitralis (siehe z. B. Fall 2). Nach meinem afürhalten kann die Bezeichnung „anämischer Klappenfehler" korkterweise nur für die Fälle schwerer Anämie, welche mit Herzverttung vergesellschaftet sind, gebraucht werden. Eine derartige („relative") muskuläre Insuffizienz bei hochgradiger Anämie mit Herzverttung ist schon von Friedreich (1861) beschrieben worden.

Auch bei der akuten und chronischen Myokarditis kommt nach einen Erfahrungen die muskuläre Insuffizienz viel seltener vor, als on manchen namhaften Autoren angenommen wird. Die meisten der ei diesen Herzmuskelerkrankungen beobachteten „Mitralgeräusche"

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können bei genauer Beachtung der von mir früher ausführlich geschilderten Merkmale der k. p. (akzidentellen) Geräusche als wirkliche valvuläre Geräusche ausgeschaltet werden. Doch ist das in diesen Fällen praktisch von geringer Bedeutung.

Sehr viel häufiger als die relative und muskuläre Insuffizienz der Mitralklappe ist die endokarditische; sie macht selbstverständlich die gleichen auskultatorischen Erscheinungen, die ich hier in Kürze nochmals wiederholen will. Die Folgeerscheinungen, welche der Ventilfehler, der allgemein als der häufigste Klappenfehler gilt, für das Herz und den Kreislauf hat, setze ich als bekannt voraus und gehe hier nicht näher auf dieselben ein. — Das Herzgeräusch bei Schlußunfähigkeit der Mitralis hat, da die Verschlußzeit fehlt, einen sehr bestimmten Charakter; das Geräusch fängt ganz genau mit der Systole an und dauert, da die Türe nach dem Vorhofe während der ganzen Systole offen bleibt, solange an wie die Systole. Das Geräusch hat seine maximale Intensität genau an der Herzspitze und verbreitet sich von da nach der Achselhöhle, ja bis zum Rücken. Das akzidentelle Geräusch an der Mitralis bleibt sehr oft ein kurzes Weilchen hinter dem Anfange der Systole zurück, es ist dem Tone angehängt, schleppt ihm nach und hat seinen Sitz viel mehr auf der Seite des Spitzenstoßes und verbreitet sich wenig oder gar nicht nach der Achselhöhle. Ein Ton kann an der Mitralis, wenn die Klappe teilweise zerstört ist und zum Schlusse nicht mehr ausreicht, nicht entstehen, denn Töne können an den Klappen, wie durch Bayer sichergestellt worden ist, nicht mehr entstehen, sobald sie anatomisch verändert sind. Die vielfach behauptete Annahme, daß der Rest der schwingenden Klappenmembran doch noch imstande sei, einen Ton zu produzieren, ist offenbar nicht richtig; der Ton, den man allerdings sehr oft bei einer unzweifelhaften Insuffizienz der Mitralklappen hört, ist nach meiner Überzeugung nichts anderes als der in den Arterien (während der Austreibungszeit) entstehende Gefäßton. Wer ein geübtes Ohr und ein feines Gefühl für Rhythmus hat, wird bei scharfer Aufmerksamkeit erkennen, daß der neben dem Geräusche hörbare Ton ein kurzes Weilchen nach dem Anfange des Geräusches auftritt. (Der zweite Ton, der bei Aorteninsuffizienz nicht selten neben dem diastolischen Geräusche gehört wird, ist nach meiner Meinung öfter nichts anderes als der 2. Pulmonalton.)

Eine besondere Besprechung verlangen noch die gar nicht seltenen Fälle, wo bei vermeintlicher Insuffizienz der Mitralklappe nicht nur an der Herzspitze, sondern auch in der Gegend der Pulmonalklappen ein Geräusch zu vernehmen ist. Oft unterscheidet sich in diesen Fällen das Schallphänomen seinem Klangcharakter nach ganz wesent-

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von demjenigen, welches an der Herzspitze gehört wird. Diese fallende Erscheinung ist schon lange bekannt. Jos. Meyer, Bamger, Skoda, Gerhardt haben das Geräusch fast jeder auf eigene ise zu erklären gesucht, auf unregelmäßige Schwingungen der dilaten und in ihrem Tonus veränderten Pulmonalarterie, auf beginde Sklerose zurückgeführt. Naunyn hat den Versuch gemacht, Geräusch, das nach seiner Angabe am lautesten etwa 2 Zoll vom ken Sternalrande („einem Orte, der nicht unerheblich differiert von n Punkte, wo in der Regel die von den Pulmonalklappen stammen- Geräusche gehört werden") zu vernehmen ist, so zu erklären, in solchem Falle das erweiterte linke Herzohr zwischen Pulmois und Brustwand sich eindrängt und die das Geräusch erzeugende tströmung in das Herzohr hineingeht. Diese Erklärung ist von rnherein schon deshalb nicht anzunehmen, weil — wie schon erhnt — das Geräusch an der Herzbasis häufig erheblich lauter, her und kratzender ist als das an der Spitze, weshalb an eine einhe Fortpflanzung gar nicht zu denken ist. Auch die von Curschnn gegebene Erklärung für das stärkere Auftreten des Geräusches der Hörstelle der Pulmonalis besonders im Beginne des Klappenlers, wo der linke noch nicht hypertrophierte Ventrikel und mit n die Herzspitze durch den überfüllten rechten Ventrikel von der ustwand abgedrängt werden, kann unmöglich richtig sein. Ich habe früheren Jahren in jedem Semester wenigstens einen derartigen ll, wo das Herzgeräusch besonders laut an der Hörstelle der Pulonalis oder sogar nur daselbst zu hören war — es sind meist jugendhe Individuen gewesen — mit der Diagnose Mitralisinsuffizienz in r Poliklinik vorgestellt. Seitdem ich gelernt habe, die kardiopulonalen Geräusche mit Sicherheit von den organischen zu differenren, ist mir kein derartiger Fall mehr vorgekommen. Schon Potain t das Naunynsche Geräusch mit Sicherheit als ein k. p. erklärt nd bestritt des bestimmtesten, daß es sich in diesen Fällen um einen lappenfehler handeln könne. Die Annahme von Lüthje, daß das eräusch an der Hörstelle der Pulmonalis, „das gerade bei einer chten Mitralisinsuffizienz besonders leicht auftreten wird", als das eräusch einer relativen Pulmonalstenose am ungezwungensten auffassen sei, haben wir als physikalisch unmöglich schon widerlegt. nzweifelhaft kann bei einer sichern Mitralinsuffizienz gelegentlich uch ein akzidentelles (k. p.) Geräusch an der Hörstelle der Pulmonalis um Vorschein kommen, aber meist handelt es sich — nach meiner Erahrung — in solchem Falle gar nicht um einen Mitralklappenehler.

Es wären noch manche Einzelheiten und verschiedene andere ge-

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legentliche Fehldiagnosen (Stenosen oder Insuffizienzen der Semilunarklappen usw.) zu besprechen, aber ich muß nun schließen.

Wenn es mir gelungen ist, das bisher so wenig beachtete, hervorragende und sehr verdienstvolle Werk Potains vor der Gefahr, vergessen zu werden, zu bewahren und wenn es mir selbst vergönnt war, etwas mehr Licht in ein noch dunkles Gebiet der Herzdiagnostik zu bringen, und wenn für den einen oder anderen Kollegen aus meinen Ausführungen ein Nutzen für das ärztliche Erkennen auf einem eminent wichtigen praktischen Gebiete erwächst, dann ist das Ziel dieses Vortrages erreicht.

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502/03.

Hämaturie.

(Chirurgie Nr. 147/48.)

Von

P. J. de Bruïne Ploos van Amstel,

Amsterdam.

Meine Praxis ermöglichte mir die Beobachtung mehrerer Patien-
, bei denen Hämaturie, die hauptsächliche Krankheitserschei-
ng war.

Ich glaube, daß das Resultat dieser Beobachtungen hinlänglich
eressant ist um dasselbe den geehrten Lesern zu unterbreiten,
on der Haupterscheinung „Hämaturie" wegen, welche hier so in
n Vordergrund trat, als wäre sie hier nicht ein Symptom, sondern
e Krankheit selber.

Diese speziellen Fälle sollen mir das Material liefern zu nach-
hender Besprechung, in welcher ich mich bemühen werde, die
ämaturie" in das Licht der heutigen Wissenschaft zu stellen.

1. Am 15. Dezember 1897 wurde ich zu der Patientin N. d. l. R. in O. gerufen.
s Resultat der Untersuchung war folgendes:

Anamnese. Bis Anfang November desselben Jahres war die 24jährige Frau
ner gesund gewesen, dann bemerkte sie plötzlich ohne jegliche Vorzeichen, daß
h Blut in ihrem Urin absonderte.

Anfänglich hatte sie keine Veranlassung, diesem Umstande besonderes Gewicht
zumessen, weil sie der Meinung war, daß diese blutige Absonderung durch die
nstruation verursacht wurde.

Als jedoch die Hämaturie fortdauerte, wurde ein Arzt konsultiert, der ihr Bett-
ne und Milchdiät verordnete.

Als nun am 14. November sich nicht nur die Hämaturie verschlimmerte, sondern
e Patientin auch sehr starke Nierenschmerzen bekam, wurde ein zweiter Arzt
Rate gezogen, der sie sofort an einen Spezialisten für zystoskopische Unter-
chungen verwies.

Dieser nun konstatierte folgendes: Die Blase war normal, das Blut stammte
s dem linken Ureter, und die Patientin empfand auch gerade die heftigsten
hmerzen in der linken Lendengegend.

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Die Urinuntersuchung ergab auch jetzt außer erheblichen Quantitäten Blut nichts Besonderes.

Auch die palpatorische Untersuchung hatte wenig Resultat: die Niere war weder vergrößert, noch beweglich oder schmerzhaft bei Druck.

Aus dem absolut negativen Resultat der Untersuchung des Urines und der Patientin erfolgte die Wahrscheinlichkeitsdiagnose auf essentielle Hämaturie.

Zwar wurde dem Gatten mitgeteilt, daß Nierenstein oder gar Nierentuberkulose in diesem Falle keinesfalls ausgeschlossen seien, wenn auch die Untersuchung in dieser Hinsicht nichts ergeben hatte.

Solange die Blutung als solche nicht gefährlich wurde, war die Therapie wie vorher: Ruhe und Milchdiät.

Die Hämaturie blieb jedoch trotz Ruhe und Diät bestehen und auch die Kolikschmerzen wiederholten sich, aus welchem Grunde die Patientin wiederum einen anderen Arzt konsultierte.

Auch eine wiederholte zystoskopische Untersuchung ergab beinahe das nämliche Resultat wie die vorige. Nur konnte als neues Symptom ein Nierentumor konstatiert werden. Der Patientin wurde der Rat erteilt, sich dieses Geschwulstes wegen in einem Krankenhause einer Operation zu unterziehen.

Dieser Vorschlag scheiterte jedoch an der Furcht der Patientin vor einer Operation, und so nahm die Behandlung ihren Fortgang: Milchdiät, Ruhe, guter Rat von guten Freunden und freundlichen Nachbarn, aber ... ohne ärztliche Hilfe.

Status praesens am 15. Dezember, als ich die Patientin zum ersten Male sah:

Patientin sehr anämisch, Lungen, Herz und Temperatur normal. Kein Ödem. Abdomen etwas schmerzend in der linken Nierengegend, wo gleichzeitig ein deutlicher Tumor fühlbar war.

Ich war daher zur Annahme geneigt, daß mein Kollege mit seiner jüngsten Diagnose das Richtige getroffen hatte, als mir der Umstand, daß wohl kaum anzunehmen sei, daß die Anwesenheit dieses Nierentumors bei allen früheren Untersuchungen übersehen worden wäre, zum Nachdenken zwang.

Ein derartiges schnelles Wachstum wäre selbst von einem malignen Tumor, beispielsweise einem Sarkom nicht zu erwarten, da dann ja in der Zeit von einigen Wochen dieser große Tumor sich müßte entwickelt haben.

Dann fiel mir ein, daß des öfteren schon Koprostase in den Cöcum oder Kolon für Nierentumor gehalten worden war, und überdies entsann ich mich des weisen Ausspruches von Jenner: „a large enema will solve all doubt on this point".

Und tatsächlich war nach einem Klysma und nach Anwendung von Oleum ricini der Nierentumor gänzlich verschwunden.

Die Untersuchung des Urins ergab folgendes Resultat: Eiweiß in geringen Quantitäten, hyaline, wie auch granulierte Zylinder, und rote und weiße Blutkörperchen. Die Reaktion des Urins war sauer. Die Anzahl der Zylinder bedeutend.

Bei der weiteren Behandlung: Ruhe, Milchdiät und Laxantia nahm die Anzahl aller dieser im Urin enthaltenen Fremdkörper ab.

Etwa Mitte Januar war der Urin sogar ganz ohne Blut und enthielt nur noch geringe Quantitäten Eiweiß und keine Zylinder, und einige Zeit später waren auch die letzte Eiweißreste verschwunden. Hierdurch war ein Beweis geliefert gegen die Behauptung Danforths[1]), daß die Anwesenheit granulierter Zylinder im Urin immer eine ungünstige Prognose zur Folge haben müsse.

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1) Danforth, The prognostic significance of Tube-casts. Medical News 1894.

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2. Ich sah Fräulein d. Hh. zum ersten Mal am 3. Februar 1906.

Anamnese. Patientin 16 Jahre alt, war stets wenn auch schwach so doch gesund esen. Sie hatte seit einiger Zeit Hämaturie, jedoch keine Nierenschmerzen. Die Hämaturie trat nicht immer gleich stark auf, hin und wieder war der Urin ar vollkommen blutfrei.

Status praesens. Patientin ist ein zartes anämisches Mädchen, und es waren ihr keine anderen Abweichungen zu konstatieren als Anämie und Schmerzen Druck an der linken Niere.

Die Urinuntersuchung hatte ein negatives Resultat bis auf die roten Blutperchen.

Patientin war nicht dazu zu bringen, sich einer zystoskopischen Untersuchung unterziehen.

Mit absoluter Gewißheit ergab sich indessen aus dem Charakter der Blutung, hier nicht von einer Blasenblutung die Rede sein konnte. Denn hier enthielt ht die letzte Quantität Urin das meiste Blut, welch letzteres ja nach Fürnger[1]) und Guyon[2]) das Hauptsymptom von Blasenblutungen ist, und auch lten hier jegliche Blasenbeschwerden. Auch der fernere Verlauf bewies, daß die ere, in casu die rechte, die Ursache der Hämaturie war. Einmal hatte sie im neren Verlauf Kolikschmerzen in der rechten Nierengegend.

Während der Dauer des ferneren Verlaufes der Krankheit, welche mit Ruhe d Diät behandelt wurde, bemerkte man bei wiederholter Urinuntersuchung, daß selbe nicht immer frei von Eiweiß blieb.

Dann und wann enthielt der Urin einige Tage lang Eiweiß. In dem jeweiligen eißhaltigen Urin wurden auch Zylinder gefunden; diese blieben zwar nicht ichzeitig mit dem Eiweiß aus, verschwanden aber doch auch bald wieder. Die ftigkeit der Hämaturie stand in keinerlei Zusammenhange mit dem Eiweiß- bzw. lindergehalt des Urins.

Nachdem dieser Zustand im ganzen 3 Monate gedauert hatte, verschwanden immer Hämaturie, Eiweiß und Zylinder, und die Patientin war wieder vollmmen gesund.

3. Herr N., 55 Jahr alt, kam in meine Behandlung am 25. April 1906.

Anamnese. Patient war nie krank gewesen, bis er vor ungefähr 7 Wochen ne blutige Färbung seines Urins konstatierte.

Da der Patient viel auf Reisen sein mußte, konsultierte er vorderhand keinen rzt, überdies verschwand die Hämaturie nach 2 Tagen.

Nach etwa 1 Woche wiederholte sich das Bluturinieren, diesmal schon intenver, aber auch nur während der Dauer weniger Tage.

So wiederholte sich nun die Hämaturie während einiger Wochen verschiedene ale, trotzdem wurde keine ärztliche Hilfe herbeigezogen und ließ sich Herr N. seiner geschäftlichen Tätigkeit nicht stören. Dann aber kam Nierenkolik hinzu, e Herrn N. veranlaßte, sich unter Behandlung eines Arztes zu stellen.

Status praesens. Patient sah sehr müde und anämisch aus. Die Untersuchung rgab keine Abweichungen der verschiedenen Organe. Die Nieren waren beide alpabel, doch nicht merkbar vergrößert und auch nicht schmerzend bei Druck.

Der Urin enthielt nur rote Blutkörperchen.

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1) Fürbringer, Die Krankheiten der Harn- und Geschlechtsorgane.

2) Guyon, Klinik der Krankheiten der Harnblase und Prostata. Bearbeitet on Mendelsohn.

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3 Tage nach der ersten Untersuchung bekam der Patient Fieber. Die Temperatur stieg abends bis 39,5, und blieb die folgenden Tage zwischen 38 und 39,7.

Außerdem enthielt der Urin nun große Quantitäten Zylinder und Eiweiß.

Die Menge des Urins nahm stark ab, und an den Füßen und im Gesicht zeigten sich deutlich wahrnehmbare, wenn auch schwache Ödeme.

Dieser Zustand dauerte verschiedene Tage, dann verschlimmerte sich das Befinden; es kamen urämische und komatöse Erscheinungen hinzu, und am 12. Mai erlag der Patient seinem Leiden.

4. Fräulein d. J. sah ich zum ersten Male im November 1906.

Anamnese. Patientin war bis vor etwa 10 Jahren stets gesund gewesen, fühlte sich kräftig, war unermüdlich. So blieb es bis 1896. Dann zeigte sich Blut im Urin, welches anfänglich für eine Folge unregelmäßiger Menstruation gehalten wurde; der hinzugezogene Arzt jedoch konstatierte Hämaturie. Während der Dauer zweier Jahre hielt die Hämaturie in abwechselnder Stärke an, bis im Jahre 1898 als neues Symptom ein frequentes Urinlassen hinzukam. Außerdem nahm der Urin eine trübe und schmutzige Farbe an, wo er bis dahin trotz des Blutgehaltes klar gewesen. In dem Satz des Urins konnte man hin und wieder blutige Substanzen konstatieren. Die früher so starke Hämaturie blieb indessen aus.

Dagegen wurden die Nierenschmerzen viel heftiger.

Während der Hämaturie klagte sie über dumpfe Schmerzen in der rechten Nierengegend; in diesem Stadium waren die Schmerzen kolikartig.

Die Urinuntersuchung, welche seit den ersten Krankheitsymptomen verschiedene Male vorgenommen wurde, sowohl während der Zeit der Hämaturie als auch nachher ergab außer dem Finden der roten Blutkörperchen, während der Zeit der Hämaturie, nie ein positives Resultat.

Jetzt aber enthielt der Urin Eiweiß, Zylinder, Epithelien und auch Tuberkelbazillen.

Eine zystoskopische Untersuchung ergab eine stark blutige Röte der Schleimhaut des rechten Uretereinganges. Dieselbe Röte wurde auch, zwar im geringeren Maße, dafür aber fleckenähnlich auf der Schleimhaut des Trigonums konstatiert.

Willy Meyer sagt von diesem letzteren Symptom: „Es sind Fußspuren im frischen Schnee", worauf er folgen läßt, „und man erkennt aus ihnen mit wundervoller Deutlichkeit die Fußstapfen des Feindes in einem bis dahin noch unbeschrittenen Feld."

Patientin wurde lokal auf Blasenleiden behandelt, doch ohne Erfolg. Später habe ich sie aus den Augen verloren.

Bei allen diesen vorgenannten Fällen handelte es sich um Hämaturie. Eine Hauptfrage ist nun: stammte das Blut aus den Nieren oder aus der Blase?

Zu ihrer Beantwortung bedarf es beinahe durchweg einer zystoskopischen Untersuchung, wenn auch hin und wieder die Anamnese ausreichendes Material für die Diagnose liefert.

Der Beweis dafür, daß der Arzt ohne zystoskopische Untersuchung leicht auf einen Irrweg geraten kann, liefert uns der bekannte Fall von Passet, der eines Tages die Entdeckung machte, daß bei einer Patientin, die er auf Blasenleiden behandelte, die Ursache der Hämaturie nicht in der Blase, sondern in den Nieren zu suchen sei.

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Auch das Ausspülen der Blase kommt als diagnostisches Hilfsttel in Betracht.

Ist für den Arzt einmal die Tatsache erwiesen, daß das Blut aus n Nieren stammt, so legt sich ihm die zweite Hauptfrage vor: elche der beiden Nieren ist als Ursache des Übels zu nunzieren?

Morris z. B. wäre die Beantwortung dieser Frage zustatte gekomen, als er in einem Falle von Hämaturie eine Niere entfernte, hrend beide die Blutung verursachten. Bei einer zystoskopischen tersuchung ist solch ein Fehlgriff natürlich ausgeschlossen. Hat n einmal konstatiert, daß beide Nieren, bzw. Ureter Blut absondern, nn ist es zweckmäßig, beide Ausflüsse für sich zu untersuchen. Zu esem Zwecke nennen Silbermann, Weir, Ebermann, Hegar, nwick u. a. verschiedene Instrumente und Methoden.

Simon aber war der erste, der uns den Weg für die Katheterierung beider Ureter zeigte. Nach ihm taten sich auf diesem Gebete noch andere, wie Pawlik, Howard, Kelly, Morris, Thompn, Boisseau, Caspar, Albarran, Berkeley, Hill, jedoch vor em Nitze hervor.

Es würde zu weit führen, hier alle die verschiedenen Instrumente beschreiben.

Trotzdem war man stetig auf der Suche nach anderen Mitteln, uptsächlich um eine Scheidung der Blase zu ermöglichen. Der ste, der dafür ein Instrument, „den Harnscheider" erfand, war Neuann aus Guben, nach ihm: Harris, Dowes, Nicolich, Luys[1]), athelin[2]). Erreicht man mit keiner dieser Methoden das erwünschte iel, so bleibt noch das von Iversen vorgeschlagene Öffnen der Blase pizystomie) mit anschließender Katheterisierung der Ureter.

Czerny und Braun empfehlen eine Nierenbeckenfistel, damit ur der Urin aus der anderen Niere in die Blase gelangen kann.

Dieses Verfahren vermag, besonders nach der Vervollkommnung on Pinner, entschieden Erfolge aufzuweisen.

Durch eine derartige Scheidung des Urins ist unbedingt die Herunft des Blutes erwiesen, d. h. welche der beiden Nieren erkrankt ist.

Natürlich erkennt man auf diese Weise auch die verhältnismäßig eltenen Fälle, bei denen nur eine Niere vorhanden ist.

Weiß man einmal, welche Niere erkrankt ist, so ist man doch och nicht berechtigt, dieselbe zu exstirpieren.

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1) Luys, Die Sonderung des Urins der beiden Nieren. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1902.

2) Cathelin, Les urines des deux reins receuillies séparément avec le diviseur vésical gradué. Annales des Maladies des Organes génito-urinaires 1902.

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Dazu bedarf es der Überzeugung, daß die andere Niere nicht nur relativ gesund, sondern auch imstande ist, die exstirpierte zu ersetzen.

Den Forschungen Prof. van 't Hoffs verdanken wir eine neue Wissenschaft, die „Kryoskopie", welche uns die Diagnose einer Niereninsuffizienz ermöglicht.

Wir wollen hier nicht näher auf die Technik und Lehre der Kryoskopie eingehen.

Um letztere, in ihrer heutigen Vollkommenheit haben sich Dreser, v. Koranyi, Lindemann, v. Illyes, Kövesi, Casper, Senator, Moritz, Kümmel u. a. verdient gemacht.

Kümmel sagt: Um die Insuffizienz der Niere zu erweisen, bedarf es

1. der Bestimmung des Harnstoffes;
2. der Bestimmung des Gefrierpunktes des Blutes;
3. der Bestimmung des Gefrierpunktes des Urins;
4. der Gefrierpunktsbestimmung des jeder einzelnen Niere durch den Uretherenkatheterismus entnommenen Urins.

Sein Ergebnis lautet:

1. Daß eine ungenügende Ausscheidung des Harnstoffes, ein Heruntergehen der Tagesmenge unter die Hälfte, ca. 16 g, die Annahme einer Niereninsuffizienz nahe legt und die eventuelle operative Entfernung einer Niere bedenklich erscheinen läßt.

2. Bei normal funktionierenden Nieren beträgt die Gefrierpunktserniedrigung des Blutes 0,56. Eine Niere mit normaler Arbeitsleistung reicht zur Erhaltung des Gefrierpunktes auf 0,56 aus. Eine Zunahme der Gefrierpunktserniedrigung auf 0,58—0,60 und darüber zeigt an, daß beide Nieren mangelhaft funktionieren. Von einem operativen Eingriff ist so lange Abstand zu nehmen, bis der Gefrierpunkt von annähernd 0,56 erreicht ist. Einseitige Krankheit bedingt keine Störung des Gefrierpunktes des Blutes.

3. Die Gefrierpunktserniedrigung des Urins unter 0,9 legt die Annahme einer Niereninsuffizienz nahe.

4. Einen weit sichereren Anhaltspunkt über die Funktionsfähigkeit jeder einzelnen Niere gibt die Untersuchung des jedem Organ gesondert durch den Ureterenkatheterismus entnommenen Urins auf Harnstoffmenge und vor allem Gefrierpunktserniedrigung.

In zweifelhaften Fällen wird man durch eine solche Untersuchung beinahe immer zu einer richtigen Diagnose gelangen.

Außerdem steht uns noch ein ungefährlicher operativer Eingriff, explorativer Lumbalschnitt, zwecks lokaler Untersuchung und ev. Nierenspaltung zur Verfügung.

1. Kümmel, Die Feststellung der Funktionsfähigkeit der Nieren vor operativen Eingriffen. Arch. f. klin. Chir. 1900. Über moderne Nierenchirurgie, ihre Diagnose

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. Resultate. Berliner klin. Wochenschr. 1906. Die Grenzen erfolgreicher Nieren-tirpation und die Diagnose der Nephritis nach kryoskopischen Erfahrungen. Arch. lin. Chir. Bd. 67.
2. v. Illyes und Kövesi, Der Verdünnungsversuch im Dienste der funktio-.en Nierendiagnostik. Berliner klin. Wochenschr. 1902.
3. Caspar und Richter, Über funktionelle Nierendiagnostik. Berliner klin. chenschr. 1900.
4. Richter, Neuere Fortschritte der Nierendiagnostik und ihre Bedeutung für Therapie. Die deutsche Klinik am Eingange des 20. Jahrhundert.
5. Casper und Richter, Was leistet die funktionelle Nierendiagnostik? teil. aus den Grenzgebieten usw. Bd. 11.
6. Achard et Castaigne, L'examen clinique des fonctions rénales.
7. Dreser, Über Diurese usw. Arch. f. exper. Path. u. Pharm. 1902.
8. Harris, A new and simple method of obtaining the urine separately from two kindneys in either sex. The Journ. of the Americ. med. Assoc. 1898.
9. Israel, Was leistet der Ureterenkatheterismus in der Nierenchirurgie? rliner klin. Wochenschr. 1899.
10. Senator, Weitere Beiträge zur Lehre vom osmotischen Druck tierischer ssigkeiten. Deutsch. med. Wochenschr. 1900.
11. v. Koranyi, Physiolog. und klin. Untersuchungen über den osmotischen uck tierischer Flüssigkeiten. Zeitschr. f. klin. Medizin 1896—1897.

Nierenblutung.

Sobald wir nun eine einseitige oder doppelte Nierenblutung kon-atiert haben, ist unser nächstes Ziel, die Ursache der Nierenerkran-ng zu erforschen.

Unser erster Gedanke ist dann die Existenz eines Nierentraumas. ne solche Nierenquetschung ruft beim Patienten fast immer einen ehr oder weniger starken Urindrang hervor. In schlimmen Fällen folgt dann sofort die Hämaturie, d. h. die Ausscheidung klaren Blutes; t die Quetschung weniger heftig, so mengt sich das Blut in der lase mit dem Urin und wird dann später als blutiger Urin ausge-chieden. Bei geringer Blutung und leerer Blase kommt es oft vor, aß das Blut in der Blase gerinnt und ihre Öffnung durch Koagula erschlossen wird. Hierdurch wird die Zystoskopie erheblich er-chwert, oft sogar unmöglich, aber die gleichmäßige Verteilung des lutes in der ausgeschiedenen Flüssigkeit berechtigt uns doch zur Dia-nose, daß hier nicht eine Blasen-, sondern eine Nierenblutung vorliegt.

Großen Wert für die Diagnose hat in diesem Falle die Anamnese, elche uns das etwaige Trauma deutlich erkennen läßt.

Menge[1]) macht uns aber noch besonders darauf aufmerksam, daß uch eine schwache Blutung die Folge eines leichten Traumas sein ann, und daß letzteres leicht übersehen wird.

1) Menge, Münch. med. Wochenschr. 1900.

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Er machte die Erfahrung, daß auch die vorherige Palpation eine leichte Nierenblutung zur Folge haben kann.

Er untersuchte in 21 Fällen den Urin vor und nach der Palpation und nur in 6 der 21 Fälle war der Urin nach der Palpation unverändert. In 15 Fällen enthielt er Blut und Eiweiß und zwar mehr Eiweiß als übereinstimmen konnte mit der anwesenden Quantität Blut.

Oftmals entsteht infolge des Traumas eine Nephritis, eine traumatische Nephritis, deren Schwere jedoch nicht immer im richtigen Verhältnis zu der Beschaffenheit des Traumas steht (Edlefsen[1]).

Die Möglichkeit ist also nicht ausgeschlossen, daß das Nierentrauma übersehen wird und unsere Diagnose der Hämaturie uns also auf falsche Fährte bringt. Dieses um so leichter, da häufig erst geraume Zeit nach dem Entstehen des Trauma die Hämaturie eintritt, während die Symptome des Trauma dann scheinbar so leichter Art sind, daß der Patient ohne Störung seinen täglichen Geschäften nachgeht.

So erzählt Morris[2]) uns einen Fall von einem Jüngling, welcher beim Fußballspiel einen Tritt in die Nierengegend erhielt, ruhig den ganzen Mittag weiterspielte und erst spät am Abend nach mehrmaligem Urinieren die Hämaturie entdeckte.

Auch kann es vorkommen, daß eine schon bestehende Hämaturie einem erst später entstandenen Trauma zugeschrieben wurde. Daß passierte in 1 Fall, über den Newman berichtet, von einem Jungen von 15 Jahren, „who after striking his side had persistent hæmaturia, which on inquity was found to have occured on previous occasions and to be due to a villous growth in the bladder".

Deshalb warnt auch Dr. Tiffany davor, in den Fällen, wo beide anwesend sind, Hämaturie und Trauma zu kombinieren. Er begründet seine Warnung durch reichliche Erfahrungen aus eigener Praxis in puncto Hämaturie in der Gegend zwischen Washington und den Capes of Cheasepeake. Die Anamnese ließ in den häufigsten Fällen auf irgendein früheres Trauma schließen; die Blutuntersuchung ergab als Ursache Malaria.

Wenn auch, wie wir gesehen haben, das Trauma bei Hämaturie eine große Rolle spielen kann, so kommt es doch bei der Besprechung sämtlicher Ursachen der Hämaturie bei weitem nicht in Betracht. So sah z. B. Küster[3]) kaum 10 Fälle von Nierenquetschungen bei

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1) Edlefsen, Nierenquetschung oder Nierenentzündung usw. Münch. med Wochenschr. 1902.

2) Morris, Surgical diseases of the kindney and ureter 1901.

3) Küster, Zur Entstehung der subkutanen Nierenzerreißungen und der Wanderniere. Deutsche Ges. f. Chir. 1895.

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00 Kranken in der Klinik in Basel; Morris in dem Middlessex spital kaum 13 Fälle bei 2610 Sektionen und Herzog kaum 17 den Sektionsprotokollen von 7805 Sektionen in dem Münchener nologisch-anatomischen Institut.

Wenn bei einem Trauma die Nierenblutung fortdauert, dann kann e Ursache nicht länger in dem Trauma selber sein, wohl aber e Nierenveränderung, welche als Folge des Traumas entstanden ist. Dieses beweist auch der Fall von Nimier.[1]) Der 17jährige Pa- t von Nimier hatte sich in der linken Nierengegend ein heftiges uma zugezogen. 8 Tage nachher entstand die Hämaturie, die nicht schwand und auch trotz vorbeigehender Besserung andauerte. Nach stirpation der Niere wurde der Urin normal blutfrei. Die Unter- hung der Niere ergab außer sklerotischen Veränderungen auch te Entzündungsherde.

Ren mobilis.

Die Hämaturie ist selten eine Begleiterscheinung bei Ren mobilis. Morris[2]) sagt hierüber: „Hæmaturia, in some cases to a rather gree, has been met with in association with movable kidney. I have plored the movable kidney in several instances, on account of pain d hæmaturia, and I have found it congested; and after the operation e symptoms have entirely ceased."

Derartige Fälle wurden wahrgenommen durch Albarran, Israel, ewman und Guyon.

Bei Guyon war es eine 38jährige Frau, die schon seit 2 Jahren inahe ohne Unterbrechung Nierenblutungen hatte. Sie unterzog sich ner Operation, die Niere wurde freigelegt und aufgeschnitten, aber s Resultat der Untersuchung war gering. Nur fand man, daß der bere Nierenpol stark fixiert war, der wurde nun losgelöst und die iere reponiert und zwar mit dem Erfolge, daß die Hämaturie aus- ieb. Ebenso ging es Albarran: auch er fand nichts an der Niere lbst, aber er erlangte Heilung durch Reposition der Niere.

Newman jedoch fand außer Ren mobilis auch Nephritis. Welche er beiden war nun die Ursache der Hämaturie?

Sehr richtig sagt darum Morris: „When hæmaturia occurs with a ovable kidney, lumbar exploration ought to be performed, and ephrorraphy practised, after full examination of the kidney and ownwards catheterisation of the ureter have shown that no other ngible cause for the hæmorrhage exists."

1) Nimier, Sur l'hématurie rénale. Soc. de Chir. 1898.
2) Morris, Surgical diseases of the kidney and ureter. 1901.

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Den Beweis für die Richtigkeit dieser Worte liefert uns der Fall von Kingston Fowler.[1])

Kingston Fowler behandelte eine Dame, welche wiederholt Anfälle von Nierenkolik und Hämaturie hatte. Die Untersuchung ergab außer dem Ren mobilis eine besondere tiefe Lage der Niere. Darauf wurde zur Operation geschritten. Dr. Gould, welcher dieselbe verrichtete, fand wohl einen Ren mobilis, aber derselbe verkehrte in einer „advanced stage of cystic degeneration".

Newman teilte in derselben Sitzung der Clinical Society zu London noch einige Fälle mit. Bei dem ersten Patienten von Newman[2]) zeigte sich: „Movable enlarged kidney with hyperæmia from torsion of renal vessels and ureter caused by strain. The symptoms were severe paroxysmal pain, hæmaturia, gastric disturbances, &c., simulating those of renal colic." Dieser Patient fand auf operativem Wege Heilung.

Auch Cabot[3]) erzählt uns von einem Fall von Hämaturie bei Ren mobilis.

Bemerkenswert war bei demselben der Umstand, daß die außergewöhnlich heftige Blutung sofort bei der Reponierung der Niere nachließ, um später beim Luxieren wieder mit alter Vehemenz einzutreten.

Anläßlich der Tatsache, daß Hämaturie oftmals geheilt wird durch Freilegen der Niere, glaubt Cabot, daß es sich in jenen Fällen um eine bewegliche Niere handelt. Die Inzision soll ein Adhäsion und dadurch eine Fixierung der Niere verursachen.

Die Niere des vierzehnten Patienten von Israel[4]) war auch abnormal beweglich und die bei diesem Patienten bestehende Hämaturie wurde auch geheilt durch Freilegen der Niere.

Israel sagt hierüber: „Daß die abnorme Mobilität der Niere in ursächlichem Zusammenhange mit der Blutung gestanden habe, ist möglich, da derartige Vorkommnisse bei Wandernieren schon beobachtet sind. Aber der Zusammenhang ist nicht bewiesen, da (hier) zwischen dem Schmerzanfall und Eintritt der Blutung 4—5 Wochen lagen und zur Zeit der Hämaturie jede Empfindlichkeit fehlte."

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1) Kingston Fowler, Clinical Soc. of London 11. December 1896. The British Medical Journ. 1896.

2) Newman, Increased vascular tension in the kidney a cause of renal pain, hæmaturia and albuminuria. The British Med. Journ. 1896.

3) Cabot, Severe hæmaturia from movable kidney. Boston Med. and Surg. Journ. 1902.

4) Israel, Über den Einfluß der Nierenspaltung auf akute und chronische Krankheitsprozesse des Nierenparenchyms. Mitt. aus den Grenzgebieten der Medizin und Chirurgie 1900.

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In unsern Fällen beweist die Untersuchung jedoch, daß es sich
t um Ren mobilis handelte.

Schon seit geraumer Zeit war es bekannt, daß Nierenblutung ein
ptom der Hämophilie sein kann, aber Senator[1]) war eigent-
der erste, welcher den Begriff „Renale Hämophilie“ erläuterte.
Er erörtert folgenden Fall: Im Februar 1890 wurde er von einer
ihrigen, an langwieriger Hämaturie leidenden Dame konsultiert.
on im Dezember 1887 war die Hämaturie konstatiert worden im
schluß an die Menstruation. Es bestand damals keine Anomalie
Beckenorgane. Im September 1889 wiederholten sich die Nieren-
tungen und hatten ununterbrochen stark fortgedauert, bis Senator
Rate gezogen wurde.

Die Untersuchung der kräftigen, wenn auch anämischen Patientin
ab keine Abweichungen der Lungen und der Nieren. Der Urin
hielt rote und weiße Blutkörperchen. Patientin war immer fieber-
gewesen. Ihr verhältnismäßig günstiges Befinden, sowie das
sultat der Urinuntersuchung veranlaßte Senator, die Anwesenheit
gewöhnlichen Ursachen der Nierenblutung, wie malignen Tumor,
berkulose und Nierenstein auszuschließen.

Aber auch die mehr seltenen Ursachen, wie Thrombose der Nieren-
äße, Aneurysma der Nierenarterien, Varix von Nierenvenen, para-
äre oder tropische Chlorose usw. ließ er unberücksichtigt. Seine
agnose lautete: Hämophilie, auf familiärer, hereditärer Grundlage
ruhende Neigung zu Blutungen, und zwar renale Hämophilie.

Die Anamnese bestätigte seine Diagnose (d. h. seiner Ansicht nach).
e Patientin hatte vier Schwestern, welche gleichwie der Vater an-
uernd von Nasenbluten geplagt wurden. Bei letzterem stellte sich
otz vollkommen gesunder Lunge des öfteren Hämoptöe ein. Die
roßmutter väterlicherseits, welche im 58. Lebensalter starb, hatte
s zu ihrem 57. Jahr heftige Menstrualblutungen gehabt. Patientin
lber war jedoch, außer obenerwähntem Falle, frei von Blutungen
ewesen.

Bei der zystoskopischen Untersuchung durch Dr. Nitze wurde kon-
atiert, daß es sich hier um Blutung der rechten Niere handelte. Das
efinden der Patientin wurde zusehends schlechter und so wurde die
iere auf operativem Wege entfernt.

Die exstirpierte Niere war, wie Senator uns mitteilt, makroskopisch
ganz normal, was Gestalt, Größe und sonstiges Aussehen betraf“.
licht nur der Wundverlauf war günstig, sondern auch die Hämaturie

1) Senator, Über renale Hämophilie. Berliner klin. Wochenschr. 1891.

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schien überwunden zu sein, so daß Patientin wieder völlig gesund wurde.

An einem Stück der Niere jedoch, das mikroskopisch untersucht ward, konstatierte man eine „leichte Verfettung einzelner Epithelien". Der andere Teil wurde von O. Israel untersucht, und dieser behauptete, daß mit Sicherheit festzustellen sei, daß „keine irgendwie ausgedehnte Nephritis bestand. Nur vereinzelte, sternförmige, ziemlich tief eingezogene Narben zeigten mikroskopisch das Bild einer begrenzten interstitiellen Nephritis".

Klemperer glaubt nicht, daß dieser Fall „renale Hämophilie" in dem Sinne Senators ist.

Erstens hatte diese Patientin vorher nie Blutungen, und Grandidier trifft doch das richtige mit seiner Behauptung, daß meistens bereits während der ersten zwei Lebensjahre „apparaissent les premiers symptômes hémophiliques; après 22 ans on en est presque toujours à l'abri".

Dieser Auffassung pflichtet Senator selber bei in dem Fall von Schede, wo er die Altersgrenze als Argument gegen die Annahme der Hämophilie als Ursache der Hämaturie anführt.

Auch die Anamnese, in welcher nur die Rede ist von Nasenblutungen und heftiger Menstruation, kann nicht als kräftiges Argument für die Diagnose in Betracht kommen.

Außerdem verlief die Zystoskopie ohne, und die Operation mit normaler Blutung.

Nun sagt Senator zwar, daß die Erfahrung bestätigt hat, daß gerade scharf geschnittene Wunden bei Hämophilen nicht so gefährlich sind und nicht so stark bluten, aber Grandidier beschreibt uns doch, wie in 40 Fällen der chirurgische Eingriff eine sehr starke Blutung nach sich zog. Heyfelder beobachtete sogar einst eine heftige Blutung nach der Vakzination.

Daß auch der von Senator mitgeteilte Fall, wo der Vater trotz gesunder Lungen Hämoptöe hatte, wenig Wert für uns hat, ergibt der Vortrag von Thorner in dem Verein für innere Medizin in Berlin.

Thorner behandelte einen ähnlichen Fall bei einer 36jährigen Dame; zuerst glaubte er an eine hysterische Blutung, jedoch der fernere Verlauf der Krankheit bewies, daß es ein Fall von Phthisis pulmonum war.

Auch bemerkt Gottschalk, daß Senators Patientin zyklische Albuminurie hatte, und das dürfte uns doch wohl veranlassen, einer anderen Ursache als der Hämophilie nachzuspüren.

Klemperer ist der Ansicht, daß Patientin auch auf Neurasthenie hin hätte behandelt werden müssen.

Senator selbst sagt jedoch hierüber: „Zyklische Albuminurie ist

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st ein Zeichen einer sehr leichten und schleichend verlaufenden
hritis."
Auch sagt er, daß letztere erst seit kurzer Zeit bestehen kann, da
vor der Operation keine Spur von ihr habe entdecken können:
h das ist kein Beweis.
Debove[1]) sagte bei der Besprechung eines Falles von Nephritis
Bleiintoxikation: „L'examen de l'urine ne révèle pas le moindre
e d'albumine, mais vous savez que pendant une période plus ou
ins longue de leur évolution, les néphrites peuvent exister, histo-
iquement tout au moins, sans s'accompagner d'albuminurie."
Der Umstand, daß Senator keine Spuren von Eiweiß entdeckte,
n doch kein Beweis dafür sein, daß während der Untersuchung
Nieren gänzlich normal waren, schließt doch nicht die Anwesen-
t von Nephritis aus. Übrigens ist auch, wie wir bereits erörterten,
Sektionsprotokoll von O. Israel schon die Rede von interstitieller
phritis.
Auch Henoch[2]), der den Namen zyklische Albuminurie unrichtig
det, sagt: „Wenn auch einzelne Fälle völliger Heilung beobachtet
rden sind, so ist doch die ganze Sache noch dunkel und der Ver-
cht einer schleichenden Nephritis nie ganz abzuweisen."
Heubner[3]) ist ebenfalls dieser Meinung.
Senator meint außerdem, daß zyklische Albuminurie nie solche
ftige Blutungen verursacht, wie gerade in diesem Falle, und ist der
einung, daß die Albuminurie aus der nicht exstirpierten Niere stammt,
d zwar infolge der Exstirpation.
Jedenfalls können wir nicht annehmen, daß Senators Fall ein hin-
lchender Beweis ist, um nur annähernd das Bestehen der renalen
ämophilie, nach seiner Auffassung, zu bestätigen.
Warum sollte auch das Wesen der betr. Hämophilie verschieden-
tig sein, nur weil zufällig das erste Symptom nicht eine Nieren-,
ndern z. B. eine Nasenblutung war?
Auch Klemperer findet diesen Fall von Senator durchaus nicht
weisend. Er spricht von „Hämaturie der Bluter", nie von renaler
ämophilie. Dies ist auch in der Tat die richtigste Auffassung, doch
durch wird auch Senators renale Hämophilie als selbständiges Krank-
eitsbild hinfällig.
Georg Elb[4]), ein Schüler Klemperers, hat in seiner Dissertation

1) Debove, Nephrite latente chez un saturnin. La Presse médicale 1901.
2) Henoch, Vorlesungen über Kinderkrankheiten.
3) Heubner, Pädiatrische Arbeiten. Festschrift. Berlin 1890.
4) Georg Elb, Zur Kenntnis der renalen Hämophilie. Inaugural-Dissertat.
erlin 1896.

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die renale Hämophilie erörtert. Er bespricht in derselben zwei Fälle aus der Klinik von v. Leyden, welche außerdem von Klemperer erörtert werden. In beiden Fällen bestand auch nicht der mindeste Zweifel darüber, daß die Patienten an Hämophilie litten. Neben anderen Blutungen wurde bei beiden Patienten auch Nierenblutung konstatiert.

Im völligen Einklange mit diesen zwei Fällen sagt Elb: „Unter renalen Hämophilie sind nur die Nierenblutungen notorischer Bluter zu verstehen."

Ein klassischer Fall von „Hämophilie der Bluter" ist durch Grosglik[1]) beschrieben. Auch dabei kommt Senators Fall zur Sprache und ist Grosglik ebenfalls der Ansicht, daß der Name „renale Hämophilie hier nicht richtig angewandt ist". „Die Hämophilie ist ja eine Allgemeinerkrankung; äußert sie sich aber ausschließlich in der Gestalt renaler Blutungen, so kann man eben nur von einer Nierenblutung auf hämophiler Grundlage reden."

Trotzdem bleibt für ihn das große Verdienst Senators, der darauf hingewiesen hat, daß bei den sog. essentiellen Nierenblutungen auch wohl Fälle vorkommen, wo die Blutungen auftreten bei Patienten, welche an Hämophilie leiden.

Grandidier[2]) hat auch einen ähnlichen Fall beschrieben, wobei ein 31jähriger Mann, der früher schon öfters an Nasen- und Zahnfleischblutungen litt, Anfälle von Hämaturie bekam.

Das Befremden, daß bei einer allgemeinen Krankheit wie der Hämophilie Exstirpation der Niere nicht nur Heilung der Nierenblutung, sondern auch dauernde Genesung der Hämophilie verursacht, beantwortete Senator mit der Behauptung, daß hier keine allgemeine Hämophilie bestand. Er sagt, daß es sich in diesem Falle nicht um sog. allgemeine Hämophilie gehandelt hat, sondern daß nur in der Niere, und zwar nur in der einen, Veränderungen vorgelegen haben müssen, auf Grund einer hämophilen Familiendisposition. Ferner sagt er auch, daß die wirkliche Hämophilie durchaus nicht immer eine allgemeine ist.

Außer den vielen Einsprüchen, welche schon gegen diesen Fall von Senator zu erheben sind, ist wohl das größte Bedenken dagegen, daß er ihn erklären will mit Hilfe einer besonderen Form der uns in Wesen und Ursache so wenig bekannten Krankheit Hämophilie.

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1) Grosglik, Über Blutungen aus anatomisch unveränderten Nieren. Sammlung klinischer Vorträge 203, Chirurgie 58.

2) Grandidier, Die Hämophilie oder die Bluterkrankheit.

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Ein sehr interessanter Fall über „renale Hämophilie“ wurde uns geteilt durch Passet[1]).

Die Patientin war Mutter von vier Kindern, deren ältestes 19 Jahre war. Die Diagnose lautete auf Blasenblutung. Das Ergebnis der mnese deutete nicht auf Hämophilie als Urheber der Krankheit

Die zystoskopische Untersuchung wurde unterlassen, nicht nur l die Patientin dieselbe nicht wünschte, sondern auch weil das ultat infolge der heftigen Blutung zweifelhaft gewesen wäre. Bei mikroskopischen Untersuchung konnte nur die Anwesenheit von t im Urin konstatiert werden. Die Blase wurde ausgespült mit er borsauren Lösung und dann wurde eine Injektion vorgenommen 100 g 1%iger Nitras-argenti-Lösung, welch letztere man eine iute auf die Blasenwand einwirken ließ. Scheinbar hatte diese Be-dlung den gewünschten Erfolg; jedenfalls ließ die Blasenblutung h. Auch jetzt noch verweigerte die Patientin die zystoskopische tersuchung. Nach $1^1/_2$ Jahren trat die Blutung wieder auf mit ganz ichartigen Symptomen. Jetzt aber blieb jede Behandlung resultat-. Die unter Narkose vorgenommene Digitaluntersuchung ergab nichts sonderes; nur wurde ein Stück Blasenschleimhaut gefunden in der öße eines Zweimarkstückes „von grobkörniger Beschaffenheit und leckt mit kurzen Trabekeln“. Endlich, nachdem die Blutung sechs ochen angehalten hatte, gab die Patientin ihre Zustimmung zur ctio alta, und nun wurde zum allgemeinen Erstaunen die Blase voll-mmen normal befunden, während die Katheterisierung der Ureter gab, daß die Blutung aus der rechten Niere herrührte. Die Nieren-eration, welche einige Tage später vorgenommen werden sollte, irde überflüssig, weil die Blutung aufhörte.

Passet erklärt uns, wie er zu einer falschen Diagnose gekommen . Guyon[2]) sagt: „Eine Vermehrung des Blutgehaltes des Harns am ade des Urinierens beweist stets mit Sicherheit, daß die Blase selbst e Quelle der Blutung ist.“

Auch Fürbringer sagt[3]): „Blasenblutung pflegt besonders blutreiche tzte Harnportionen zu liefern.“

Hier war dieses tatsächlich der Fall, so daß Passet annimmt, daß as Blut sich gewissermaßen gleich als Sediment im Urin absonderte, m somit in der Hauptsache erst mit der letzten Menge Urin abzu-

1) Passet, Über Hämaturie und renale Hämophilie. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1894.

2) Guyon, Klinik der Krankheiten der Harnblase und Prostata. Bearbeitet von Mendelsohn.

3) Fürbringer, Die Krankheiten der Harn- und Geschlechtsorgane.

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fließen. Der scheinbare Erfolg der Nitras-argenti-Therapie war die Ursache, daß Passet auf der falschen Fährte blieb. Erst nach der letzten Operation kam Passet zu der Diagnose Hämophilie. Er sagt: Nierenblutungen entstehen gewöhnlich infolge Nierentumor, Nierentuberkulose, Nierenstein oder Nephritis suppurativa. Gegen Nierentumor sprechen hier die folgenden Momente: die Abwesenheit einer palpabelen Geschwulst, keine Schmerzen und Kachexie und keine Veränderungen des Urins. Tuberkulose war ausgeschlossen, weil in dem Urin keine Tuberkelbazillen gefunden wurden, und wegen der langen Dauer der Blutung und des Charakters derselben, welche bei Tuberkulose nicht so profus zu sein pflegt. Die Argumente gegen Nierentuberkulose waren nicht sehr stichhaltig, denn es ist eine irrige Auffassung, daß die Blutung bei Nierentuberkulose weder von langer Dauer, noch profus sein kann. Nierenstein kam für ihn nicht in Betracht, da die Patientin nie Nierenstein oder Sand ausgeschieden hatte, und wegen der Abwesenheit von Kolik und Fieber. Auch diese Beweisführung ist nicht ganz richtig, denn wir wissen, daß Nierensteine tatsächlich Blutungen verursachen können ohne Fieber und Kolik, und außerdem erlaubt das Gestern in bezug auf das Ausscheiden der Nierensteine keinerlei Schlußfolgerung auf das Heute. Der Allgemeinzustand, d. h. der fieberfreie Zustand, der Patientin spricht sehr gewichtig gegen Nephritis suppurativa. Demnach blieb Passet nichts anderes übrig als anzunehmen, daß Hämophilie die Ursache des Leidens war. Zum Durchführen dieser Diagnose bei der Mutter einer 19jährigen Tochter (es hatte sich früher nie Hämophilie bei ihr gezeigt) bedurfte Passet eines neuen Begriffes: der relativen Hämophilie. Unter relativer Hämophilie versteht Passet einen Zustand, bei welchem hämophile Erscheinungen an dem Individuum oder in der Familie bis zum Auftreten der Nierenblutung vorher nicht beobachtet wurden. Dieser Begriff kleidet allerdings den Fall in das Gewand der Hämophilie, aber der Begriff Hämophilie wechselt dadurch seine Bedeutung. Außerdem vergaß Passet aber, daß nicht nur die Nephritis suppurativa, sondern auch andere Arten der Nephritis Ursache der Hämaturie sein können, und somit wird sein Motiv (exclusionum) für die Diagnose: renale Hämophilie hinfällig.

Nierensteine.

Nierenblutung ist eine der häufigsten Erscheinungen bei Nierenstein. Die Diagnose ist aber meistens schwierig. Zwei Fälle von Brook[1]) zeigen uns die Schwierigkeit der Diagnose; dieselben sind von

1) Brook, Two cases of nephrolothotomy illustrating the value of heavy

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er Wichtigkeit, weil Brook uns das diagnostische Mittel nennt zur ıng dieser Schwierigkeit. Die erste Patientin war ein 18jähriges chen, welches dem Swansea-Hospital überwiesen wurde, weil sie n seit 5 Jahren klagte über Schmerzen: „in the left iliac region". rst kamen die Schmerzen immer gleichzeitig mit der Menstruation, während der letzten 3 Jahre zu allen Zeiten. Die Unter- ung ergab Druckschmerzen des linken Ovariums. Der Urin zeigte e besonderen Merkmale. Bei der Operation fand man eine Ova- yste, welche entfernt wurde. Nach einer Periode von 2 Monaten en die Schmerzen wieder auf.

„The abdominal pain was now more general, but was most severe ut a point midway between the umbilicus and the anterior supe- spine. It extented into the left flank, and deep pressure over the ey caused pain, but the seat of greatest tenderness corresponded hat of the greatest pain."

Alle klassischen Symptome von Nierensteinen fehlten hier und doch gnostizierte McBurney Nierenstein, weil: „Heavy percussion w the tip of the last rib caused acute stabbing pain in the loin." Der zweite Patient, ein 22jähriger Mann, kam in das Krankenhaus gen Appendizitis. Auch hier zeigte der Urin keine besonderen rkmale. Die Schmerzen dauerten schon 3 Jahre hindurch, hatten auch nicht im Ruhestand, nachgelassen, verschlimmerten sich aber jeder Anstrengung. The pain, which was situated in the right c region, was most intense midway between the umbilicus and the erior superior spine. Die Schmerzen waren also genau dieselben in dem vorhergehenden Falle, nur traten sie gerade auf der ent- gengesetzten Seite auf. Die rechte Niere war gar nicht oder doch sehr wenig schmerzhaft bei Druck, but heavy percussion imme- tely over the kidney caused stabbing pain in the loin. In beiden llen bestätigte die Operation die Richtigkeit der Diagnose. Das wicht der entfernten Steine betrug 123 bzw. 80 g, und nach der eration waren beide Patienten endgültig kuriert.

Jacobson erörtert in seinem Werke über „Operative Surgery" ses diagnostische Hilfsmittel, aber Jordan Lloyd war derjenige, lcher es in das Licht der allgemeinen Aufmerksamkeit lenkte.

Bei meinen vier Fällen kam es nicht in Betracht, aber da, wo es ch wohl um Nierenstein handelte, habe ich den großen diagnosti- hen Wert kennen gelernt, und so wundert es mich, daß nur Brook

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rcussion in the loin in the diagnosis of renal calculus. The British Medical urnal 1896.

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und Schede[1]) es in der Literatur berührt haben. Hätte Israel[2]) das Hilfsmittel gekannt, so hätte er vielleicht nicht gesagt: „Aber die Sicherheit der Diagnose fehlt zunächst noch bei dem Nierenstein.“ Allerdings die absolute Gewißheit bringt nur die Palpation des Nierensteins, und dazu ist eigentlich nur Gelegenheit vorhanden bei bloßgelegter Niere. Wenn es auch feststeht, daß bei einem Patienten ‚früher‘ Nierenstein entfernt wurde, so ist das noch kein Beweis dafür, daß die Schmerzen ‚jetzt‘ auch auf Nierenstein zurückzuführen sind. Wie wenig verläßlich die Kolik ist für die Diagnose, ersehen wir aus einem Fall, den uns Israel berichtet, bei welchem unter Voraussetzung des Vorhandenseins von Nierenstein Inzision der Niere stattfand, aber ohne Resultat. Später ergab sich, daß es Lumbalneuralgie war, verursacht durch den Anfang einer Spondylitis eines Brustwirbels.

Auch die Urinuntersuchung kann uns im Stiche lassen, ebenso die Art der beobachteten Hämaturie. Zwar behauptet man, daß starke Blutungen darauf schließen lassen, daß es sich nicht um Nierenstein, sondern um einen Tumor handelt, aber es hat auch schon Fälle von Nierenstein gegeben, deren Hauptsymptome profuse Blutungen waren. Ebenso kann ich der Meinung nicht beipflichten, daß Ruhe oder Bewegung des Patienten einen direkten Einfluß auf die Stärke der Blutung hat. Auch das Vorhandensein etwaiger Kristalle im Urin oder das Fehlen derselben ist kein Anhaltspunkt.

Israel will deshalb in zweifelhaften Fällen die Niere bloßlegen und hält diese Operation, von geübter Hand verrichtet, für durchaus ungefährlich. Ist auch dieses noch nicht hinreichend, so empfiehlt er die Akupunktur. Wenn dieselbe auch nicht zum gewünschten Ziele führt, so bleibt noch als letztes Mittel die Öffnung des Nierenbeckens oder die Nierenspaltung. Die Nierenspaltung ist vorzuziehen, weil Nierenwunden weniger gefährlich sind und schneller heilen als Wunden des Nierenbeckens.

Nach Entfernung des Steines hat man die Wahl, entweder die Heilung der Nierenwunde durch Nähen zu beschleunigen, oder dieselbe ganz der Natur zu überlassen. Die erste Art hat den Nachteil, daß die dabei abgesonderten Blutkoagula oft unter heftigen Schmerzen durch den Ureter ausgeschieden werden müssen. Dafür wird aber die Wunde schneller gestillt und bleibt die Wunde urinfrei. Tamponade empfiehlt Israel nicht. Er berichtet sogar einen Fall mit tödlichem Ausgang, bei welchem ein nicht vorhandener Nierenstein durch Nieren-

1) Schede, Die Steinkrankheit der Harnwege und die Nierensteine. Handbuch der praktischen Chirurgie von v. Bergmann, v. Bruns und v. Mikulicz. 1903.

2) J. Israel, Über Operation und Diagnose der Nierensteine. Berliner klin. Wochenschr. 1891.

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ung entfernt werden sollte. Bei der Sektion, welche 2 Stunden Eintreten des Todes vorgenommen wurde, „zeigte sich eine e primäre Verklebung der Nierenwunde". Der Tod war eingen unter Symptomen von Darmokklusion; durch den doppelten k der Tamponade und des Druckverbandes war der Ileus entden, welcher dem Patienten verhängnisvoll wurde.

eder weiß zur Genüge aus seiner eigenen Praxis, daß solche felhafte Fälle oft vorkommen. Israel[1]) sagt hierzu: „Die landge Meinung, daß das Steinleiden auf Grund seiner prägnanten oxysmen am leichtesten unter allen Nierenkrankheiten zu erkennen entspringt einer ungenügenden Erfahrung." Wenn man dem Umde Rechnung trägt, daß nur in sehr wenig Fällen der Stein mittels tgenstrahlen gefunden werden kann oder palpabel ist, „so kann die beste Diagnose nur auf ein hohes Maß von Wahrscheinlich-Anspruch machen". Er begründet diesen Anspruch mit der großen der Nierenoperationen, verrichtet von gewiegten Chirurgen, bei chen anstatt der erwarteten Nierensteine andere Krankheitsprozesse nden wurden.

Ebensowenig wie die Hämaturie ein sicheres Kennzeichen für die gnose ist, ebensowenig, sagt Israel, bedingt sie oft die Operation, . wenn man dabei ausschließlich urteilt nach der Vehemenz der tungen. Die Diagnose auf Nierenstein wird in vielen Fällen noch urch erschwert, daß der Zweifel bestehen bleibt, ob es sich nicht leicht um Tuberkulose oder einen Tumor handelt. Deshalb sagt ael: Wenn die Diagnose nicht absolut feststeht, „dürfen wir uns t bei der Vermutung eines Nierensteines oder gar der Verlegentsdiagnose einer essentiellen Hämaturie beruhigen, sondern müssen ch eine Explorativoperation feststellen, ob ein bösartiges Leiden runde liegt, von dessen frühzeitiger Entdeckung die Zukunft des nken abhängt".

Auch ein Fall von Pepper[2]) zeigt uns die große Schwierigkeit der rensteindiagnose. Sein Patient hatte Anfälle von heftiger Steinik, jedoch ohne Blutabsonderung. Auch Hämaturie trat zuweilen , aber schmerzfrei. Pepper sagt hierzu: „In the first case the scence of hæmaturia is remarkable considering the frequency of attacks of pain and their violently paroxysmal nature. This seems show us that the pain is caused more by distention of the kidney

1) J. Israel, Operationen bei Nieren- und Uretersteinen. Arch. f. klin. Chir. 0.

2) Pepper, Two cases of nephro-lithotomy. Recovery. Remarks. The Lancet 9.

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owing to the ureteral office being temporarily blocked by the calculus, than to the movements of the latter."

Das Gegenstück hierzu ist ein Fall von Schede. Der Patient starb infolge tödlicher Nierenblutung, und diese war dadurch verursacht, daß ein Stein im Nierenbecken einen Hauptast der Arteria renalis durchbohrt hatte.

Auch Rovsing[1]) empfiehlt eine Explorativoperation für alle jene Fälle, bei denen eine exakte Diagnose nicht zu stellen ist. Er bekämpft entschieden die Behauptung Klemperers, daß eine Operation unterbleiben muß, wenn z. B. die Ursachen einer Hämaturie unbekannt sind. Klemperer steht auf dem Standpunkte der essentiellen Hämaturie, d. h. er glaubt an Blutungen gesunder Nieren. Rovsing dagegen behauptet, daß bei Hämaturie ein negatives Resultat der Untersuchung durchaus nicht heißen soll: Nierenstein, Tumor oder Tuberkulose sind ausgeschlossen. Er sagt: „Da solche Fälle (Blutungen aus gesunden Nieren) so selten sind, daß sie als reine Ausnahmen bezeichnet werden müssen, und da ernste Leiden nicht ausgeschlossen werden können, muß stets bei einer unilateralen unerklärlichen Hämaturie ein explorativer Lumbalschnitt gemacht werden, und zwar um so mehr, als ein solcher erfahrungsgemäß heilend auf die Hämaturie einwirkt."

Zweifelt man daran, daß es sich um Nierenstein handelt, so kann man auch eine Sondierung der Ureter von der Blase aus vornehmen. Dieses ist nach den Verfahren von Kelly und Pawlik bei Frauen weder schwierig, noch gefährlich. Man kann sich dabei sogar einer metallenen Sonde bedienen. Bei Männern ist letzteres nicht möglich; dazu bedarf es einer elastischen Sonde, aber auch damit hat Albarran günstige Resultate erzielt. Diese Art der Untersuchung aber ist schwierig und nicht ohne Gefahr, weil dabei leicht eine Infektion stattfinden kann. Musser[2]): „I feel with Holländer, we can in a large number of cases make a diagnosis without the aid of catheterisation and I appreciate with him the danger of infection from below—of which he cites cases."

Auch Morris[3]) ist der Meinung, daß diese Methode selten angewendet werden wird. Er nennt sie: „difficult of application in the male, not altogether free from danger and not always to be relied upon as a result". Auch die Radiographie wurde in der letzten Zeit ein Hilfsmittel für die Diagnostizierung der Nierensteine. Ringel aus Hamburg war der erste, dem es gelang, deutliche Bilder der Nieren-

1) Rovsing, Über unilaterale Hämaturien zweifelhaften Ursprungs und ihre Heilung durch Nephrotomie. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1898.

2) Musser, Renal calculus. 1898.

3) Morris, Surgical diseases of the kidney and ureter. 1901.

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ie zu erzielen. Ihm folgten Wagner und Leonard. Morris be-
tet die Behauptung des letzteren, man müsse unter allen Umständen
Hilfe der Radiographie zu der richtigen Diagnose gelangen. Morris
von den Röntgenstrahlen: „Hitherto they have afforded but little
ble help in this direction." Tüchtige Forscher, sagt er, haben
tten entdeckt von Steinen, die in Wirklichkeit nicht existierten,
umgekehrt.

Auch in späteren Schriften bleibt Leonard[1]) seiner Überzeugung
. Er sagt: „The greatest value of the Röntgen ray in physical
nosis is not in penetrating all tissues but in producing differen-
ons between tissues that will lead to logical conclusions and ab-
te diagnosis. Thus in examining for calculi we do not want rays
will penetrate all tissues; we want a differentiation in the shadows
will demonstrate beyond a doubt that all calculi, no matter what
r relative density, would cast shadows if present in the field exa-
ed. Such definition and differentiation make the negative diagnosis
olute. The object, therefore, when examining for renal calculi by
method of diagnosis, is to obtain negatives in which the shadows
shown of tissues less opaque than the least opaque calculus.
ere such definition is obtained it is certain that not even the smallest
ulus can escape detection, no matter what its composition is."

Nähere Einzelheiten zu dieser Untersuchung, welche hier nicht
er erörtert werden soll, können wir u. m. finden in den Fort-
ritten auf dem Gebiete der Röntgenstrahlen, Bd. 3, 1900, von
ers-Schönberg, Levy-Dorn, Wagner, Lauenstein und Levy;
Zentralblatt für Chirurgie von Ringel[2]), im Lehrbuch der Röntgen-
ersuchung von Gocht, in den Annales des Maladies génito-urinaires,
2, von Verhoogen (Le diagnostic des calculs du rein par la radio-
phie) usw.

1) Leonard, The Roentgen Ray Diagnosis of Renal Calculus. Annals of Surgery
).

Nierentuberkulose.

Bei Nierentuberkulose kommen häufig Blutungen vor. Beinahe
ner ist das erste Symptom Polyurie ohne Veränderung des Urins
uyon). Aber an Stelle der Polyurie als ersten Symptoms können

1) Leonard, The Röntgen Method in the diagnosis of renal and ureteral
culi. Medical News 1902.

2) Ringel, Diagnose der Nephrolithiasis durch Röntgenbilder. Zentralbl. f.
r. 1898.

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Blutungen treten. Schede[1]) sagt hierzu: „Wir sahen schon, daß Blutungen bei der Nierentuberkulose zuweilen sehr früh und sehr heftig auftreten können. Nicht selten sind sie das allererste Symptom der Krankheit und nicht selten findet man die Nierenerkrankung dann noch außerordentlich gering."

Diese Blutungen können so schlimm sein, daß sie bereits im allerersten Stadium der Krankheit einen chirurgischen Eingriff notwendig machen. Albarran nahm zwei Nierenoperationen bei Hämaturie vor, nachdem auf zystoskopischem Wege konstatiert worden war, welcher Niere die Blutungen entstammten. In beiden Fällen war die Niere scheinbar makroskopisch normal, und sogar nach dem Sektionsschnitt war es äußerst schwierig, die vorhandenen wenigen Tuberkel anzudeuten.

Loumeau[2]) berichtet einen Fall eines 27jährigen Mädchens, welches an hartnäckiger, keiner Behandlung weichender Hämaturie litt. Die Blutungen hielten bereits seit 8 Monaten an. Er verrichtete die Exstirpation und fand in der makroskopisch normalen Niere nur vier winzig kleine Tuberkel.

Wir ersehen hieraus, daß die Heftigkeit der Hämaturie oft in keinem Verhältnisse steht zu der Ausdehnung des Tuberkuloseprozesses. So können auch umgekehrt in den Nieren bedeutende Läsionen vor sich gehen ohne Hämaturie. Dieses ist übrigens nicht nur der Fall bei der Hämaturie, sondern auch bei vielen anderen Symptomen der Nierentuberkulose. Tilden Brown[3]) beschreibt uns einen Fall, bei welchem nicht ein einziges Symptom auf Nierentuberkulose deutete, während tatsächlich beide Nieren erkrankt waren. Auch Morris spricht von der Möglichkeit, daß alle Symptome: „are entirely absent or but little marked". Auch erörtert er sehr richtig und mit Nachdruck: „the difficulty of diagnosis". Gleicher Ansicht wie Morris ist Kinghorn[4]).

Weitere Erörterungen der Hämaturie finden wir noch bei: Fagge: „Hæmaturia is neither constant nor profuse." Bouman[5]): Charakteristischen Wert haben die durch Tuberkulose erweckten Blutungen nicht." Morris: „The hæmaturia is in many cases intermittent and quite independent of rest or exercise."

1) Schede, Die Tuberkulose der Niere. Handbuch der praktischen Chirurgie v. Bergmann, v. Bruns und v. Mikulicz. 3. Bd. 1903.

2) Loumeau, Nephrektomie wegen primärer Tuberkulose. 21. Congrès de Chirurgie. La Presse médicale. Ref. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1898.

3) Tilden Brown, Tuberculose rénale. Annales des maladies des organes génito-urinaires 1898.

4) Kinghorn, Renal tuberculosis. Montreal Medical Journal 1901.

5) N. W. Bouman, Haematuria in graviditate. 1901.

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Und doch sind auch diese Merkmale nicht entscheidend für die gnose, weil es auch bei anderen Nierenaffektionen vorkommen kann, Ruhe oder Bewegung keinen Einfluß ausüben auf den Grad der naturie. Schede sagt auch: „Weder Blutungen, noch Schmerzen, h Vergrößerungen der Niere haben etwas absolut Charakteristisches; selbst der röntgengraphische Nachweis von Steinen in der Niere ließt nicht aus, daß sich zur Tuberkulose sekundär Steinbildung ellt hat, oder umgekehrt. Das einzige, dem Schede großen Wert mißt, ist die lokale Reaktion auf Tuberkulininjektionen, weil die- be beim Fehlen der Tuberkulose ausbleibt. Schede warnt vor Über- ätzung der Schwierigkeit, zu einer richtigen Diagnose zu gelangen: ür unser therapeutisches Handeln ist diese, oft nicht mit voller herheit zu lösende Schwierigkeit der Differentialdiagnose glück- nerweise nicht so wichtig. Wenn wir wissen, daß es sich um Eiter- er Steinniere, um Nierentuberkulose oder Neoplasma handeln kann, erwächst uns unter allen Umständen die Pflicht, die Niere bloß- egen und uns eventuell durch den Sektionschnitt die nötige Sicher- t zu verschaffen."

Wie wir weiter oben sahen, ist dieses auch die Ansicht Israels. Glaubt man die Diagnose „Nierentuberkulose" stellen zu dürfen, versteht es sich von selber, daß man wiederholt den Urin auf berkelbazillen untersucht. Häufig aber wird man diese letzteren, gar bei weit fortgeschrittener Nierentuberkulose, nicht vorfinden, und ßerdem zeigt van Leyden[1]) uns die gefährlichen Irrtümer, welche ese Untersuchung nach sich ziehen kann. Sehr häufig werden nämlich e Smegmabazillen, welche öfters mit dem Urin ausgeschieden werden d in bezug auf Form und Farbenreaktion den Tuberkelbazillen sehr nlich sind, für Tuberkelbazillen gehalten. Der Smegmabazillus ist ar schlanker und kommt nur selten gruppenweise vor, aber diese nterschiede sind meistens nicht ausreichend, um mit Bestimmtheit en Bazillus zu definieren. In solchen Fällen ergibt natürlich nur die ierinokulation ein entscheidendes Resultat.

Auch Tuffier[2]) wurde durch starke Hämaturie bei Nierentuberkulose eranlaßt, Nephrotomie und Nephrektomie zu verrichten. In beiden ällen trat Heilung ein nach der Operation, aber bei der Patientin, n welcher Nephrotomie verrichtet wurde, zeigte sich nach 2 Jahren ieder Blut im Urin.

1) van Leyden, Über die Diagnose der Nierentuberkulose. Berliner klin. Wochenschr. 1896.

2) Tuffier, Die Ergebnisse von 153 Nierenoperationen. Vortrag gehalten auf em 12. internationalen Kongreß zu Moskau. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1898.

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Paul Rosenstein hält in den Fällen, bei welchen Zystoskopie ausgeschlossen ist, die Palpation der Ureter für notwendig für die Bestätigung der Diagnose „Nierentuberkulose". Bei dem Kongreß der Deutschen Gesellschaft für Chirurgie (6.—9. April 1904) äußerte er die Ansicht, daß der Ureter immer vom Anfang der Krankheit an alteriert ist. Mit Hilfe der Vaginal- und der Rektalpalpation kann man also konstatieren, welche der beiden Nieren erkrankt ist, und braucht also nicht die Ureterkatheterisation vorzunehmen, welch letztere übrigens bei tuberkulöser Blase erhebliche Schwierigkeiten mit sich bringt.

Hämaturie und Nierentumor.

Nierenblutungen treten häufig bei Nierentumor auf und sind gewissermaßen deren erstes Symptom. Chevalier[1]) kam zu dem Resultat, daß dieses bei 26,6% aller von ihm beobachteten Fälle zutraf.

Der Urin ist dann rot oder, wenn das Blut bereits einige Zeit in der Blase gestanden hat, bräunlich gefärbt. Israel nennt weiße oder hellgelbe und rötliche wurmförmige Koagula charakteristische Merkmale des Nierentumors.

Die Hämaturie kann aber auch während des ganzen Krankheitsprozesses ausbleiben. Wagner[2]) berichtet einen Fall von Nierensarkom: „Hämaturie fehlte während des ganzen Krankheitsverlaufes." In diesem Fall, sagt Wagner, fehlten alle vier Hauptsymptome des Nierentumors (wahrnehmbare Geschwulst, Schmerzen, Hämaturie und Kachexie) bis auf die Schmerzen. Bei Erwachsenen bilden die Schmerzen sogar in 28% der Fälle das Initialsymptom.

Doch kommt es vor, daß gewissermaßen alle Symptome sich gar nicht oder nur sehr schwach zeigen. Zuweilen ergibt die Urinuntersuchung ein positives Resultat, indem man Tumorzellen findet, aber auch dieses Symptom kann fehlen. So berichtete Pinard in der Société d'anatomie et de physiologie in Bordeaux im Jahre 1897 einen Fall von Carcinoma renis. Der Tumor hatte die Größe eines Kinderkopfes, und hatte doch nie Urinbeschwerden oder Hämaturie veranlaßt.

Gerade so fehlten diese Beschwerden und die Hämaturie in einem gleichartigen Fall, den Taboulay berichtet (Société des sciences médicales, Lyon 1898). Auch hier konnte der Tumor ausschließlich durch Palpation konstatiert werden. Gleichartiges beschreibt auch Maidlow[3]).

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1) Chevalier, De l'intervention chirurgicale dans les tumeurs malignes du rein. Thèse Paris 1891.

2) Wagner, Zur Kasuistik des primären Nierensarkoms. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1894.

3) Maidlow, A case of exstirpation of the kindney for sarcoma. The British Medical Journal 1898.

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Denaclara[1]) konstatiert, daß bei 68,88 von hundert Fällen die maturie das Initialsymptom des Nierentumors ist.

Aber auch wenn dem so ist, so muß man doch zugeben, daß die maturie bei Nierentumor keinen charakteristischen Wert hat. Zwar fert Guillet uns sieben Merkmale dieser Hämaturie, aber bei näherer trachtung sehen wir, daß sie uns für die Diagnose wenig nützlich d. Was hilft es uns z. B. zu wissen, daß die Hämaturie bei Nieren- nor „meistens profus" ist? Dies ist um so mehr bedauerlich, da se Diagnose so besonders schwierig ist. Bereits Bright bedeutete s dieses: „I have known the enlarged kidney to be mistaken for ease of the spleen, of the ovary, of the uterus, and for a tumor veloped in the concave part of the lever; nor is it perhaps possible, the greatest care and the most precise knowledge, althogether to oid such errors."

Trotzdem versteht ein gewiegter Forscher, wie Israel es ist, sogar, einere Tumoren, etwa in der Größe eines kleinen Apfels oder einer rsche, aufzufinden. Er nimmt dann die Untersuchung vor in der n ihm empfohlenen „halben Seitenlage". Auch empfiehlt er uns, dem Augenblicke des Überganges des Einatmens zum Ausatmen e Spitzen des Zeige- und Mittelfingers unter den Rippenbogen ein- ingen zu lassen, während gleichzeitig die andere Hand einen Druck sübt auf die Nierengegend, wodurch die infolge der Inspiration ge- nkene Niere in die alte Lage zurückkommen kann.

Noble[2]) zeigt uns eine neue Untersuchungsmethode der angeblich anken Niere, und zwar die Probeinzision in der Nierengegend. ach Spaltung der Fettkapsel ist es nicht schwierig, die Niere von m umliegenden Gewebe loszulösen und dann mit zwei Fingern den Teil der Oberfläche zu befühlen. Er empfiehlt die absolut un- fährliche Methode hauptsächlich für eine Untersuchung auf Nieren- eine hin. A priori würde sich also diese Methode auch eignen bei ierentumor, aber auch ich bin der Meinung, daß ein vollständiges reilegen der Niere gerade so ungefährlich ist und außerdem den Vor- il hat, daß der eventuelle Tumor sofort exstirpiert werden kann.

Auch Wendel[3]) befürwortet das Freilegen der Niere, um eine chere Diagnose zu ermöglichen. Er sagt, daß das noch nicht sehr freuliche Resultat der Operationen des Nierentumors nicht verursacht

1) Denaclara, Des hæmaturies dans les néoplasmes du rein. Thèse Lyon 99.

2) Noble, A new method of examing the kidney, especially for stone. Medical ews 1894.

3) Wendel, Die Entwicklung der Nierenchirurgie in den letzten Jahren. The- peutische Monatsh. 1899.

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wird durch einen Mangel der operativen Technik, sondern dadurch, daß die Diagnose meistens zu spät gestellt wird. „Von Frühsymptomen ist der Schmerz und die Blutung besonders wichtig. Beide sind aber inkonstant. In der Tat kann einer ausgedehnten Anwendung probatorischer Operationen nicht eindringlich genug das Wort geredet werden, da alle anderen Anhaltspunkte die Diagnose oft nicht hinreichend sichern können."

Es würde zu weit führen, an dieser Stelle die Differentialdiagnose näher zu erörtern; es seien nur einige weitere Krankheiten in der Nierengegend hervorgehoben, welche leicht eine Verwechslung veranlassen können.

1. Erweiterung der Lymphdrüsen.
2. Koprastase im Cöcum oder Kolon. Jenner sagt hierzu: Again collections of stools in the colon may be mistaken for an enlarged kidney.
3. Bösartige Tumoren des Kolons.
4. Erweiterungen der Leber und der Milz.
5. Ovarialtumoren.
6. Blinddarmentzündung.
7. Supperitoneales Myom im graviden Uterus.
8. Tumoren des Omentums, des Pankreas und des Mesenteriums.

Den Weg in diesem diagnostischen Labyrinthe zeigt uns, im Anschluß an die Ausführungen Israels Clado[1]), indem er uns den großen Nutzen der Palpation vor Augen führt. Ich möchte aber nicht verfehlen, die Aufmerksamkeit darauf zu lenken, daß sich nach einer gründlichen Palpation der Niere vielleicht Eiweißspuren im Urin zeigen, welche aber keinen differential-diagnostischen Wert haben (Schreiber[2]).

Tropische Hämaturie.

Zu den selteneren Ursachen der Hämaturie gehören auch verschiedene Parasiten und zwar: Distomum haematobium Bilharz, Filaria sanguinis, Strongylus gigas und der in Japan existierende Nephrophages sanguinarius. Alle diese Parasiten zeigen sich eigentlich nur in den Tropen. Morris sagt hierzu: „In the colder countries neither

1) Clado, Du ballottement rénal, valeur diagnostique. Médecine contemporaine, journal de l'hydrothérapie 1888.

2) Schreiber, Über renalpalpatorische Albuminurie und ihre Bedeutung für die Diagnose von Distopien, sowie von Tumoren im Abdomen. Zeitschr. f. klin. Medizin Bd. 55.

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rasites in the blood or urinary organs nor any lesion of the kidney discoverable."

Und zeigt sich trotzdem hin und wieder ein solcher Fall in der mäßigten Zone, so wird die Anamnese fast immer ergeben, daß ch der Keim der Krankheit bei einem Aufenthalte in den Tropen bildet hat. Derartiges erfuhren: Nitze[1]), Gutch[2]), Cobbold und mpson[3]).

Der Fall von Socoloff.

Noch seltener und merkwürdiger wie die Parasitenhämaturie ist der ll, den uns der russische Gelehrte Socoloff[4]) berichtet, und wir nnen denselben gewissermaßen als ein Unikum in der Geschichte r Hämaturie hinstellen.

Socoloffs Patient war ein Offizier, welcher im Jahre 1865 in birien großer Kälte ausgesetzt war. Während des Winters des genannten Jahres klagte er über fortwährende Müdigkeit, Schwindel und rmanentes Frostgefühl, besonders in den Füßen. Dazu kam Hämatrie mit leichten Schmerzen in der linken Nierengegend. Mit der ämaturie, welche nur wenige Stunden anhielt, verschwanden auch e übrigen Symptome. Der gleiche Komplex von Symptomen wiederolte sich öfters, und zwar immer dann, wenn der Patient der Kälte usgesetzt war. Sein Leiden reagierte gewissermaßen unverzüglich uf jedes Fallen und Steigen der Temperatur. Es war nicht möglich, ußer dem Temperaturwechsel eine Ursache der Krankheit zu entecken. Von weiteren Fällen von Nierenblutung in seiner Familie ar dem Patienten nichts bekannt, und zwischen je zweien der obenenannten Anfälle fühlte er sich verhältnismäßig wohl. Um den Zuammenhang zwischen Krankheit und Temperatur zu ergründen, verrdnete Professor Botkin ihm am 12. März einen Spaziergang bei iner Temperatur von 9° Celsius.

Tatsächlich traten die erwähnten Symptome auf. Die Untersuchung er ersten Urinmenge ergab viel Eiweiß und blutige Färbung, jedoch ein Sediment. Eine Stunde später war der Urin vollkommen schwarz.

1) Nitze, Über einen Fall von tropischer Hämaturie. Berliner med. Ges. 8. Januar 1891. Berliner klin. Wochenschr. 1891.

2) Gutch, A case of hæmaturia due to Bilharzia. The British Medical Journal 1900.

3) Simpson, Remarks on a case of hæmaturia from the presence of the Bilharzia haematobia. The British Medical Journal 1872.

4) Socoloff, Über einen Fall von wiederkehrender Nierenblutung im Zusammenhang mit jedesmaliger Erkältung der Integumenta communia. Berliner klin. Wochenschr. 1874.

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Jetzt zeigten sich bei der mikroskopischen Untersuchung Blutkörperchen. „Sie waren meistens entstellt; weiter fanden sich auch Faserstoff und Blutzylinder und gruppenweise unveränderte Nierenepithelien; irgendwelche Kristalle konnte man selbst bei der genauesten Untersuchung nicht entdecken."

Ein Spaziergang am 26. März bei einer Temperatur von 0 Grad hatte genau dieselben Folgen, nur in weniger heftigem Maße.

Zwischen beiden Anfällen war der Urin unverändert.

Socoloff erklärt den Fall folgendermaßen: Unter dem Einfluß der Kälte periodisch auftretende Blutung der linken Niere. Der Gedanke an Nierenblutung als Folge einer Entzündung der Nierensubstanz selbst kann, nach Socoloff, mit einiger Gewißheit ausgeschlossen werden, und zwar wegen des Umstandes, daß der Urin zwischen den einzelnen Anfällen normal war.

Wenn wir nun auch zugeben müssen, daß dieser Fall erheblich von dem üblichen Krankheitsbild der nephritischen Nierenblutungen abweicht, so dürfen wir doch auch nicht vergessen, daß die Abwesenheit von Eiweiß und Formelementen durchaus kein Beweis ist gegen Entzündungsprozesse der Nieren.

Außerdem kommt für Socoloff das Moment „Nierenstein" nicht in Betracht. Diese Folgerung ist zum mindesten voreilig. Er sagt, daß dazu die Schmerzen zu gering waren, und außerdem zeigte der Urin nicht die charakteristischen Merkmale des Steines: Kristalle, Sand usw.

Wenn nun aber der Stein z. B. eingekapselt wäre, und dadurch würden die Symptome des Vorhandenseins fehlen, so hätte er doch eine gewisse Reizung des Gewebes und infolgedessen Entzündung verursacht, und die Spuren dieser Entzündung hätte man doch im Urin wiederfinden müssen. Zwar enthielt das Sediment Schleim und Eiterteile, aber deren geringe Menge konnte ebensogut der gleichzeitig vorhandenen chronischen Urethritis zugeschrieben werden. Eine vorherige Ausspülung der Urethra hätte in dieser Hinsicht Gewißheit geben können.

Socoloff erklärt seinen Fall folgendermaßen: Er setzt einen abgeschwächten Tonus der Gefäße der linken Niere voraus. Diese Voraussetzung gründet er auf verschiedene Symptome, wie z. B. Atrophie der Muskeln der linken Hand und des linken Fußes, welche auf ein Gehirnleiden syphilitischen Ursprunges hindeuten würden. Die niedrige Temperatur nun bewirkt ein Zurückströmen des Blutes in die inneren Organe, also auch in die linke Niere, woselbst die Gefäße stellenweise, infolge des obenerwähnten abgeschwächten Tonus, dem erhöhten Blutandrange nicht widerstehen können. Er versucht dieser

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klärung weiteren Halt zu geben durch Hinweis auf den Umstand, ß Professor Tetschenoff Hämaturie konstatierte nach dem Zerneiden der Plexus renalis.

Auch diese Erklärung ist weither geholt. Übrigens ist der ganze fall Socoloff" in meinen Augen ein vergebliches Suchen nach dem usammenhange zwischen Ursache und Wirkung, und er wird auch hl, da eine Nephrektomie oder Sektion nicht stattgefunden hat, stets r uns ein interessantes klinisches Rätsel bleiben!

Einen annähernd gleichartigen Fall beschreibt Hassal[1]).

Der Patient von Dr. Hassal hatte ebenfalls periodische Hämaturie, ar ebenfalls wohlauf während der Zwischenperiode, jedoch der Urin thielt unter dem Einfluß der Kälte außer Blut auch Nierenzylinder, d die Behandlung weist darauf hin, daß Hassal an ein nephritihes Leiden glaubte.

Auch Pavy[2]) beobachtete vier dergleichen Fälle, bei welchen der rin ebenfalls außer Blutteilen auch Nierenzylinder und bei zweien rselben auch oxalsauren Kalk enthielt. Es wird sich also auch hier cht um Blutungen aus normalen Nieren handeln. Dasselbe halte ich n dem Falle Socoloff, und glaube deshalb auch, daß Professor otkin mit seiner Hypothese: „über den Zusammenhang der Nierenutung mit dem Leiden des zentralen vasomotorischen Apparates der ieren" den Nagel nicht weniger als auf den Kopf trifft.

Zu den seltenen Ursachen der Hämaturie gehört zweifellos die rinretention. Guyon[3]) hat es versucht, einen derartigen Fall durch as Tierexperiment zu erklären.

Gleich selten ist gewiß die Hämaturie infolge Arzneigenusses: wie B. nach Urotropine (Forbes, Milligan und Griffith[4]).

Den kuriosesten Fall von Hämaturie, dessen Verlauf an das Koische grenzt, berichtet uns Lepriore[5]). Bei einer 20jährigen Frau eobachtete er eine Veränderung in der Menstruation und zwar wie epriore behauptet infolge einer „Excesse in Venere". Die Mentruation wiederholte sich alle 8 Tage, dauerte gleich wie die hinzuommende Hämaturie einige Stunden und hinterließ eine Retentio

1) Hassal, Intermitent or Winter-hæmaturia. 1865.

2) Pavy, On paroxysmal hæmaturia. Urine from a case of paroxysmal hæmaria. A case of paroxysmal hæmaturia.

3) Guyon, De l'hématurie au cours de la rétention d'urine. Bulletin médial 1893.

4) Griffith, Hæmaturia following the administration of urotropine. The British Medical Journal 1901.

5) Lepriore, Un caso rarissimo di ematuria. Gazz. degli osped. e delle clin. 1902. Zitiert in Monatsber. f. Urologie 1902.

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urinae. Lepriore nahm die Katheterisierung vor, verschrieb Ergotin, Tannin und Bettruhe und bekämpfte so mit Erfolg die Hämaturie. Es gelang ihm außerdem die Hämaturie endgültig zu beseitigen, aber nicht etwa auf therapeutischem Wege, sondern indem er dem Ehepaare den Rat erteilte, den Dreyer nennt „eine Hygiene des Ehebettes". Dreyer bezweifelt, auch mit Rücksicht auf die ungenaue Untersuchung, stark, daß die Ursache hier tatsächlich in der Excesse in Venere zu suchen sei.

Auch ich glaube nicht, daß Lepriore das Richtige getroffen hat, dafür kommt Hämaturie in unserer Praxis zu vereinzelt vor.

Hämaturie und Schwangerschaft.

Als erster behandelt Guyon [1]) die Nierenblutungen im Zusammenhange mit Schwangerschaft.

Während der 2. und 3. Schwangerschaft beobachtete er bei einem seiner Patienten Hämaturie, welche ungefähr 2 Wochen dauerte und sich in dunkelroter Färbung des Urins äußerte. Nach der Entbindung zeigte die Hämaturie sich aufs neue und hielt nun 5 Monate an. Im Hospital Necker verordnete Guyon der Wöchnerin, das Stillen des Kindes einzustellen, und innerhalb 2 Tagen war die Hämaturie verschwunden. Einige Tage später nahm Albarran, weil die Patientin sich über Schmerzen in der rechten Nierengegend beschwerte, die Nephrotomie vor. Die Operation ergab nicht als Resultat den Erwartungen entsprechend einen Tumor, sondern eine makroskopisch normale Ren mobilis.

In einem anderen Fall, welchen Guyon und Champetier de Ribes beobachteten, zeigte die Hämaturie sich während der 4. und 5. Gravidität, und war das Ergebnis der zystoskopischen Untersuchung eine renale Hämaturie. Nach der 5. Schwangerschaft zeigte sich die Hämaturie auch unabhängig von der Gravidität, und da stellte sich heraus, daß sie verursacht wurde durch Nierentuberkulose.

Außer diesen 2 Fällen liefert Bouman [2]) uns noch 15. Es würde nicht schwer fallen, diesen 17 Fällen noch verschiedene hinzuzufügen, wie z. B. diejenigen von Chiaventone [3]).

Die beiden ersten Fälle stammen aus der Klinik von Treub. Die

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1) Guyon, Sur les hématuries rénales et vésicales. Annales des maladies des organes génito-urinaires 1897. Annales des maladies génito-urinaires 1899. — Guyon et Albarran, Hématuries de la grossesse. La Presse médicale 1899.

2) N. W. Bouman, Haematuria in gravidate. 1901.

3) Chiaventone, De l'hématurie de la grossesse. Annales des maladies des organes génito-urinaires 1901.

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ste Patientin hatte bereits im 12jährigen Alter Hämaturie nach dem nnehmen von Pulver gegen Enuresis nocturna. Während der 1. hwangerschaft schonte sie sich zu wenig und verhob sich. Am chsten Tage stellte die Hämaturie sich ein, dauerte 6 Tage und ederholte sich dann während dieser 1. Schwangerschaft nicht ährend der 2. Schwangerschaft stellte die Hämaturie sich wieder n, diesmal nach einer heftigen Gemütserregung. Die Untersuchung r Blase und des Urins lieferte keine Anhaltspunkte. Der Zustand r Patientin machte eine Unterbrechung der Schwangerschaft erforrlich und man führte diese Unterbrechung durch den Eihautstich rbei. Das Fruchtwasser sonderte sich ab, der Urin klärte sich allähnlich und war 4 Tage später frei von Blut. Die Entbindung ließ ch dem Eihautstich noch 18 Tage auf sich warten und hatte sodann nen normale Verlauf. 8 Monate später wurde sie abermals wegen ämaturie in der Klinik aufgenommen, diesmal ohne Schwangerschaft. ie Blase war ebenso wie das 1. Mal normal, der Urin dagegen entelt rote und weiße Blutteile aber keine Zylinder.

Nach 9tägiger Ruhe und Milchdiät verschwand die Hämaturie.

In der Literatur ist mehrfach die Rede davon, daß Hämaturie eine ünstliche Unterbrechung der Schwangerschaft veranlaßte. Einen lchen Fall berichtet uns Jameson[1]): „Her anæmia having reached extreme degree I decided to induce labour." Dasselbe geschah ei der 2. Schwangerschaft und in beiden Fällen erreichte die Unterechung ihren Zweck: das Verschwinden der Hämaturie. Jameson gt: „I am inclined to regard the hæmaturia as due to congestion f the kidney, from pressure in the renal vein by the enlarged womb." iese Hypothese kann nicht richtig sein, denn in der ganzen Literatur eht nicht ein einziges Mal geschrieben, daß Tumoren des Unterleibes ämaturie verursachen können.

Es ist unrichtig, diesen Fall als Haematuria graviditate zu bezeichen, vielmehr kommt die Hämaturie hier nur als Begleiterscheinung er Schwangerschaft in Betracht. Denn sonst müßte man einer aneren Ursache nachspüren für die Hämaturie während des nichtchwangeren Stadiums.

Auch in dem 2. Fall, ebenfalls aus der Treubschen Klinik, hanelt es sich wahrscheinlich in der Hauptsache um eine hämorrhaische Nephritis. Bei der Untersuchung des Urins wurden Nierenzylinder efunden.

Im 3. Fall aus der Veitschen Klinik konnte neben der Schwanger-

1) Jameson, Hæmaturia as a cause of miscarriage. The British Medical ournal 1900.

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schaft noch die Erweiterung und das Schmerzgefühl der rechten Niere in Betracht kommen. Auch hier wurde die Schwangerschaft unterbrochen und verschwand die Hämaturie nach der Entbindung. Die Symptome der rechten Niere aber dauerten fort. Aller Wahrscheinlichkeit nach bestand hier ein Kausalzusammenhang zwischen der Hämaturie und der schmerzhaft vergrößerten rechten Niere, und nicht zwischen der Hämaturie und der gleichzeitigen Schwangerschaft.

Auch in den beiden bereits oben erwähnten Fällen von Guyon konstatierte man Abweichungen der Niere, und zwar Ren mobilis bzw. Nierentuberkulose.

In einem 3. Fall von Guyon war die Hämaturie schon älteren Datums und hatte sich schon beim Beginn der Schwangerschaft gezeigt. Albarran nahm die zystoskopische Untersuchung vor und konstatierte eine beiderseitige Nierenblutung bei normaler Blase. Das Ausbleiben der Hämaturie fiel zusammen mit dem Ende der Schwangerschaft. Es ist nicht möglich in diesem Falle den Beweis zu liefern, ob die Nieren krank oder gesund waren.

Der nächste Fall von Guyon ist nicht eine Nieren- sondern eine Blasenblutung. Auf zystoskopischem Wege sah er, daß das Blut der erweiterten Venae der Gegend des Trigonums entstammte.

Der Krankheitsverlauf, den Kehrer[1]) uns berichtet, ist noch weniger ein Beweis. Seine Patientin war die Tochter eines Hämophilisten und war früher viel von Nasenbluten geplagt worden. Die Hämaturie blieb nach dem 3. Monat der Schwangerschaft aus, und dann traten die Nasenblutungen wieder in den Vordergrund. Es handelt sich also hier einfach um eine Hämophilika, bei welcher Epistaxis und Hämaturie natürliche Erscheinungen waren.

Dann folgen die beiden Fälle von Barton Cooke Hirst und Byrd Joung.

In beiden Fällen wird uns nichts mitgeteilt, was als Beweis dienen könnte, ob wir es mit gesunden oder kranken Nieren zu tun hatten.

Daudois[2]) berichtet uns folgendes: Bei einer 30jährigen Frau wurde Nephrotomie verrichtet wegen Hämaturie und Seitenschmerzen. Bei der Operation war absolut keine Abweichung zu konstatieren. Später wurde die Frau schwanger und da kalkulierte man folgendermaßen: Die Konzeption fand statt ausgerechnet 8 Tage vor dem Eintreten der Hämaturie, ergo, war der Zusammenhang zwischen der Schwangerschaft und der Hämaturie bewiesen.

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1) Kehrer, Arch. f. Gyn. 1870.

2) Daudois, L'hématurie congestive symptomatique de la grossesse. Annales de la Société Belge de Chirurgie 1895.

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Treub[1]) berichtet von einer starken Hämaturie bei einer Schwan-
ren. Als die Hämaturie trotz der Beendigung der Schwangerschaft
tdauerte und in dem Urin Tuberkelbazillen gefunden wurden, dachte
an Nierentuberkulose. Dieses bestimmte ihn zur Nephropexie.
e Operation ergab zwar keine Tuberkulose, aber doch eine Abwei-
ung der Niere. Man fand eine Erweiterung des Nierenbeckens und
ige Zysten, die eine trübe Flüssigkeit enthielten. Auch hier fehlt
eder der Zusammenhang zwischen der Schwangerschaft und der
maturie.

Der Zusammenhang fehlt gleichfalls in dem Fall, den Fridondani
der Gaz. med. de Pavia 1892 beschrieben hat. Es wurde bei der
tientin wegen besonders heftiger Blasenblutungen der Abortus ein-
eitet. Fridondani ist der Meinung, daß die Hämaturie durch
weiterung der Blasenkapillaren infolge der Schwangerschaft ent-
nden ist. Wie läßt sich damit vereinbaren, daß Blasenblutungen
ne so seltene Erscheinung bei Schwangerschaft sind?

Dann sind die Fälle von van de Wal, Niemeyer[2]), Stephan[3])
d P. Yff[4]). Niemeyer behandelte seine Patientin noch während
Monate nach der Schwangerschaft, ohne daß die Hämaturie wieder
ftrat. Dieses wäre ja ein schwacher Beweis dafür, daß es sich hier
n Hämaturie infolge der Schwangerschaft handelte; Niemeyer
lber jedoch glaubte an Tuberkulose.

Stephan schreibt in seinem Fall die Hämaturie Blasenvaricen zu.
Yff fand keine Zylinder im Urin, wohl aber amorphe Mengen
lzium und Magnesiumphosphat. Hier kann also Nierenstein die
rsache gewesen sein, oder der Urin selber, wenn derselbe eine starke
lzlösung enthielt.

Keiner dieser Fälle kommt also in Betracht um den Beweis zu
fern, daß es möglich ist, einen Zusammenhang zu konstatieren zwi-
hen Hämaturie und Schwangerschaft.

Chiaventone glaubt für die Erklärung der Hämaturie bei Schwan-
eren die fötale Intoxikationstheorie für Eklampsie in Anwendung
ringen zu dürfen.[5]) Die Richtigkeit hiervon möchte ich bezweifeln,
enn Eklampsie und Hämaturie treten selten gleichzeitig auf. Auch

1) Treub, Annales de la Société obstétricale de France 1890.

2) Niemeyer, Haematuria graviditatis. Med. Weekblad v. N. en Z. Nederland
900—1901.

3) Stephan, Haematuria graviditatis. Med. Weekblad v. N. e. Z. Nederland
900—1901.

4) Yff, Een geval van haematuria graviditatis. Med. Weekblad etc. 1901—1902.

5) Molas, Contribution à l'étude des hémorrhagies liées à l'éclampsie puer-
érale. Thèse de Paris 1877.

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müßte man außerdem die üblichen Abweichungen der eklamptischen Niere sowohl wie die übrigen Symptome der Eklampsie konstatieren können.

Ist also die Erklärung Chiaventones nicht ausreichend, auf welchem Wege ist dann die richtige Erklärung zu finden?

Wie wir gesehen haben, kann Hämaturie auf vielerlei Art verursacht werden. Die Schwangerschaft neutralisiert keineswegs eine dieser Ursachen und deshalb ist es durchaus begreiflich, daß Hämaturie auch bei schwangeren Frauen eintreten kann.

So betrachte ich denn die Hämaturie, welche sich bei Schwangeren zeigt, nicht als eine bestimmte Art, sondern als eine zufällige und unabhängige Begleiterscheinung der Schwangerschaft. Der Name „l'hématurie de la grossesse", wie Chiaventone sie nannte, ist also nicht richtig.

Richtig dagegen ist die erste These Boumans: „Haematuria in graviditate ist immer die Folge primärer Niereninsuffizienz." Eventuelle Blasenblutungen bleiben hierbei natürlich außer Betracht. Die Ursache der Niereninsuffizienz jedes einzelnen Falles ist hier nicht die Hauptsache. Die Hauptsache ist, daß die Hämaturie bei Schwangeren auf verschiedene Art verursacht werden kann, wie Tuberkulose, Nierenstein, Tumor usw., aber die Schwangerschaft als solche nicht genannt werden darf. Es ist nicht ausgeschlossen, daß die Schwangerschaft eine bestehende Hämaturie ungünstig beeinflußt, weil die Schwangerschaft nach der fötalen Intoxikationstheorie der Eklampsie den Nieren überhaupt und besonders kranken Nieren nicht zuträglich ist. Aber auch dem können wir keinen Glauben schenken. Wäre das Vorstehende richtig, dann würde Hämaturie während der Schwangerschaft weit häufiger vorkommen. In der ganzen Literatur sind kaum 20 Fälle zu finden, inklusive den nicht ganz einwandfreien d. h. bei welchen auch Nierentuberkulose oder Blasenblutungen die Ursache sein konnten.

Albutt[1]) spricht seinen sehr berechtigten Zweifel darüber aus, daß Haematuria in graviditate verursacht werden könne durch mechanische Ursachen wie z. B. starken Druck des vergrößerten Uterus. Dann müßte die Hämaturie viel häufiger auftreten, auch bei anderen Unterleibstumoren.

Kurzdauernde Nierenblutung durch Überanstrengung nennt Klemperer[2]) jene Fälle, deren Ursache er körperlicher Überanstrengung meint zuschreiben zu müssen.

1) Albutt, Albuminuria in pregnancy. The Lancet 1897.

2) Klemperer, Über Nierenblutungen aus gesunden Nieren. Deutsche med. Wochenschr. 1897.

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Er erzählt, wie sich Nierenblutungen zeigten bei einem 45jährigen
izier nach einem Ritt von 7 Stunden und bei einem 26jährigen
nne nach einer Radtour Berlin—Brandenburg und zurück.
Klemperer sagt: „Die ‚Nierenblutungen durch Überanstrengung'
l meines Wissens in der Literatur noch nicht beschrieben." Dieser
stand wäre sonderbar, wenn tatsächlich Überanstrengung Hämaturie
ursachen könnte. Aber auch nachdem Klemperer seine „Nieren-
tungen durch Überanstrengung" der allgemeinen Aufmerksamkeit
er gerückt hatte, wurden sie anderweitig nicht mehr wahrgenommen
r in der Literatur erörtert. Im Jahre 1903, also ausgerechnet sechs
re nach der Mitteilung Klemperers, nennt Schede[1]) im Zu-
nmenhang mit dieser Art der Hämaturie nur die Namen Klem-
rer, Leyden und Senator. Schede will sie einer Blutüberfüllung
chreiben, welche, wie er sagt, gleichermaßen durch Kongestion wie
rch Stauung hervorgerufen werden kann.

Die Literatur berichtet uns nichts von Nierenblutungen bei Soldaten
er Sportfreunden, welche doch gewiß körperlichen Überanstrengungen
sgesetzt sind. Es werden sich aber unter ihnen wenige, um nicht
sagen keine, befinden, deren Nieren nicht gesund sind. Daraus
nnte man den Schluß ziehen, daß die Hämaturie bei Überanstrengung
r auftreten kann bei nicht völlig gesunden Nieren. Sie wäre dann
va als traumatische Nierenblutung zu betrachten. Das Trauma als
ches käme also bei gesunden Nieren nicht in Betracht.

Außerdem kann bei Nierenstein- und dergleichen Kranken Nieren-
utung eintreten nach Überanstrengung, ohne jedoch durch letztere
rursacht zu werden. Sehr richtig betont deshalb Klemperer die
ichtigkeit der Differentialdiagnose, hauptsächlich in bezug auf Nieren-
ein. Aber auch wenn man nach möglichst genauer Untersuchung
e anderen Momente, welche als Ursache der Hämaturie in Betracht
mmen können, eliminiert hat, ist es fehlerhaft der Überanstrengung die
nzige Ursache zuzuschreiben. Wir wissen viel zu genau, daß, ohne
ikroskopische Untersuchung der Niere, Nierenstein und Nierentuber-
ulose bei Hämaturie nie mit absoluter Gewißheit auszuschließen sind.

Betrachtet man also die Klempererschen „Nierenblutungen durch
beranstrengung" als Symptom, welches auf eine kranke Niere schließen
ßt, so weiß man von vornherein, daß diese Fälle zu den seltenen gehören.
Wir haben schon weiter oben gesagt, daß Leute, welche fortwährend
örperlicher Überanstrengung ausgesetzt sind, beinahe ohne Ausnahme
icht an Nierenerkrankungen leiden werden, aber auf der anderen

1) Schede, Verletzungen und Erkrankungen der Nieren und Harnleiter. Hand-
uch der praktischen Chirurgie von v. Bergmann, v. Bruns und v. Mikulicz 1903.

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Seite werden Leute, deren Nieren nicht gesund sind, sich nicht dem Sporte oder sonstiger körperlicher Überanstrengung widmen.

Hämaturie als Begleiterscheinung anderer Krankheiten.

An erster Stelle muß hier die Malaria genannt werden.

In der Literatur finden wir diesbezüglich nur einige vereinzelte Fälle. Smith[1]) und Claverie[2]) berichten uns 5 resp. 1 Fall. In dem letzteren Fall war die Blutung sehr heftig. Sparkmann[3]) und Smith sind der Meinung, daß Chinin bei Malariahämaturie kontraindiziert ist.

Weiter kann die Hämaturie auftreten bei Intoxikations- und Infektionskrankheiten, wie z. B. bei Febris typhoidea. Derartige Fälle berichten uns Guinon[4]), Robin, Greenhow, Duckworth, Liebermeister, Griesinger, Murchison, Amat u. a. Mir selber begegnete in meiner Praxis ein Fall von akuter Miliartuberkulose[5]), aber der Fall von Askanazy (Hämaturie bei Diabetes) liefert den Beweis, daß man in derartigen Fällen ohne Sektion nicht ohne weiteres die Miliartuberkulose oder die Diabetes als Ursache der Hämaturie betrachten darf. In dem Askanazyschen Fall konstatierte man bei der Sektion nephritische Läsion der Nieren.

Hämaturie bei gesunden Nieren.

Bei allen diesen Fällen ist es das Symptom der Nierenblutung, welches am meisten in den Vordergrund tritt. Früher würde man sich die Frage vorgelegt haben: Welche Krankheit ist die Ursache dieser Nierenblutung? jetzt aber heißt es in erster Linie: Handelt es sich überhaupt um eine kranke Niere oder um Blutungen aus gesunden Nieren? Klemperer[6]) hat den Versuch gemacht, in einer sehr scharf-

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1) Smith, Malarial Hæmaturia. New York Med. Journal 1900.

2) Claverie, Observation de fièvre intermittente tierce hématurique. Revue médicale de l'Afrique du Nord 1901.

3) Sparkmann, Hæmorrhagic malarial fever; its treatment. The Therapeutic Gazette 1901.

4) Guinon, Fièvre typhoide à forme rénale hématurique survenant dans les cours d'un purpura exanthématique récidivant. Société médicale des hôpitaux 1898. La Presse médical 1898.

5) de Bruïne Ploos van Amstel, Über einen Fall von akuter Miliartuberkulose mit dem ausgeprägten Bilde des Abdominaltyphus. Berliner klin. Wochenschr. 1894.

6) Klemperer, Über Nierenblutungen bei gesunden Nieren. Deutsche med. Wochenschr. 1897.

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igen Abhandlung den Beweis zu liefern, „daß ein Organ, welches et, trotzdem gesund sein kann". Sogar unter Berücksichtigung es Ausspruches wird man der Meinung Professor Pels[1]) beipflichten sen, daß es ratsam ist, den sogenannten Hämaturien sine materia gewisses Mißtrauen entgegenzubringen. Klemperer nennt in er Linie die Menstruation, welche er, ebenso wie vor ihm bereits klinghausen, das physiologische Beispiel der neuropathischen renblutungen nennt. Dann nennt er uns Beispiele hysterischer ungen, beschrieben von Cohen, Tittel, Huß, und im Anschluß n zwei Fälle aus eigener Erfahrung, bei welchen es sich um natemesis resp. Hämoptöe hysterischer Art handelte, weil Magen Lunge bei der Sektion keinerlei Abweichungen aufwiesen. Es t noch nicht fest, welche Erklärung für diese neuropathischen renblutungen die richtige ist. Brown-Sequard und Ebstein achten sie als eine Reizung der vasomotorischen Nerven, welche n derartig erhöhten Druck auf die Blutgefäße nach sich zieht, daß e letzteren nicht mehr genügenden Widerstand zu leisten vermögen. pian glaubt an eine Lähmung der vasomotorischen Zentra mit Abvächung des Gefäßtonus und übermäßiger Füllung der kleineren tgefäße.

Der Behauptung Brown-Sequards könnte man entgegnen, daß der ganzen Literatur nicht ein Fall aufzuweisen ist von neuropacher Apoplexie ohne Arteriosklerose.

Dasselbe gilt auch für die Behauptung Vulpians; weshalb findet übermäßige, bis zum Zerspringen gesteigerte Füllung der Blutße nicht auch im Gehirn statt? Außerdem müßte man die Frage verfen, ob ein Organ, welches so hyperämisch ist, daß die Blutße zerspringen, als gesund betrachtet werden darf. Was diese ge speziell mit Rücksicht auf die Nieren anbetrifft, zitiert Klemer etwa 8 Fälle, um zu beweisen, daß tatsächlich Nierenblutungen vollkommen gesunden Organen beobachtet worden sind. Den en Fall nennt Sabatier[2]) selber „Néphralgie hématurique". In Untersuchungsprotokoll der wegen heftiger Lendenschmerzen und naturie exstirpierten Niere steht geschrieben: „On constate dans ce quelque peu d'inflammation conjonctive sans aucune tendence à suppuration, mais déterminant plutôt de la sclerose." Im Anschluß das Resultat dieser Untersuchung sagt Sabatier: „Les lésions, on

1) Pel, Die Nierenentzündung (M. Brightii) vor dem Forum der Chirurgen. eilungen aus den Grenzgebieten der Medizin und Chirurgie. 1901.

2) Sabatier, Névralgie hématurique. Néphrectomie. Guérison. Revue de rurgie 1889.

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le voit, sont si peu affirmées que nous n'hésiterons pas à reconnaître l'intégrité de l'organe enlevé."

Klemperer teilt ohne weiteres die Meinung Sabatiers in bezug auf den gesunden Zustand der Niere, trotzdem er u. m. als Resultat der mikroskopischen Untersuchung nennt: eine bindegewebige Entzündung, ohne Tendenz zur Eiterung, sondern mehr sklerosierender Natur.

In Wirklichkeit kann dieser Fall gelten als Beweis, daß Nephritis sich äußern kann mit Kolik und Blutung.

Ebenfalls beschreibt Klemperer den ersten Fall von Anderson[1]). Dieser Fall betraf ein 24jähriges Mädchen, welches seit 4 Jahren Anfälle von Lendenschmerzen und Hämaturie hatte. Anderson vermutete die Anwesenheit von Nierenstein, welcher aber bei der Operation nicht gefunden wurde. Er legte die Niere frei. Als er sie aber bei der direkten Inspektion und Betastung völlig normal fand, schloß er die Wunde, ohne die Niere herausgenommen zu haben. Es trat völlige Heilung ein.

Anderson selbst sagt, daß die Niere „was carefully examined by palpation and acupuncture, but without the discovery of any abnormality".

Es ist ohne Zweifel eine gefährliche Sache, bloß auf die makroskopische Untersuchung hin die Behauptung aufzustellen, daß die Niere vollkommen gesund ist. Daß Anderson nicht exstirpierte, ist begreiflich; nicht begreiflich ist aber, daß Klemperer diesen Fall hinstellt als einen Beweis für die Möglichkeit der Blutungen aus gesunder Niere, denn es ist sehr wohl anzunehmen, daß eventuelle nephritische Prozesse vorhanden waren und bei der Inspektion nicht gefunden wurden.

Der Fall von Schüller[2]) bestätigt übrigens die Richtigkeit meiner Vermutung. In diesem Falle handelte es sich um eine 49jährige Frau, welche bereits seit 10 Monaten renale Hämaturie hatte, und zwar kamen die Blutungen, wie die zystoskopische Untersuchung ergeben hatte, aus der rechten Niere. Makroskopische Untersuchung sowie Spaltung dieser rechten Niere ergaben keine Abweichung. Darauf wurde die Kapsel abgezogen und die Wunde geschlossen. Die Blutung hielt noch etwa 14 Tage an und blieb dann für immer fort. Die mikroskopische Untersuchung eines durch eine Probeexzision entfernten

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1) Anderson, Two cases of renal exploration for suspected calculus with subsequent nephrectomy in one. The Lancet 1889.

2) Schüller, Beitrag zur Lehre von den Blutungen aus anscheinend unveränderten Nieren. Wiener klin. Wochenschr. 1904.

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erenteilchens ergab, daß die Niere an chronischer Nephritis erkrankt war.

Ebenso wie der Fall Anderson, ist auch der schon zitierte Fall Passet[1]) kein starker Beweis für die Klempererschе Theorie.

In diesem Falle schloß man auf gesunde Nieren, ohne mikroskopische, ja sogar ohne makroskopische Untersuchung derselben.

Passet nennt diese Blutung renale Hämophilie, während als einzige Basis für diese Diagnose der Habitus haemophilicus des Patienten in Frage kommen kann. Klemperer, welcher diese Diagnose bestätigt, hat außerdem betont, daß die Blasenspülung der Blutung ein Ende gemacht hat, gerät hierdurch in Widerspruch mit sich selbst, weil selbstverständlich das letztere das erstere ausschließt. Der Umstand, daß die unnötige Blasenoperation ein so günstiges Resultat, das Ausbleiben der Blutung, aufzuweisen vermochte, darf lediglich ein Zufall genannt werden. Dieser Fall darf also meiner Ansicht nach keinesfalls in Betracht kommen als Beweis für die Möglichkeit der Blutungen aus normalen Nieren. Die Diagnose auf renale Hämophilie bei einer Frau mittleren Alters kann richtig sein, es fehlt aber jeglicher Beweis dieser Diagnose.

Passet schweigt über die Einzelheiten der Urinuntersuchung und konstatiert ausschließlich die Anwesenheit des Blutes. Ist damit denn hierdurch die Möglichkeit des Vorhandenseins weiterer nephritischer Prozesse ausgeschlossen? Korteweg[2]) sagt hierzu: Bei vielen Nierenoperationen hat man konstatieren können, daß sogar bei einer Nephritis in weit fortgeschrittenem Stadium der Urin eiweiß- und zylinderfrei sein kann.

Gleich wenig beweist der Fall von Broca.[3]) Auch er diagnostiziert auf renale Hämophilie, trotzdem keine Rede war von hereditärer Belastung und die Historia morbi uns nichts berichtet von hämophilischen Blutungen vor dem Erscheinen der Hämaturie. Auch eine kurz vorher stattgehabte Entbindung war in dieser Beziehung normal verlaufen. Seine Patientin beklagte sich über Hämaturie und heftige Schmerzen in der rechten Nierengegend. Im Urin wurden außer Blut wohl Epithelien und Zylinder, aber keine Spur von Nierenstein gefunden. Als Bettruhe und Milchdiät erfolglos blieben, wurde zur Operation geschritten. Die Diagnose lautete auf Tuberkulose oder Tumor.

1) Passet, Über Hämaturie und renale Hämophilie. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1894.

2) Korteweg, Die Indikationen zur Entspannungsinzision bei Nierenleiden. Mitteilungen aus den Grenzgebieten der Med. und Chir. 1901.

3) Broca, Hémophilie rénale et hémorrhagies rénales sans lésions connues. Annales des maladies des organes génito-urinaires 1894.

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Die freigelegte Niere zeigte keine Erweiterung und war von normaler Härte und Farbe, während auch die Untersuchung des Nierenbeckens und der Ureter kein positives Resultat ergab. Der erste Urin nach der Operation war noch bluthaltig, dann aber blieb die Hämaturie aus, und eine Untersuchung, welche 3 Jahre später stattfand, ergab, daß Patientin vollkommen geheilt war. In diesem Falle fehlt jeglicher Beweis sowohl für die Diagnose Hémophilie rénale, als auch dafür, daß die blutende Niere vollkommen gesund war. Nehmen wir an, daß die Blutungen tatsächlich durch Hämophilie verursacht wurden, dann bleibt es doch unverständlich, daß diese Operation, welcher von vornherein jeder praktische Nutzen bei einer Konstitutionskrankheit wie Hämophilie abgesprochen werden muß, Heilung bringen konnte, auch wenn man an eine außerordentliche Lokalisation dieser Krankheit glauben wollte. Und ebenso, wie bei den Klempererschen Fällen, möchten wir auch hier bezweifeln, daß die Niere gesund war, da dieses nicht auf mikroskopischem Wege festgestellt worden ist.

Auch Israel[1]) ist der Meinung, daß das Freilegen der Niere keinen großen Wert hat für die Diagnose.

Israel sagt hierzu: „daß allein eine ausgiebige Inzision der Niere selbst Klarheit verschaffen könnte, da die einfache Freilegung nicht mehr lehren würde, als die Abtastung des ganzen Organs durch die unverletzten Bauchdecken". Allerdings war in seinem Fall die Abtastung leicht vorzunehmen. Bei der 52jährigen Patientin Israels stellte sich am 15. Januar 1893 plötzlich ohne jegliche Veranlassung eine heftige Hämaturie ein, welche 11 Tage anhielt, und am 26. desselben Monats wurde wegen Nierentumor zur Operation geschritten.

Da die zystoskopische Untersuchung ergab, daß das Blut aus der linken Niere kam, wurde dieselbe freigelegt und darauf gespalten. Es erfolgte eine genaue Untersuchung der gespaltenen Niere, „welche nicht die geringste Abweichung von der Norm, weder abnorme Vaskularisation, noch Ekchymosen des Beckens oder des Nierenparenchyms ergab." Daraus folgerte Israel: „daß es sich um eine essentielle Nierenblutung ohne anatomisch erkennbare Grundlage handelte". Grosglik[2]) geht bei der Besprechung dieses Falles noch weiter: „Mit einem Worte, die Niere war ganz gesund." Der bereits genannte Fall von Schüler beweist, daß diese Folgerung nicht berechtigt ist. Nach der Operation (die Schnittwunde wurde vernäht und die Niere reponiert) blieb die Hämaturie dauernd aus. Israel kann zwar dieses günstige

1) Israel, Erfahrungen über Nierenchirurgie. Arch. f. klin. Chir. 1894, Bd. 47.

2) Grosglik, Über Blutungen aus anatomisch unveränderten Nieren. Sammlung klin. Vortr. von Volkmann Nr. 203 (Chir. Nr. 58).

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sultat nicht erklären, aber betrachtet es doch als Indikation, um in gleichen Fällen zur Operation zu schreiten.

Ferner zitiert Klemperer noch einige Fälle von Legueu[1]). Bei em 26jährigen Patienten zeigte sich im Jahre 1885 Hämaturie mit tigen Schmerzen. Die Schmerzen hielten an, trotzdem der Urin erhalb weniger Stunden blutfrei war. Im nächsten Jahre wiederlte sich der Anfall und dabei blieb es nicht. Im ganzen wurde ch etwa 40mal Blut im Urin konstatiert; Nierenstein wurde jedoch Urin nicht gefunden. Als weitere Symptome sind zu nennen: ufiges Erbrechen und Schmerzen im rechten Beine. Im Jahre 1891 rde endlich zur Operation geschritten; die linke Niere wurde freiegt und, da man keinerlei Abweichung fand, wieder reponiert. Die maturie blieb aus, die Schmerzen ebenfalls, aber nur bis zu dem genblicke, da der Patient erfuhr, daß außer der Nephrotomie keine entliche Operation stattgefunden hatte.

Auch hier wurde also die Niere für gesund erklärt, weil makroskopisch ine Abweichung konstatiert wurde. Die geringe Zuverlässigkeit der kroskopischen Untersuchung beweist nicht nur der Schüllersche ll, sondern auch der Fall von Braatz[2]). Braatz verrichtete den ktionsschnitt wegen Nephralgie. Die anscheinend normale Niere urde natürlich nicht exstirpiert, sondern reponiert, und zwar mit dem sultat, daß eine 3jährige Heilung eintrat. Dann zeigten sich dieselben Symptome von früher, worauf die Exstirpierung erfolgte. Die ikroskopische Untersuchung ergab, daß die exstirpierte Niere tuberlös war; ein jetzt genesener Herd muß also die Ursache der früheren eschwerden gewesen sein.

Auch der Fall von Schede[3]) ist für uns nicht ein beweisender in ieser Hinsicht.

Klemperer, der uns diesen Fall mitgeteilt hat, sagt, daß ein Djähriger Mann seit 13 Monaten an Hämaturie litt. Die Diagnose urde gestellt auf Nierenstein, Tuberkulose oder Tumor, weshalb die peration beschlossen wurde. Nach Eröffnung der Blase und Katherisierung der Ureteren ergab sich, daß das Blut aus der rechten iere stammte. Klemperer sagt dazu: „Bei der Untersuchung ergab e sich als vollkommen gesund.“ Schede jedoch teilt uns mit, daß chon während der Operation sich zeigte, daß die Nierensubstanz leicht

1) Legueu, Des névralgies rénales. Annales des maladies des organes génitorinaires 1891.

2) Braatz, Über operative Spaltung der Niere. Deutsche med. Wochenschr. 900.

3) Schede, Neue Erfahrungen über Nierenexstirpation. Jahrbuch der Hamburger Staatskrankenanstalten 1889.

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zerreißlich war. Auch bemerkt er noch, daß bei der pathologisch-anatomischen Untersuchung außer Anämie und einigen kleinen Extravasaten nichts Besonderes gefunden wurde.

Nun wissen wir, wie wir z. B. bei Eichhorst[1]) lesen, daß kleine Extravasaten, ebenso wie das leicht Zerreißliche der Nierensubstanz für die Diagnose großen Wert haben. Eichhorst sagt in Beziehung zur Nephritis: „Einen außerordentlich häufigen, fast regelmäßigen Befund stellen Hämorrhagien dar, die sich meist an das Gebiet der Nierenrinde halten und den Umfang eines Stecknadelknopfes nur ausnahmsweise überschreiten. In bezug auf Konsistenz der entzündeten Organe ist zu bemerken, daß das Nierenparenchym ungewöhnlich morsch und brüchig ist."

Schede bemerkt auch, daß die Urinuntersuchung kein positives Resultat gab, nur einmal wurden in demselben Nierenbecken-Epithel gefunden. Wenn man keinen besonderen Wert darauf legt, daß diese Nierenbecken-Epithelien gefunden sind, so beweist die Abwesenheit von Eiweiß und Formelementen der Niere nichts gegen die Diagnose Nephritis. Wir wissen auch, daß Nierenzylinder fehlen können, und zwar entweder ganz fehlen oder nach Sehrwald wohl anwesend, aber durch das Pepsingehalt des sauren Urins aufgelöst. Auch Leichtenstern und Sörensen haben konstatiert, daß bei Nephritis normaler Urin abgesondert werden kann. Die Ursache davon kann sein eine totale Anurie des kranken Nierengewebes, wodurch zeitweise nur von gesundem Nierengewebe Urin abgesondert wird. Doch ist außerdem die Abwesenheit von Eiweiß nicht Grund genug, die Diagnose Nephritis zu verwerfen. Dieulafoy spricht in Rücksicht auf die Albuminurie, wie von „un symptome infidèle et inconstant". Pel[2]) gibt uns noch die Möglichkeit, daß in diesen Fällen wohl Eiweiß anwesend sein kann, doch in so geringen Quantitäten, daß wir es mit unseren Reagentien nicht beweisen können.

Auch Goodal und Bartels beobachteten Fälle von Nephritis, wobei der Urin kein Eiweiß enthielt. Henoch[3]) sagt in dieser Hinsicht: „Aber auch hier wie überall in der Medizin gilt der Satz: Keine Regel ohne Ausnahme. Es gibt unzweifelhafte Fälle, in welchen trotz wiederholter Untersuchung des Urins (wenigstens mit den in der Praxis gewöhnlich benutzten Methoden) weder Eiweiß, noch mikroskopische Elemente der Nephritis in demselben nachzuweisen sind."

Die Behauptung Klemperers, daß in dem Falle von Schede die

1) Eichhorst, Handb. der spez. Path. und Ther.
2) Pel, Die akute und chronische Nierenentzündung. (Morbus Brigthii.)
3) Henoch, Vorlesungen über Kinderkrankheiten.

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Niere sich als vollkommen gesund ergab, ist unrichtig und übertrieben. Höchstens konnte man konkludieren, daß die Nieren vermutlich keine ernstlichen Läsionen zeigen werden.

Auch zitiert Klemperer den Fall von Durham[1]).

Durham operierte eine 41jährige Frau wegen Schmerzen und Hämaturie, die er einem Nierenstein zuschrieb. Bei der Operation bestätigte sich diese Diagnose jedoch nicht, weshalb die Niere nicht exstirpiert wurde. Da jedoch dieselben Beschwerden auch nach der Operation fortdauerten, wurde die Niere 2 Jahre später exstirpiert. Die Patientin starb bald nach der Operation und die exstirpierte Niere zeigte sich als normal.

Grosglik zitiert diesen Fall auch als zweifellosen Beitrag zu der uns interessierenden Frage.

Mit dieser Behauptung beweist Grosglik nur, daß er einen Fall zitiert, ohne ihn gelesen zu haben. Und er ist nicht der einzige, der so zitiert. Klemperer, der den Fall auch bespricht, sagt, ihn gelesen zu haben in dem British Medical Journal von 1889, während derselbe beschrieben wurde in dem British M. J. von 1872. Klemperer sagt auch: „Deswegen wurde nunmehr die Niere ausgeschnitten; ein Stein wurde nicht gefunden, die Niere erwies sich als gesund.“ Daß die Niere wirklich gesund sei, ist in der Beschreibung dieses Falles in dem British M. J. nicht zu lesen.

Lannois[2]) nennt diesen Fall auch und erzählt uns dazu, daß derselbe eine 43jährige Frau betrifft, die schon seit 2 Jahren an heftigen Schmerzen in der linken Nierengegend litt. „Croyant à des calculs, Durham fait une néphrolithotomie, ne trouve pas de calculs et en reste là. Cinq mois plus tard, les douleurs ayant reparu, exstirpation du rein, qui était sain.“

In Wirklichkeit verrichtete Durham bei dieser Patientin nie Nephrolithotomie, sondern er legte einfach die Niere frei. Ferner war hier nicht ein Zeitraum von 5 Monaten, sondern von 2 Jahren zwischen beiden Operationen. Zum Schlusse ersehen wir aus nichts die Wahrheit der Worte von Lannois betreffs der Niere: „qui était sain“.

Auch Brodeur[3]) erzählt uns, daß Durham erst eine Nephrolithotomie verrichtete.

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1) Durham, Removal of the kidney. The British Medical Journal 1872, p. 534 und 565.

2) Lannois, De l'exstirpation du rein ou néphrectomie. Revue de Chirurgie 1881.

3) Brodeur, De l'intervention chirurgicale dans les affections du rein. Thèse de Paris 1886.

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Durham selbst sagt von diesem Falle nur folgendes: „On Tuesday, Mr. Durham removed the right kidney of a woman at Guy's Hospital. The patient who is 43 years of age, is the same in whom Mr. Durham exposed the kidney, which was removed on Tuesday, with the expectation of finding a calculus in or about its pelvis. No calculus being found on that occasion, and the kidney appearing healthy, the wound was closed. The patient recovered from the effects of the operation well and the wound healed, but she was not relieved from the severe pain, hæmaturia and other symptoms, from which she had suffered. The patient early on Thursday morning was apparently progressing satisfactorily in every way; there being no sign of any bad symptom; the temperature was normal; the pulse beating ninety per minute, and although weak, not more so than frequently had been the case before the operation; and the patient had less pain than she had constantly suffered for long previously."

Das ist alles.

Dieser Fall ist, n'en déplaise Grosglik, beweisend für nichts. Höchstens sieht man wieder einmal daraus, daß das Extirpieren einer Niere nicht ungefährlich ist, jedoch dafür braucht man keinen Beweis, das ist eine alte Geschichte.

Was die Ursache ist, daß dieser anfänglich so günstig verlaufende Fall letal endet, erzählt Durham uns nicht. Das einzige, was wir hierüber vernehmen, ist, das wir auf S. 565 (British Medical Journal 1872) lesen: „Mr. Durham's operation of exstirpation of the kidney has ended fatally." Dennoch macht dieser nichts beweisende Fall die Runde in der Literatur und wurde dann entweder zitiert nach origineller Mitteilung!!! oder nach Legueus Zitat in seinem Artikel: „Des névralgies rénales. Annales des maladies des organes génito-urinaires, 1891.

Der einzige, der diesen Fall richtig zitiert, war Sabatier.[1])

Er aber sagt uns, daß bei der ersten Operation das Freilegen der Niere „la vue et la palpation permirent d'en reconnaître l'intégrité".

Nun wissen wir aus Erfahrung und aus der Literatur, wie wenig man geben kann auf den makroskopisch scheinbar normalen Aspekt einer freigelegten Niere.

Grosglik zitiert auch den zweiten Fall von Lauenstein[2]). Dieser behandelte einen 42jährigen Seemann, der schon seit 12 Jahren Schmerzen in der rechten Nierengegend hatte. Dazu traten im Jahre 1886 Beschwerden über Hämaturie und heftigen Urindrang; er mußte

1) Sabatier, Néphralgie hématurique. Néphrectomie. Guérison. Revue de Chirurgie 1889.

2) Lauenstein, Zur Chirurgie der Nieren. Deutsche med. Wochenschr. 1887.

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16mal in 24 Stunden Urin lassen. In dem Urin selbst wurden außer roten auch weiße Blutkörperchen gefunden.

Da die Urinuntersuchung jedoch nicht zu einer sicheren Diagnose führte, so wurde die Operation beschlossen, weil Lauenstein einen großen Nierenstein vermutete. Im August 1886 wurde die Operation verrichtet; die Niere wurde freigelegt, aber kein Stein gefunden. Auch das Nierenbecken wurde geöffnet, aber auch ohne Resultat. Um die Hilus in die Wunde zu ziehen, um so in das Nierenbecken zu gelangen, zog Lauenstein drei Fäden durch das Nierengewebe. Die hierbei unwillkürlich verrichtete Akupunktur ließ auch nicht die Anwesenheit eines Steines erkennen. Da man ferner auch nichts Abnormales an der Niere konstatieren konnte, wurde die Wunde tamponiert, nach Reposition der Niere. Die Wunde heilte gut, und das weitere Resultat war, daß der Mann vollkommen gesund wieder zur See ging, und 3 Jahre später hatte Grosglik Gelegenheit zu konstatieren, daß sein Patient noch immer total genesen war. Grosglik erzählt uns diesen Fall unter dem Titel: „Über Blutungen aus anatomisch unveränderten Nieren."

Lauenstein sagt in Beziehung auf das geöffnete Nierenbecken: „Aus dem eröffneten, offenbar erweiterten Nierenbecken floß weder Blut, noch Eiter." Auch wenn man sich der Ansicht Lauensteins anpassen will, daß eine Niere mit erweitertem Nierenbecken trotzdem normal ist, so fehlt hier doch jede pathologisch-anatomische Untersuchung und steht auch die Behauptung einer gesunden Niere ohne jeglichen Beweisgrund.

Morris[1]) operierte einen jungen Mann von 20 Jahren, da derselbe infolge heftiger Anfälle von Hämaturie, welche sich schon seit 10 Monaten wiederholten, sehr anämisch geworden war. Da der Patient besonders über Schmerzen in der rechten Nierengegend klagte, wurde die Operation der rechten Niere beschlossen. Morris sagt hierüber: „The right kidney was cut into and thoroughly examined with negative result; nothing was found to explain the hæmaturia, and no improvement in the symptoms resulted." 10 Tage nach der Nephrotomie verrichtete er die Nephrektomie, weil die Hämaturie stets fortdauerte. Jedoch hörte dieselbe auch nach der Nephrektomie nicht auf und starb der Patient am Tage nach der Operation. Morris sagt über die exstirpierte Niere: „There was nothing in the naked-eye appearances of the kidney removed, or of the left kidney or of the bladder, or any part of the urinary system to explain the cause of the hæmorrhage. Neither a microscopic nor a bacteriologial exami-

1) Morris, Surgical diseases of the kidney and ureter. 1901.

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nation of the kidneys was, I regret to say, made, their naked-eye appearances being so normal that further examination was unfortunately omitted." Mit den Worten: „I regret to say" beweist Morris, daß er mit uns derselben Meinung ist, daß der makroskopische, den „naked-eye" Aspekt der Niere, selbst in scheinbar sicheren Fällen, doch nicht überzeugend genug ist, um ohne mikroskopische Untersuchung zu einer unveränderten Niere zu konkludieren.

Lauenstein ist auch sehr schlecht zu sprechen über das diagnostische Resultat seiner Operation.

Er konkludierte nicht, daß die Niere mit dem erweiterten Nierenbecken gesund sei, sondern daß das Diagnostizieren auf dem Gebiete der Erkrankungen der Harnwege mit Schwierigkeiten verknüpft ist.

Auch glaubt er nicht, daß der Erfolg dem Eingriffe zu verdanken sei, denn er sagt, daß der fernere Verlauf befriedigender war, wie die gestellte unrichtige Diagnose, und daß vollständige Genesung erfolgte, „trotz des Eingriffes".

Dieselbe Frage wegen des Resultates ist wohl auch zu stellen in dem von Mannino[1]) mitgeteilen Fall über Hämaturie.

Mannino behandelte einen 2jährigen Knaben, bei welchem Anfälle von Hämaturie bemerkt wurden, die bis zu 3 Tagen dauerten. Angesichts des negativen Resultates der Blasenuntersuchung, mit Rücksicht auf die Anwesenheit von Konkrementen, und weil die Eltern des Kindes an Syphilis litten, nahm man an, daß man es hier nicht mit Nierenstein, sondern mit hereditärer Syphilis zu tun habe. Die Diagnose wurde dadurch sicher, weil eine Kur von 62 Tagen, während welcher Zeit das Kind mit Quecksilber und Jodkali behandelt wurde, totale Genesung als Resultat hatte. Nirgends findet man in der Literatur Hämaturie als Symptom von hereditärer Lues verzeichnet. Dazu kommt noch, daß man bei einem 2jährigen Kinde nicht so einfach konstatieren kann, ob vielleicht nicht doch während der 62 Tage ein kleiner Stein abgesondert wurde. Wenn dieses wohl der Fall war, dann ist das gute Resultat der antisyphilitischen Behandlung zu begreifen, auch bei einem nicht syphilitischen Kinde. Jede Behandlung würde dann wohl scheinbar die Ursache der Genesung gewesen sein. Ebenso ist es mit dem Resultate der Behandlung von einem Patient von Pope.[2])

Dieser Patient litt seit $2^1/_2$ Jahren an Hämaturie, „so persistant, so continuous and moreover marked, considering the constant drain

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1) Lorenzo Mannino, Hämaturieanfälle infolge von hereditärer Lues. La Riforma medica. Ref. Deutsche med. Wochenschr. 1887.

2) Pope, A case of persistant hæmaturia; treatment; cure. The Lancet 1889.

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n the system, by such slight constitutional disturbance, that it may e well to record it, with the treatment, which was happy succesful."

Jede Behandlung war ohne Erfolg geblieben. Der Urin enthielt ne große Quantität roter und eine geringere weißer Blutkörperchen. as spezifische Gewicht war 1024, und sagt Pope: „Supernatant yer gave a faint trace of albumen." Pope meint, daß hier kein Grund war, ein akutes oder subakutes Nierenleiden, Entzündung, teinleiden usw. zu diagnostizieren. Ebensowenig konnte man an arasiten denken, als: „I therefore made up my mind that the hæmatria was not symptomatic, so to speak, but in itself the result of ome functional failure of the kidney."

Hier haben wir also wieder die Diagnose Hämaturie aus einem natomisch normalen Organ, ohne daß dasselbe untersucht worden st und trotzdem die Anwesenheit von Eiweiß in dem Urin festgetellt wurde.

Die Behandlung blieb ohne Erfolg, bis eine Dosis Cascara gegen lie Obstipation angewendet wurde. Daraus konkludierte Pope die Anwesenheit von „some altered condition of blood-pressure, in all probability marked increase which caused the red blood cells to be forced through the minute vessel walls into the secreting tubes thence o be excreted, and therefore an effort was made to diminish this ncreasing pressure". Um diesen Zweck zu erreichen, gab er Laxantia und zwei „vapour baths" in jeder Woche. Die spätere Genesung schrieb er seiner Behandlung gegen den erhöhten Blutdruck zu.

Daß durch Obstipation der Blutdruck so erhöht werden kann, daß Nierenblutungen von solcher Heftigkeit verursacht werden, dürfte wohl niemand glauben. In Wirklichkeit soll Blutdruckerhöhung in dem Bauche, durch welche Ursache sie entstanden sei, doch wohl nie diese zur Folge haben.

Klemperer behauptet, daß in allen seinen Fällen von Hämaturie und Nierenkolikanfällen durch die Operation die vollkommene anatomische Intaktheit der blutenden Nieren sicher festgestellt wurde. Wir sehen aber, daß die angenommene Gesundheit oder anatomische Intaktheit der Nieren in all diesen Fällen die Probe der Kritik nicht bestehen kann, und verfällt somit auch die Erklärung, die er uns zu diesen Blutungen gibt. Klemperer glaubt, daß die Blutungen entstanden sind durch Lähmung der vasomotorischen Nerven. Als einzigen Beweis für diese Behauptung sagt er, daß es angioneurotische Ödemen gibt, also gibt es auch angioneurotische Blutungen. Außerdem, meint er, kann der gute Erfolg von so verschiedenartigen Eingriffen, wie Freilegung der Niere, Akupunktur und Sektionschnitt, wohl nicht anders erklärt werden, als eine Einwirkung auf das Nerven-

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system. Wir werden aber sehen, daß diese Operationen ihren Erfolg verdanken der dadurch verursachten Verminderung der Spannung des Nierengewebes.

Daß Klemperer selbst in dieser Hinsicht seine Ansicht wohl etwas verändert hat, bemerkt man daraus, daß er in seinem Lehrbuche sich über diesen Gegenstand nicht so positiv ausspricht.

Klemperer[1]) sagt darin in dem Kapitel „Die Krankheiten der Nieren": „Die Bedeutung der renalen Hämaturie ist in den meisten Fällen eine symptomatische. Wohl hält er in einzelnen Fällen eine angioneurotische Hämaturie für möglich, aber wenn man gut untersucht, dann folgt aus dieser Sachlage jedenfalls, daß man bei länger dauernden Hämaturien immer von neuem zu untersuchen hat, ob nicht irgendeine somatische Ursache für dieselben zu entdecken ist; erst bei vollkommener Ergebnislosigkeit dieser Bemühungen darf man sich der Diagnose der angioneurotischen Blutung zuwenden."

Befolgt man diesen Rat von Klemperer genau, so wird man finden, daß diese Diagnose nach genauer wiederholter Untersuchung nicht mehr vorkommen wird.

Die Diagnose der angioneurotischen Blutung ist doch auch in Wirklichkeit nicht viel mehr als eine Umschreibung der früheren essentiellen Blutung, Néphralgie hématurique oder lokalen Hämophilie. Mit diesem Namen umschreibt man jedoch weniger das Krankheitsbild, als die Unwissenheit über das Essentielle der Krankheit.

Je mehr man in der Literatur über diese Blutungen nachliest, desto mehr Fälle findet man, wobei durch die mikroskopische Untersuchung die richtige Diagnose gestellt wurde, während ohne dieselbe in vielen dieser Fälle man sie wie Klempersche angioneurotische Nierenblutung betrachten würde. So beschreibt Routier[2]) einen Krankheitsfall von einem 28jährigen, immer gesunden Mann, bei dem plötzlich in der Nacht Hämaturie auftrat. Nachdem dieselbe 17 Tage gedauert hatte, trat rechtsseitige Nierenkolik hinzu, und zeigte die zystoskopische Untersuchung, daß das Blut aus der rechten Niere stammte. Da man nun die Diagnose auf malignen Tumor stellte, wurde die Nephrektomie bei dem inzwischen sehr anämisch gewordenen Patienten verrichtet. Makroskopisch zeigte die ausgeschnittene Niere keine Abnormalität. Bei der mikroskopischen Untersuchung aber war die Diagnose mit Gewißheit auf beginnende primäre Nierentuberkulose zu stellen.

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1) Klemperer, Lehrbuch der inneren Medizin. 1905.

2) Routier, Tuberculose rénale. Hématurie. Néphrectomie. Guérison. Annales des maladies des organes génito-urinaires 1895.

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Trautenroth[1]) erzählt auch einen ähnlichen Fall, wobei die Nierenutung jedoch nicht von Kolik begleitet war. Auch hier wurde die ephrektomie beschlossen, da man einen malignen Tumor vermutete. In der makroskopisch nicht veränderten Niere fand man nur ein utkoagulum in dem Nierenbecken. Bei der mikroskopischen Unterchung zeigte es sich aber, daß diese Niere diffus tuberkulös erankt war.

Auch Israel[2]) teilt uns ähnliche Fälle mit. Einer seiner Patienten, n 58jähriger Herr, litt seit einiger Zeit an Hämaturie, zeitweise ohne nd dann wieder mit Kolikschmerzen. Während der Zeit, da er keine ämaturie hatte, war der Urin eiweißfrei. Die Urinuntersuchung gab weiter, daß keine Zylinder, wohl aber Kristalle von Uraten genden wurden. Die Nieren waren nicht schmerzhaft beim Druck. ei absoluter Bettruhe blieb die Hämaturie während 6 Wochen rt, kehrte jedoch unmittelbar nach körperlicher Bewegung zurück. rael legte nun die Niere frei wegen seiner Diagnose Nierenstein. n der Niere war auch nach dem Sektionsschnitt nichts Abnormales sehen, nur bemerkte man eine geringe Erweiterung des Nierenckens. Der auch an Aortainsuffizienz leidende Patient starb 35 Stunden ach der Operation, und nun bemerkte man bei der durch Professor ansemann verrichteten mikroskopischen Untersuchung, daß in beiden ieren eine diffuse Nephritis bestand. Ohne diese mikroskopische ntersuchung würde auch dieser Fall beschrieben sein als essentielle ierenblutung.

Israel gibt uns noch eine Serie von 14 solcher Fälle. Für uns nd von diesen Fällen von besonderer Wichtigkeit die Fälle 2 und 4, elche er nennt: „Fälle mit mangelnder mikroskopischer Untersuchung, hne makroskopisch nachweisbare Veränderungen im Nierenparenchym, hne chemische und mikroskopische Anomalien des Urins, ohne Lageeränderungen der Niere.“ In diesen beiden Fällen war nicht von ämaturie die Rede, wohl aber hatten die beiden Patienten Nierenoliken. Beim Fall 2 war keine einzige Nierenanomalie zu konstaeren, außer einer sehr starken Verwachsung mit der Nierenkapsel. uf Grund dieser starken perirenalen Verwachsung betrachtet Israel iesen Fall als ein Nierenleiden, welches wahrscheinlich seinen Urprung hatte in einer vorangegangenen Perityphilitis. Er betrachtet

1) Trautenroth, Lebensgefährliche Hämaturie als erstes Zeichen beginnener Nierentuberkulose. Mitteilungen aus den Grenzb. d. Med. u. Chir. 1895.

2) Israel, Über den Einfluß der Nierenspaltung auf akute und chronische rankheitsprozesse des Nierenparenchyms. Mitteilungen aus den Grenzgebieten er Med. und Chir. 1900.

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Fall 4 als eine Lokalisation in der Niere von einem bestehenden rheumatischen Leiden. Hier bestand in der Tat schon seit 8 Jahren Gelenkrheumatismus, und traten die Nierenkoliken sowohl zu gleicher Zeit mit den Gelenkschmerzen auf, als auch einzeln. Diese rheumatische Form des Nierenleidens erinnert uns sehr an die durch Mabbaux[1]) beschriebene „Hématurie goutteuse". Doch sind Israel und Mabbaux sehr verschiedener Ansicht in ihren Erklärungen betreffs dieser wahrgenommenen Lokalisation eines rheumatischen oder arthritischen Prozesses. Während Israel meint, es mit einer Lokalisation von rheumatischen Prozessen in der Niere zu tun zu haben, sagt Mabbaux, daß die Hämaturie nicht verursacht wird von einem bestehenden Nierenleiden, sondern zu betrachten ist als das erste und oft einzige Symptom von einem arthritischen Leiden. Der dadurch entstandene größere Gehalt des Blutes an Uraten ist dann Ursache einer Nierenkongestion, und diese wieder von Nierenblutungen.

Den ersten Fall von Israel betrachtet er auch als eine gichtische Affektion der linken Niere. Israel jedoch diagnostiziert hier einen chronischen entzündlichen Prozeß der Niere mit akuten schmerzerregenden Exazerbationen, ohne nachweisbare Veränderungen des Urins.

Pel[2]) will diese Diagnose nicht anerkennen, ebensowenig die von Fall 1, wie von Fall 4. Er ist ungefähr derselben Meinung wie Mabbaux. Pel ist überzeugt, daß sehr kristallreicher Urin auch ohne Anwesenheit von Nierenstein imstande ist, Koliken mit Ureterkrampf, sowie Blutungen und sonstige Symptome von Nierenstein zu verursachen.

Israel betrachtet seinen vierten Fall wie eine kongestiv-entzündliche Lokalisation der rheumatischen Erkrankung in der Niere mit intrakapsulärer Spannungserhöhung. Pel ist hiermit nicht einverstanden, sondern betrachtet diesen Fall wie Lithiasis, ohne Anwesenheit von Nierenstein, aber mit sehr kristallreichem Urin.

Gegen diese Behauptung von Pel ist folgende praktische Erwägung zu stellen. Bei allen Patienten mit Nierenstein ist der Urin sehr kristallreich. Somit müßten also nach Pel dann auch ohne daß ein Stein in den Ureter gelangt, nur durch den Einfluß des kristallreichen, saturierten Urins, Blutungen und Kolikschmerzen auftreten. Doch ist dieses bis jetzt noch nicht mit Sicherheit zu konstatieren gewesen. Bei Patienten, bei denen wirklich Nierensteine anwesend sind, sieht man nie Blutungen und Kolikschmerzen auftreten, ohne daß ein Stein den Ureter passiert. Nun ist es wohl wahr, daß nicht nach

1) Mabbaux, De l'hématurie goutteuse.

2) Pel, Die Nierenentzündung (M. Brightii) vor dem Forum der Chirurgen. Mitteilungen aus den Grenzgebieten der Med. und Chir. 1901.

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jedem Anfall von Nierensteinkolik ein Stein abgesondert wird; oft sind dazu mehrere Anfälle notwendig. Solange sich der Stein dann nicht gezeigt hat, kann man die konstatierten Symptome dem Einfluß des kristallreichen Urins zuschreiben, anstatt dem des Steines. Doch welcher Nutzen resultiert daraus für den Patient? Dasselbe können wir jedoch auch fragen in den Fällen, wo man nach einer genauen Untersuchung, auch mit den besten und neuesten Hilfsmitteln der Wissenschaft, Nierenstein mit Sicherheit auszuschließen vermeinte. Dann ist die Diagnose von der bestimmten Form der Lithiasis, die Pelsche Lithiasis, Grund genug, mit ihm zu sagen, daß hier keine Indikation zur Operation besteht. In einzelnen Fällen soll dadurch der Patient einer hinterher unnötigen Operation entgehen, doch in wievielen Fällen wird wohl die nötige Operation dadurch versäumt oder zu spät verrichtet werden? Zunächst müssen wir auch nicht vergessen, daß die Diagnose der Pelschen Lithiasis auf dasselbe herauskommt, wie die Diagnose essentielle Hämaturie, nämlich einen Namen geben einer uns dunklen Krankheit.

Auch Morris[1]) bespricht diese Streitfrage, wenn er abhandelt: Lithiasis and nephralgia due to functional derangement of the urine. Er sagt darüber: „There is a great resemblance between the symptoms of renal calculus and those of hyper-acid urine. There is no more certain cause of pain in the loins, of frequent micturition, and even of a slight amount of pus or blood in the urine, than very acid urine, and urine overcharged with uric acid or calcium oxalate crystals." Demnach sollen also keine organischen Nierenveränderungen entstehen, aber jedoch würden Fieber, Hämaturie und Pus in dem Urin, selbst während eines Zeitraums von 1—2 Jahren, möglich sein. Hierbei können sich auch mehr oder weniger heftige Schmerzen in den Lenden einstellen. Israel hat diese Behauptung von Pel im voraus beantwortet. Israel macht uns darauf aufmerksam, daß in den Fällen von Morris die Hämaturie stets wenig heftig war, daß dagegen Eiweiß im Urin ein konstantes Symptom war, und daß Morris sagt, daß niemals „actual or permanent degeneration of the renal tissues" zu konstatieren war, während in seinen mikroskopisch untersuchten Fällen wohl Nephritis gefunden war. Auch ist noch ein Argument gegen die Behauptung von Pel, daß die Hämaturie und die Schmerzen stets einseitig vorkommen, was unbegreiflich ist, wenn der Einfluß, welcher davon die Ursache ist, der kristallreiche Urin auf beiden Nieren stets ähnlich einwirkt.

1) Morris, Surgical diseases of the kidney and ureter including injuries malformations and misplacements. 1901.

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Aber man kann auch sagen, daß, wenn in der Tat diese Pelsche Lithiasis Hämaturie und Nierenkoliken verursachen kann, auch das Vorkommen von essentieller Hämaturie, von den Klempererschen Nierenblutungen aus gesunden Nieren eine Tatsache ist.

Pel selbst macht nicht diese natürliche Folgerung seiner Theorie.

Im Gegenteil mißtraut Pel dieser Form der Hämaturie. Mit ihm sind wohl die meisten Mediziner der Ansicht, daß, falls diese Hämaturie wirklich vorkommt, sie doch äußerst selten ist.

Das Urteil von Dsirne[1]) ist: „Nach genauem Studium der Literatur bleiben nur wenige Fälle zurück, die diesen Formen entsprechen." Auch Albarran[2]) ist sehr mißtrauisch gegenüber den beschriebenen Fällen von Néphralgies hématuriques, und von Blutungen aus gesunden Nieren. Er will keinen einzigen Fall anerkennen, wobei keine mikroskopische Untersuchung der Niere stattfand.

Noch eine Einwendung gegen die Pelsche Theorie ist, daß wenn wirklich ein erhöhter Gehalt des Urins an Uraten und Oxalaten Ursache wäre von Nierenblutungen aus gesunden Nieren, dann würden diese Blutungen, besonders da ihnen so viel Aufmerksamkeit gewidmet wird, viel mehr wahrgenommen werden.

Pel macht, hinsichtlich der Mitteilungen von Israel die Bemerkung, daß es unbegreiflich ist, daß diese Nierenblutungen mit Erfolg bekämpft sind mit so verschiedenartigen Eingriffen, wie das Freilegen der Niere, der Sektionsschnitt usw. Wir haben schon gesehen, daß diese Eingriffe nicht so abweichend sind in ihrem Einfluß auf das Nierengewebe, denn durch alle diese Operationen wird die Spannung des Nierengewebes vermindert. Aber man kann auch Pel die Frage stellen, warum diese verschiedenen Eingriffe Erfolg haben können, wenn man die Pelsche Lithiasis als Ursache der Blutungen betrachtet.

Pel wünscht, daß man in den Fällen von Pelscher Lithiasis nicht zur Operation schreitet. Wir haben gesehen, daß dadurch vielleicht eine notwendige Operation versäumt wird, aber doch kann man sich der Ansicht Pels anschließen, wenn man den Rat von Morris dabei befolgt.

Morris[3]) sagt hierüber: „The diagnosis in such cases will be cleared up by the improvement which follows upon alkaline treatment or a course of piperazine. The citrate or tartrate of potash or soda,

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1) Dsirne, Beitrag zur Frage der Nephrolithiasis, der Hydro- und Pyonephrose. Monatsberichte für Urologie 1902.

2) Albarran, Diagnostic des hématuries rénales. Ann. des maladies des organes génito-urinaires 1898.

3) Morris, Surgical diseases of the kidney and Ureter.

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given every four hours during the day, neutralises the acidity of the urine as it is secreted, and thus the cause of the symptoms disappears, and with it the symptoms themselves."

In den Fällen von Pelscher Lithiasis kann man also, wie Pel es will, nicht sofort operieren, aber erst einige Tage die Behandlung von Morris probieren. Hat diese in kurzer Zeit keinen Erfolg, dann gehe man bald zur Operation über.

Dsirne steht nicht allein mit seiner Meinung hinsichtlich der Blutungen aus gesunden Nieren. So sagt z. B. Suter[1]): „Die essentielle Hämaturie hat vor wenigen Jahren als ein bestehendes, wohlabgegrenztes Krankheitsbild noch eine bedeutende Rolle gespielt. Heute sind sichere, nicht nur klinisch sondern auch pathologisch-anatomisch konstatierte Fälle extrem selten." Auch Malherbe und Legueur[2]) sind dieser Meinung, nur glauben sie, daß diese Fälle auch nicht extrem selten, sondern niemals vorkommen. Ihre Konklusion war: „Il n'y a pas d'hématurie essentielle. Toutes les hématuries sont symptomatiques, et relèvent d'une cause générale (toxique ou infectieuse) ou d'une affection locale."

Suter teilt uns einen sehr interessanten Fall mit, wobei klinisch nur einseitige renale Hämaturie konstatiert wurde. Bei der Operation schien die Niere makroskopisch normal zu sein. Wäre dieselbe also nicht exstirpiert, sondern reponiert, dann wäre auch dieser Fall diagnostiziert als essentielle Hämaturie, oder Nierenblutung aus gesunden Nieren. Bei der Untersuchung bemerkte man jedoch, daß das Nierenbecken krank war, besonders in der Umgebung der Ureteröffnung. Bei der mikroskopischen Untersuchung zeigte es sich, daß die Diagnose Angiomen, oder angiomatöse Entartung der Blasenschleimhaut war. Wie Suter uns mitteilt, ist in der Literatur nur sehr wenig betreffs dieser Läsion zu finden. Nur Fenwick[3]) beschreibt zwei derartige Fälle. Der erste seiner Patienten litt schon seit 5 Jahren an linksseitiger intermittierender Nierenblutung. Bei der Operation fand er im Nierenbecken, daß eine der Papillen umgeben war von einem Bündel Gefäße. Diese Papille wurde exstirpiert, mit dem Erfolg, daß der Patient geheilt war von seiner Hämaturie. Fenwick erzählt von der Untersuchung der exstirpierten Papille, daß hier nicht, wie er gedacht hatte, ein Papillom anwesend gewesen sei, „but that the vessels of the mucous membrane clothing the papilla were mark-

1) Suter, Über einseitige renale Hämaturie bedingt durch Teleangiektasien des Nierenbeckens. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1902.

2) Malherbe et Legueur, Il n'y a pas d'hématurie essentielle. Quatrième session de l'association française d'urologie. Paris 1900.

3) Fenwick, Renal papillectomy. British Medical Journal 1900.

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edly varicosed. There was no evidence of growth, but there was a congestion of the vessels with extravasation of blood and an increase in the cellular stroma. This might be an early stage of a fibromatous condition, such as is not uncommon in the tips of the papillae."

Bei der zweiten 30jährigen Patientin, welche schon seit 14 Tagen an heftigen Nierenblutungen litt, fand Fenwick in dem eröffneten Nierenbecken, daß die Spitze einer Papille rot und varicös war. Auch hier wurde diese Papille mit gutem Erfolg für den Patient exstirpiert. Hierüber sagt er: „The vessels in this part of the kidney are distended with blood, and there is some extravasation."

Diese Diagnose ist niemals zu stellen ohne Eröffnung des Nierenbeckens. Je nach der Ausbreitung des Prozesses kann man die Niere exstirpieren, oder nach Fenwick nur die kranke Papille entfernen.

Debersaques[1]) teilt uns noch einen Fall von essentieller Hämaturie mit. Sein Patient, ein 38jähriger Mann, der nie zuvor krank war, hatte schon seit 20 Jahren Schmerzen in der linken Nierengegend, und seit 8 Jahren auch Anfälle von Hämaturie, und einmal auch eine Nierenkolik. Wegen der großen Abmagerung des Patienten wurde Operation beschlossen. Die Niere wurde freigelegt, zeigte sich aber auch beim Sektionsschnitt, und bei Akupunktur vollkommen normal und wurde deshalb wieder reponiert. Nach der Operation blieben die Schmerzen und Blutungen während 3 Monaten fort. Daß dieser Fall ohne mikroskopische Untersuchung des Nierengewebes nicht viel Wert für uns hat, als Beweis für das Bestehen der essentiellen Nierenblutungen, spricht wohl von selbst.

Ebensowenig Wert für uns hat die durch Comby[2]) gestellte Diagnose in einem, von ihm beschriebenen Fall von Hämaturie.

Bei einem 5jährigen Mädchen trat Hämaturie auf, welche nach 3 Wochen Bettruhe und Milchdiät wieder verschwand. Da bei seiner Patientin andere Ursachen fehlten, und dieselbe behandelt war mit Karbolinjektionen im Ohr, sollte nun, wie Comby dachte, der Karbol wohl schuld haben an der Hämaturie. Auch Nitze äußert sich als ein großer Gegner des Begriffes essentieller Hämaturie, als er bei der Diskussion über den Vortrag von Klemperer sagte: „Es ist doch eine sonderbare Gesundheit eines Organs, wenn aus demselben eine

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1) Debersaques, Un cas d'hématurie rénale essentielle. Néphrotomie. Guérison. Annales de la Société Belge de Chirurgie 1898.

2) Comby, Dangers du phénol chez l'enfant; hématurie provoquée par des irrigations phéniquées de l'oreille chez une fillette od cinq ans. Société Médicale des Hôpitaux 1898.

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tödliche oder wenigstens gefahrdrohende zur höchsten Anämie führende Blutung erfolgt."

Solch ein Fall von tödlicher Hämaturie ohne bekannte Ursache teilt Spencer[1]) uns mit. Das Hauptsymptom war: increasing hæmaturia, wogegen nichts, auch nicht die Infusion, etwas vermochte. Auch die Sektion gab keine Erklärung von den Ursachen der tödlichen Hämaturie.

Auch verurteilte Nitze den Begriff essentielle Hämaturie, als er sagte: „Zurzeit ist für mich kein Unterschied in der Symptomatologie eines Tumors und Fälle von essentieller Hämaturie. Beide bluten oft kolossal, bei beiden pflegen die Kranken keinerlei Beschwerden zu haben; kurz die Symptomatologie ist völlig die gleiche und wird ganz durch die Blutung beherrscht."

Auch Nitze muß daher bei jeder einigermaßen langdauernden Hämaturie mit uns der Meinung sein, daß Freilegen der Nieren eine Notwendigkeit ist. Mit großem Mißtrauen muß sicher auch die eventuelle Diagnose hysterische Hämaturie begrüßt werden. Solche zweifelhafte Fälle sind u. a. beschrieben von Guisy[2]).

Harris[3]) beschreibt 2 Fälle von Hämaturie, wobei man keine Ursache derselben finden konnte, und welche man deshalb auch wohl zur hysterischen Hämaturie zählen konnte. Er sammelte noch 16 derartige Fälle aus der Literatur, auch um zu beweisen, daß in solchen Fällen die Nephrotomie stets Erfolg hat und daher man sich niemals für Nephrektomie entscheiden darf.

Hämaturie bei Nephritis.

Daß der Urin bei Nephritis Blut enthalten kann, war schon lange bekannt. Wir denken hierbei nicht an die chronische hämorrhagische Nephritis, von welcher Pel[4]) sagt: „Nebst der Anwesenheit von Ödemen und von mäßiger Hypertrophia cordis bildet hier der fast fortwährend stark, wohl immer schon mit unbewaffnetem Auge wahrnehmbar hämorrhagische Harn das klinische Kriterium."

Bei den nephritischen Zuständen, welche wir im Auge haben, fehlen Ödeme und Herzhypertrophie beinahe immer.

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1) Spencer, Fatal hæmaturia of unknown origin. Clinical Society of London. The British Medical Journal 1904.

2) Guisy, Trois cas d'hématurie hystérique. Annales des maladies des organes génito-urinaires 1901.

3) Harris, Renal hæmaturia without known lesions. Philadelphia medical Journal 1898.

4) Pel, Die akute und chronische Nierenentzündung. (Morbus Brightii.)

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Wir haben es dann mit dem zu tun, was Naunyn[1]) nennt: Massenblutung aus den Nieren bei Nephritis.

Sobald wir in der Literatur über diesen Gegenstand nachschlagen, so stoßen wir immer wieder auf den großen Mann der Nierenchirurgie J. Israel[2]).

Letzterer hat der Wissenschaft nicht allein auf dem Gebiete der Nierenchirurgie enorme Dienste geleistet, sondern er hat uns auch über diesen Gegenstand Klarheit gebracht.

Israel hat dabei auch bewiesen, daß Nephritis einseitig vorkommt.

Dennoch war er in dieser Hinsicht nicht der erste, sondern war uns eine Mitteilung betreffs der unilateralen Nephritis mit Hämaturie schon im Jahre 1897 durch de Keersmaecker[3]) gemacht.

Die Patientin von de Keersmaecker, eine 43jährige Frau, bemerkte im Dezember 1894 zuerst Blut in ihrem Urin, und zwar zunächst in der Form von langgestreckten Fäden geronnenen Blutes. Danach dauerte die Hämaturie ohne Unterbrechung fort. de Keersmaecker diagnostizierte erst Zystitis, kompliziert mit Hämaturie ohne bekannte Grundlage. Die zystoskopische Untersuchung der Blase ergab jedoch, daß dieselbe gesund war. Mit einem für diesen Fall neu konstruierten Ureterendoskop konstatierte er, daß der Urin, welcher von der linken Niere stammte, Blut enthielt, während der aus der rechten Niere frei von Blut war.

Die Hämaturie dauerte ungefähr 3 Jahre, und dann entschloß de Keersmaecker sich zur Nephrektomie. Bei der mikroskopischen Untersuchung der exstirpierten Niere ergab sich, daß diese nephritisch erkrankt war.

Die Blutung war in diesem Falle eine sehr heftige. Lécorché und Talamon[4]) glauben, daß Hämaturie bei Nephritis nie heftig sein könne; dieser Fall beweist, daß diese Ansicht fehlerhaft ist.

Bei der Diskussion über diesen Fall, sagte de Keersmaecker sehr richtig, daß unrechterweise unter den Ursachen der Haematuria renalis die Nephritis nicht genannt wird. Er im Gegenteil glaubt, daß sie in den meisten Fällen die Hauptursache ist, und das nun beobachtete unilaterale Auftreten der Nephritis sollte viele Bedenken gegen diese Auffassung eliminieren. Depage ist nicht von derselben

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1) Naunyn, Hämaturie aus normalen Nieren und bei Nephritis. Mitteilungen aus den Grenzgebieten der Med. und Chir. 1900.

2) Israel, Über den Einfluß der Nierenspaltung auf akute und chronische Krankheitsprozesse des Nierenparenchyms. Mitt. usw. 1900.

3) de Keersmaecker, Néphrite chronique unilatérale avec hématurie continue pendant deux ans et demi. Annales de la Société Belge de Chirurgie 1897.

4) Lecorché et Talamon, Traité de l'albuminurie.

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Ansicht, daß die Nephritis unilateral sein würde, weil der eiweißfreie Urin der anderen Niere kein entscheidender Beweis ist für das Intaktsein der anderen Niere.

Debaissieux, Daudois, Lambotte und Gallet brachen in dieser Diskussion noch eine Lanze für die essentielle Hämaturie, deren Dasein durch die Mitteilungen von de Keersmaecker ernstlich in Zweifel gebracht wurde.

Debaissieux beobachtete einen Fall von „hématurie rénale essentielle", wobei er die Niere vollkommen normal befand. Nur konstatierte er zwei kleine Knoten auf der Schleimhaut des Nierenbeckens. Man ist jedoch nicht berechtigt, eine solche Niere vollkommen normal zu nennen. Sehr wahrscheinlich ist es wohl, daß diese stecknadelkopfgroßen Knoten Tuberkel waren. Allein auch vor de Keersmaecker hat Crooke[1]) derartige Fälle von „Unilateral interstitional Nephritis" beschrieben.

In den Fällen von Crooke war die Ursache Druck auf den Ureter, und mikroskopisch fand er Läsionen „being histologically analogous, if not identical, with the changes found in that form of disease known as granular contracting kidney". In dem einen Fall war es eine Zyste, die bei einem 16jährigen Mädchen Druck verursachte, in dem andern ein Sarkom, das auf den linken Ureter Druck ausübte. In beiden Fällen, sagt Crooke, war die Niere an der anderen Seite „natural".

In der Diskussion über diese Mitteilungen sagte Carter, daß er einen ähnlichen Fall beobachtet hatte. In diesem Fall war es ein Aneurysma, das auf eine Niere Druck ausübte. Doran teilte dann mit, daß er eine Anzahl solcher Fälle gesehen habe.

Wie interessant die Mitteilung von Crooke auch ist, und wie sehr sie uns sicher eine Stütze sein kann für die Behauptung von Israel, daß einseitige Nephritis vorkommt, so ist doch der hier beschriebene Zustand so ganz anders, als der von den Fällen Israels, um die beiden auf eine Stufe zu stellen.

Senator[2]) protestiert gegen die Behauptung von Israel, daß einseitige Nephritis möglich wäre. Er äußert sich darüber so positiv wie möglich, indem er sagt: „Einen einseitigen Morbus Brightii gibt es nicht, es müßte denn nur eine Niere vorhanden sein."

Senator ist dabei gegen die Annahme, daß Nephritis die Ursache von Hämaturie sein soll, in Fällen, wenn nur einzelne kleine Herde

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1) Crooke, Unilateral interstitional Nephritis. Pathological Society of London. The Lancet 1889.

2) Senator, Nierenkolik, Nierenblutung und Nephritis. Deutsche med. Wochenschr. 1902.

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in der exstirpierten Niere gefunden wurden. Als Beispiel nennt er seinen durch uns zitierten Fall von renaler Hämophilie, der durch verschiedene Schriftsteller auf Grund der mikroskopischen Untersuchung von Prof. O. Israel als ein nephritischer Prozeß betrachtet wurde. Als Beweis teilt er uns in einer Anmerkung mit, daß bewußte Dame seit 12 Jahren total gesund und frei von Hämaturie ist. Diese Gesundheit spricht jedoch mehr für die frühere Anwesenheit eines nephritischen Prozesses, als für die einer Allgemeinerkrankung, wie die Hämophilie.

Israel[1]) sagt: „Wenn es somit feststeht, daß Einseitigkeit der Symptome kein Beweis für die Beschränkung der Erkrankung auf eine Niere ist, so darf man doch bei den Fällen, welche nach einseitiger Operation vollständig geheilt sind, annehmen, daß auch der Prozeß wirklich nur eine Seite betroffen hat."

Diese sehr deutlichen Worte dulden nicht viel Widerspruch, wenigstens, wenn man an einer Niere das Bestehen eines nephritischen Prozesses konstatiert hat. Weniger fest steht der Fall jedoch, wenn man die Ansicht Senators teilt, daß die Nephritis nur ein Symptom ist von einer Allgemeinerkrankung, m. a. W. mit ihm glaubt, daß die Nierenblutung ein Symptom einer Hämophilie ist.

Natürlich beantwortete Israel die obengenannten durch Senator geäußerten Bemerkungen.

Israel gibt allein zu, daß, weil Morbus Brightii nun einmal der historische Name für doppelseitige Nierenentzündung ist, ein einseitiger Morbus Brightii auch nicht existieren kann. Doch sehr richtig bemerkt er: „Damit soll aber nicht präjudiziert werden, daß nicht die anatomischen Veränderungen einseitiger Nierenaffektionen identisch sein können mit denen der doppelseitigen Brightschen Krankheit."

Zum Beweis von dieser Behauptung zitiert Israel einzelne Fälle aus der Literatur. An erster Stelle nennt er uns einen Fall von Rayer[2]). In diesem Falle starb ein 26jähriger Mann, nachdem er 3 Monate an Hämaturie gelitten hatte. Bei der Sektion fand man eine chronische Nephritis in der linken Niere, während die rechte Niere sich als normal erwies. Da hier jedoch die mikroskopische Untersuchung fehlt, so kann man diesen Fall nicht als Beweis anerkennen.

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1) Israel, Über den Einfluß der Nierenspaltung auf akute und chronische Krankheitsprozesse des Nierenparenchyms. Mitteilungen usw. 1900. Nierenkolik, Nierenblutung und Nephritis. Deutsche med. Wochenschr. 1902.

2) Rayer, Traité des maladies des reins. Paris 1841.

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Auch nennt Israel noch den schon zitierten Fall von de Keersmaecker; dann solche von Rovsing[1]), Pousson[2] und Hofbauer[3]).

In diesen vier Fällen hat man in der Tat zu tun mit einer interstitiellen Nephritis mit „Massenblutung", wobei nach Nephrektomie der kranken Niere der Patient sich vollkommen genesen zeigte, und der Urin kein Eiweiß oder Zylinder mehr enthielt. Doch kann man erwidern, daß die Nephritis verlaufen kann, ohne daß Eiweiß oder Zylinder in dem Urin gefunden werden, die Abwesenheit derselben nicht das Recht gibt, zu konkludieren, daß die nicht exstirpierte Niere gesund ist.

Senator sagt, daß bei Nephritis Eiweiß und Zylinder nicht anwesend zu sein brauchen, und daß diese Tatsache schon von Bright bekannt war, und daß sie bestätigt ist von Bartels und v. Cassel.

Vielleicht kann man auch den Fall von Dorst[4]) hier nennen.

Dorst ist mit uns der gleichen Ansicht, daß die sog. essentielle Hämaturie nur als ein Symptom zu betrachten ist und zwar als ein Symptom von chronischer Nephritis. In seinem Fall von scheinbar essentieller Hämaturie zeigte sich bei der mikroskopischen Untersuchung des entfernten Nierengewebes, daß eine Glomerulonephritis bestand. Später zeigte sich, daß der Urin kein Eiweiß und Zylinder mehr enthielt, so daß es sehr wahrscheinlich ist, daß die nephritische Niere durch den Einfluß des Sektionsschnittes genesen sei, und daß die andere Niere gesund war und blieb. Jedoch haben wir auch hier dieselbe Bemerkung, daß die Abwesenheit von Eiweiß und Zylinder nichts beweist betreffs der Gesundheit der Niere. Dasselbe gilt auch für den von Nicolich[5]) beobachteten Fall.

In diesem Fall wurde die Niere exstirpiert und von den Histologen in Wien als normal erklärt; erst eine spätere Untersuchung in dem Hospital Necker von Albarran und Motz zeigte das Bestehen einer Glomerulonephritis.

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1) Rovsing, Über unilaterale Hämaturie zweifelhaften Ursprungs und ihre Heilung durch Nephrotomie. Zentralbl. für die Krankheiten der Harn- und Sexualorgane 1898.

2) Pousson, De l'intervention chirurgicale dans les néphrites médicales. Annales des maladies des organes génito-urinaires 1902.

3) Hofbauer, Ein Fall von 2jähriger unilateraler Nierenblutung. Mitt. aus den Grenzgebieten der Med. und Chir. 1900.

4) Dorst, Een geval van pseudo-essentieele haematurie. Ned. Tijdschrift voor Geneeskunde 1902.

5) Nicolich, Sur un cas de néphrite hémorrhagique unilaterale. Comptes rendus de l'Association francaise d'urologie 1901.

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Auch Edebohl[1]) ist der Ansicht, daß eine unilaterale Nephritis möglich ist. Er hat 18 Fälle von Nephritis ausführlich beschrieben, und nur in 9 dieser Fälle waren beide Nieren erkrankt. In 4 Fällen war nur die linke, in 4 anderen Fällen nur die rechte Niere erkrankt.

Edebohl war nicht erstaunt, als er diese unilaterale Nephritis konstatierte. Wohl ist man daran gewöhnt, daß Nephritis fast immer beiderseitig vorkommt, aber das wahrscheinlichste ist doch, daß im allerersten Anfang der Krankheit nur eine der Nieren angegriffen ist. Die gesunde Niere, oder besser das gesunde Nierengewebe verrichtet dann auch die Arbeit für die erkrankten Teile. Die Krankheitsursache wirkt aber auch ein auf die bisher gesunde Niere, und in den meisten Fällen wird auch die zweite Niere krank, nur etwas später als die erst erkrankte Niere. Wenn man nephritische Nieren bei der Sektion beobachtet, dann findet man immer beide Nieren erkrankt, aber man muß nicht vergessen, daß man Nephritis im Anfang der Krankheit niemals zur Sektion bekommt; bei der Sektion sieht man nur weiter fortgeschrittene Fälle.

Edebohl meint, daß er durch seine große praktische Erfahrung imstande sei, makroskopisch konstatieren zu können, ob eine Nephritis anwesend ist oder nicht. Wie groß nun auch seine Erfahrung sein mag, so wird doch mancher, und mit Recht, an der Richtigkeit dieser allein makroskopisch gestellten Diagnose zweifeln. Die Schwierigkeit solch einer Diagnose wird illustriert durch den zitierten Fall von Nicolich.

In der Tat, Männern, wie Pousson und Albarran, ist es passiert, daß eine von ihnen bei makroskopischer Untersuchung für gesund gehaltene Niere bei der mikroskopischen Untersuchung sich als krank erwies.

Edebohl, obgleich voll Vertrauen zu seiner Diagnose, fühlt sich doch scheinbar nicht ganz sicher, wenigstens sagt er:

„In six of the eight cases in which chronic Bright's disease is recorded as unilateral, the healthy condition of the other kidney was verified at operation, when both kidneys were brought out of the wound for careful and critical examination. It cannot be denied that, in some instances at least microscopical examination of a kidney classed as healthy might have revealed evidences of incipient changes of an inflammatory character; to the unaided sight however, and to the touch, the organ was healthy."

1) Edebohl, The cure of chronic Brights disease by operation. Medical Record 1901.

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Später gibt Edebohl[1]) uns noch 72 Historiae morbi. Im ganzen konstatierte er in 11 Fällen unilaterale Nephritis. Er sagt: „That the diagnosis of chronic nephritis affecting one kidney only was made in each case upon the signs and characteristics presented upon inspection and palpation of the kidney at operation; that it is not without the pale of possibility that one or other kidney normal to sight and touch might on microscopic examination have presented evidences of slight or incipient inflammation."

Die Unterstützung, die Israel also von Edebohl erhält, ist allein von moralischem Werte, und als solche hat sie, in bezug auf die sehr großen Erfahrungen und Verdienste von Edebohl, für uns sehr große Bedeutung, ist aber nicht imstande, die Gegner, wie Senator, zu entwaffnen.

Israel[2]) selbst gibt die von Edebohl gemachten Bedenken zu, indem er sagt: „Der strikte Beweis der Einseitigkeit kann, streng genommen, nur durch die Sektion geführt werden."

Mit dieser Erkenntnis fällt nicht die große Wahrscheinlichkeit, wohl aber die Sicherheit, daß die so talentvoll gemachten Konklusionen von Israel richtig sind.

Israel behauptete: „Es gibt einseitige Nephritiden." Zum Glück jedoch hat er lange nach der Diskussion mit Senator von Stich[3]) den Beweis empfangen, daß seine These richtig sei.

Stich hat diesen Beweis geliefert mit der Beschreibung von einem von ihm beobachteten Fall von einseitiger chronischer diffuser Nephritis mit enormer Hämaturie.

Die Patientin von Stich, eine 30jährige Frau, erkrankte im März 1901 mit Symptomen von Hämaturie, wozu sich 2 Tage später heftige Schmerzen in der rechten Nierengegend gesellten. Zystoskopische Untersuchung war unmöglich, da nach Ausspülungen der Blase mit 8 l 1%iger Borsäure die wegfließende Flüssigkeit stets rot gefärbt blieb. Nach einigen Tagen kam allerdings ein Blutkoagulum zum Vorschein, das ein getreuer Abguß des Nierenbeckens und Ureters war, so daß die Tatsache, daß man mit einer Nierenblutung zu tun hatte,

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1) Edebohl, The histories of seventy-two patients operated upon by the autor for chronic Bright's disease up to the end of the year 1903. The surgical treatment of Bright's disease 1904; Questions of priority in the surgical treatment of chronic Bright's disease. Medical Record 1902; Renal decapsulation for chronic Bright's disease. Medical Record 1903.

2) Israel, Nierenkolik, Nierenblutung und Nephritis. Deutsche med. Wochenschr. 1902.

3) Stich, Über Massenblutungen aus gesunden und kranken Nieren. Mitt. aus den Grenzgebieten der Med. und Chir. 1904.

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allein dadurch schon feststand. Am 6. April starb die Patientin. Bei der Sektion konstatierte man eine doppelseitige akute Pyelonephritis. Die rechte Niere dagegen bot, abgesehen von diesen akuten Veränderungen, das ausgesprochene Bild einer chronischen parenchymatösen Nephritis.

Natürlich stehen diese Veränderungen nicht im kausalen Verband mit den Abszessen, die in beiden Nieren gefunden wurden, infolge des akuten Prozesses.

Castaigne und Rathery teilen auch einzelne Fälle von einseitiger Nephritis in der Semaine Médicale 1902 mit, doch ihre drei Beobachtungen betrafen eiternde Nephritis, so daß sie nicht mit dem Fall von Stich zu vergleichen sind. In der Tat ist der Fall von Stich ein Unikum, jedoch der Name Stich, ein bekannter ernster Untersucher, genügt, um ihn als Wahrheit zu betrachten. Ich habe in der Literatur vergebens nach einem zweiten gleich beweiskräftigen Fall gesucht, doch dieser eine macht die Konklusion von Israel, trotz der Bekämpfung von Männern wie Senator, glaubwürdig.

Während ihres Lebens war bei der Patientin von Stich die chronische Nephritis nicht zu diagnostizieren, da alle typischen Symptome davon fehlten. Daß die gefundenen chronischen Veränderungen hier nicht eine Folge waren von dem Übergang des akuten Prozesses in einen chronischen Zustand, ist schon daraus deutlich, daß die ganze Krankheit der Frau nur 3 Wochen gedauert hatte.

Alle anderen möglichen Ursachen für die Nierenblutung und Nierenkolik waren hier, wie Stich mitteilt, auszuschließen. Vollkommen richtig macht Stich daher diese Folgerung. In der großen Mehrzahl der Fälle soll Morbus Brightii bilateral sein, aber sicher ist es, daß es Fälle gibt, die, klinisch durch Hämaturie und Koliken ausgezeichnet, anatomisch auf der kranken Seite das Bild einer diffusen chronischen, parenchymatösen und interstitiellen Nephritis darbieten, während die andere Niere normal ist.

Mit dieser Bestätigung von Stich ist also die geniale Arbeit von Israel als eine neue Tatsache zu betrachten; wir Mediziner wissen nun alle, daß es unzweifelbar einseitige Nephritis gibt.

Wenn wir langdauernde einseitige Hämaturie beobachteten, so wurde früher nicht an Nephritis gedacht, sowohl weil Massenblutungen nur äußerst selten dabei vorkamen und auch weil die Einseitigkeit der Symptome ein wichtiges Argument gegen diese Diagnose war. Doch dank Stich und vor allem dank der scharfen Beobachtung von Israel wissen wir jetzt besser. Die sechste These von Israel ist: „Es gibt Nephritiden mit anfallsweise auftretenden profusen Blutungen.“ Senator sagt betreffs dieser Mitteilung, daß sie zwar richtig, aber schon lange

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bekannt war. Richtig sagt Israel, daß einem so belesenen Manne wie Senator wohl alles bekannt sein wird, was in der Literatur zu finden ist, aber daß diese These gewiß der großen Mehrzahl der Mediziner neu sein wird. Israel erinnert uns dabei an das berühmte Lehrbuch von Rosenstein, worin man findet: „Fast niemals aber ist in den chronischen Formen (der diffusen Nephritis) so viel Blut beigemengt, daß der Urin tiefrot wird, meist zeigt er auch dann nur Fleischwasserfarbe."

Auch in dem Lehrbuche von Pel: „Die akute und chronische Nierenentzündung (Morbus Brightii)" wird das intermittierende Auftreten von profusen Nierenblutungen nicht erwähnt. Pel sagt über die chronisch diffuse Nephritis, daß die hämorrhagische Form zu erkennen ist an dem fast fortwährend stark hämorrhagischen Harn.

Obwohl Senator zugibt, daß bei Nephritis Blutungen vorkommen können, so will er doch nicht die Nephritis als Ursache der Hämaturie betrachten, weil diese notfalls auch andere Ursachen haben könnte.

Als Beweis hierfür bezieht Senator sich auf den bekannten Fall von Braatz[1]). Braatz legte eine Niere frei wegen Nephralgie, verrichtete auch den Sektionsschnitt und reponierte dann wieder die Niere, weil es sich zeigte, daß diese Niere normal war. 3 Jahre später exstirpierte er diese Niere, da die erst verschwundene Nephralgie zurückgekehrt war. An dieser Niere konnte er nun einen tuberkulösen Abszeß konstatieren, sowie tuberkulöse Eruptionen. Außerdem fand er aber auch einen alten, schon geheilten tuberkulösen Herd, der wahrscheinlich die Ursache war der früheren Nephralgie. Nun könnte man Senators Worte zitieren: „Dies ist gleichfalls richtig, aber gleichfalls bekannt."

Wir wissen doch schon lange, daß es sehr wohl möglich ist, bei der makroskopischen, ja selbst bei der mikroskopischen Untersuchung eines Teiles des Nierengewebes, daß eine Nephritis nicht bemerkt wird. Senator erkennt den kausalen Verband zwischen Nephritis und Hämaturie und doch will er die letztere zuschreiben einer unbekannten Ursache, auch in Fällen, wobei eine Nephritis konstatiert ist. Damit kann gewiß niemand einverstanden sein.

Durch die Untersuchungen von Israel haben die Begriffe essentielle und angioneurotische Hämaturie, sowie renale Hämophilie keinen praktischen, sondern nur einen historischen Wert. Sehr richtig doch sagt Israel, daß, wenn Senator leugnet, daß die negative mikroskopische Untersuchung eines ausgeschnittenen Stückes Nierengewebes

1) Braatz, Über operative Spaltung der Niere. Deutsche med. Wochenschr. 1900.

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das Recht gibt, zu konkludieren, daß die nephritischen Veränderungen in einem anderen Teil der Niere anwesend sind, diesen negativen Resultaten der Untersuchung gewiß nicht das Recht gibt, die ganze Niere für normal zu erklären.

Die ferneren Folgerungen von Israel sind:

Es gibt durch Nephritis erzeugte Nierenkoliken, welche Nierensteinkoliken gleichen.

Es gibt doppelseitige Nephritiden, welche nur einseitige Kolik erzeugen. Ein solcher Fall ist mitgeteilt von Martens[1]) in der Freien Vereinigung der Chirurgen Berlins.

Es gibt schwere Nephritiden mit eiweißfreiem Urin und Abwesenheit von Zylindern.

Trotz großen Reichtums an hyalinen, gekörnten und epithelialen Zylindern kann der Urin eiweißfrei sein.

Nephritische Blutungen können mit oder ohne Koliken eintreten oder verlaufen. Die Blutung ist nicht die Ursache der Kolik. Beide Erscheinungen sind Folgezustände der Nierenkongestion.

Eine große Zahl der bisher als Nephralgie, Néphralgie hématurique, angioneurotische Nierenblutung bezeichneten Krankheitsbilder sind auf nephritische Prozesse zu beziehen.

Senator ist besonders nicht einig mit der Behauptung, daß in diesen Fällen Nierenkongestion Ursache der Nierenblutung und Kolik sei. Erstens macht er darauf aufmerksam, daß klinisch so oft Nierenkongestion konstatiert wurde, z. B. bei akuter Nephritis, Kompensationsstörungen usw., ohne daß dabei Nierenblutungen und Koliken auftreten. Ferner weist er uns darauf, daß die Historia morbi der Fälle von Israel nicht spricht für die Anwesenheit einer Nierenkongestion, da wir in den meisten Fällen die Niere beschrieben finden als „auffallend klein und weich", oder auch „die Niere zeigt keine Abweichung in bezug auf Größe und Konsistenz".

Israel beantwortet diese Anfechtung seiner Thesen mit der Bemerkung, daß seine Fälle nicht die einzigen sind, welche seine Behauptungen beweisen, sondern daß die schon zitierten Fälle von Pousson, Potherat, Albarran, de Keersmaecker, Harrison und Schede auch dazu dienen können.

Die Hauptsache ist aber, daß Israel niemals behauptet hat, daß fortdauernde Zustände von Spannungserhöhung in seinen Fällen anwesend sein müssen. Er sagt: „Vielmehr habe ich, was ja schon in

1) Martens, Über einen Fall von chronischer Nephritis mit häufiger auftretender einseitiger Nierenkolik und Nierenblutung. Deutsche med. Wochenschr. 1902.

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dem Worte „Kongestion“ implicite liegt, von plötzlich auftretenden und verschwindenden Fluxionen mit renaler Spannungszunahme gesprochen. Wenn daher der Kongestionszustand nicht bei der Operation gefunden wird, so darf Senator nicht daraus schließen, daß er nicht bestanden hat. Daß er in der Tat anwesend ist und gefunden wird, wenn man zur richtigen Zeit operiert, beweist der dritte Fall von Israel, wobei die Niere „auffallend resistent blau“ war und alle Symptome „intensiver Spannung und Kongestion“ zeigte.

Senator ist auch nicht der Ansicht Israels, daß fast alle Fälle von essentieller Hämaturie wirklich die Folge von nephritischen Prozessen seien.

Senator sagt, daß man seine beiden Fälle und den Fall von Sabatier unrichtigerweise für Nephritis angesehen hat. Doch Israel sagt mit Recht, daß diese drei Fälle nicht die einzigen seien, worauf er sich berufen kann, sondern daß er auch noch nennen kann die Fälle von Hofbauer[1]), Pousson[2]), Poirier[3]), Desmons[4]), Peau[5]) und Schede[6]).

In der Klinik von Professor Lichtheim zu Königsberg sind noch eine Serie von 11 Fällen von chronischer Nephritis beobachtet, in deren Verlauf „Massenblutungen“ mit und ohne Nierenschmerzen vorgekommen sind.

Diese Fälle sind beschrieben von Askanazy[7]).

In 3 von diesen Fällen ist die Diagnose Nephritis durch die Autopsie bestätigt. In den anderen 8 Fällen war der klinische Verlauf und die Urinuntersuchung derartig, daß an der Diagnose nicht gezweifelt werden konnte.

Der interessanteste Fall von Askanazy ist sicher der zehnte. Bei einem 34jährigen Manne, der an einem diabetischen Koma verschied, war niemals Eiweiß in dem Urin zu konstatieren. Bei diesem Diabetiker deutete die Untersuchung des Urins in keiner Weise darauf hin, daß eine Nephritis vorlag. Während des Komas trat eine starke Hämat-

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1) Hofbauer, Ein Fall von 2jähriger unilateraler Nierenblutung. Mitt. aus den Grenzgebieten der Med. und Chir. 1900.

2) Pousson, De l'intervention chirurgicale dans les néphrites médicales. Annales des maladies des organes génito-urinaires 1902.

3) Poirier, Bull. de la Soc. de Chir. 10. mai 1899.

4) Desmons, Association française de chirurgie. Congrès 1898.

5) Brodeur, De l'intervention chirurgicale dans les affections du rein.

6) Schede, Neue Erfahrungen über Nierenexstirpationen. Jahrbuch des Hamburger Stadtkrankenhauses 1889.

7) Askanazy, Profuse Hämaturien und kolikartige Schmerzen bei Nephritis. Zeitschr. f. klin. Med. 1906, Bd. 58.

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urie auf, und bei der Sektion zeigte sich eine parenchymatöse Nephritis, die klinisch völlig symptomlos verlaufen war. Ohne Autopsie wäre dieser Fall vielleicht zu den essentiellen Hämaturien gerechnet worden, oder vielleicht hätte man daraus gefolgert, daß dergleichen Massenblutungen auch bei Diabetes vorkommen.

In einem der Fälle von Askanazy wurde als Ursache angegeben, daß der Patient sich auf der Eisbahn erkältet hätte. Wie wir gesehen haben, wurde Erkältung als Ursache der Hämaturie auch genannt von Lewitt, Schede, Stich, Socoloff u. a.

In dem elften Fall wurde das Aufheben von einem schweren Sack Kartoffeln als Ursache genannt.

In einigen Fällen trat die Hämaturie ohne, in anderen wieder mit heftigen Nierenschmerzen auf. Manchmal irradiierten die Schmerzen in den Testikel oder in den Penis, was ja auch von Israel beobachtet ist und von ihm beschrieben ist in seiner „Chirurgische Klinik der Nierenkrankheiten".

Auch Askanazy gibt zu, daß die Hämaturie bei Nephritis nichts Eigentümliches hat, so daß man nicht daraus die Diagnose Nephritis stellen kann. Wohl konkludiert er aus der Art des Auftretens, daß wir dabei sicher nicht zu tun haben mit einer akuten Exazerbation von einem chronischen Prozeß. Fieber und Hydrops sieht man doch nur äußerst selten in diesen Fällen, und auch ist das meist einseitige Auftreten der Blutungen nicht in Übereinstimmung mit dem Gedanken an eine akute Exazerbation von einer doppelseitigen chronischen Nephritis. Doch außerdem fehlen in diesen Fällen die schweren Allgemeinerscheinungen während der Attacken, wie sie bei akuten Nephritiden kaum je vermißt werden dürfen, besonders auch die Symptome urämischen Charakters.

Askanazy ist ein Anhänger von Israel, was anbelangt dessen Meinungen über die Ursachen der Hämaturie bei Nephritis. Er sagt: „Wir sind mit Israel der Ansicht, daß Hämaturien und kolikartige Schmerzen bei nephritischen Prozessen auf dieselbe Ursache und zwar auf paroxysmale Kongestionen zurückzuführen sind."

Askanazy hat noch eine sehr interessante Beobachtung gemacht, die eine sehr kräftige Stütze ist für die Kongestionstheorie von Israel.

Er selbst fand in den zur Sektion gekommenen Fällen Kongestionen und Ekchymosen im Nierenbecken, wie ja auch vielfach von Guyon, Poirier, Naunyn, Pousson u. a. gefunden war. Dadurch kam er auf den Gedanken, in den Sektionsprotokollen nachzuforschen, ob diese Veränderungen auch zu konstatieren waren in den Fällen, welche ohne Hämaturie, ohne Massenblutungen verlaufen waren. Nun zeigte es sich ihm, daß sich Blutungen auf der Nierenbeckenschleimhaut bei

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Nephritis relativ oft, sicherlich in ¼ aller Fälle, vorfinden. Er bespricht danach, daß diese Ekchymosen nur entstehen können durch venöse Stasis, Entzündung und Kongestion, und kommt dann zu dem Satz, daß die Ekchymosen bei Nephritis als Residuen voraufgegangener Kongestionen aufzufassen und auch die Massenblutungen bei Nephritikern auf die nämliche Ursache zurückzuführen sind.

Die erste Behauptung Israels ist somit eine feststehende Tatsache geworden durch die Mitteilung von Stich, und die anderen genannten Thesen hat er ruhmvoll selbst verteidigt gegen die Anfälle von Senator und anderen. Auch seine neunte Konklusion: „Die Inzision der Niere beeinflußt in vielen Fällen den nephritischen Prozeß und seine Symptome günstig", ist richtig. Mit der Besprechung dieser Konklusion kommen wir vollauf in das Gebiet der Nierenchirurgie. Der große Freund der chirurgischen Eingriffe bei Morbus Brightii ist jedoch nicht so sehr Israel, als Edebohl. Harrison[1]) war jedoch der erste, der bei Nephritis operierte, sei es auch, ohne es zu wollen. Er suchte nach Eiter, ohne diesen zu finden, und sagt: „This was not the case, I closed the proceeding with the feeling, that I had made an error in diagnosis." Trotz der unrichtigen Diagnose heilte der Patient schnell nach der Operation von seinen früheren Beschwerden, ebenso wie von der Operation selbst. Die früheren Beschwerden waren Nierenschmerzen und Albuminurie. Der zweite Patient von Harrison wurde operiert wegen Nierenstein, welche Diagnose gestellt wurde wegen Hämaturie, Kolikschmerzen und Albuminurie. Kein Stein wurde gefunden, aber doch war der Patient wieder ganz gesund nach der Operation. Auch der dritte Patient von Harrison wurde operiert wegen Nierenstein, „but no calculus could be discovered". Jedoch genas auch dieser Patient vollkommen.

Es würde hier zuweit führen, das Für und Wider des chirurgischen Eingriffes bei Nephritis näher zu besprechen. Natürlich haben die Gegner so wie Pel nicht zu hohe Erwartungen. Pel sagt auch noch, daß die Hämaturie selbst schon eine Verminderung der Nierenspannung verursacht, doch vergißt er dabei, daß die Nierenblutung nicht gleich zu stellen ist mit einem chirurgischen Eingriff, und außerdem schreitet man oft zur Operation, um die gefährliche Hämaturie zu bestreiten.

Es sei genug, außer Harrison noch einige Namen von Männern

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1) Harrison, On the treatment of some forms of albuminuria bi reni-punctura. The British Medical Journal 1896; Renal tension and its treatment by surgical means. The British Medical Journal 1901; A contribution to the study of some forms of albuminuria associated with kidney-tension and their treatment. The Lancet 1896.

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zu nennen, die sich auf diesem Gebiete verdienstlich gemacht haben ohne zu behaupten, daß nicht vielleicht noch mehr sind, welche nicht genannt wurden. So nennen wir: Israel[1]), Edebohl[2]), Pousson[3]), Ferguson[4]), Mongour[5]), Scheben[6]), Wolff[7]), Caillé[8]), Guitéras[9]), Stern[10]), Nouëne[11]), Henry[12]), Korteweg[13]), Wendel[14]), Bakes[15]), Rovsing([16]), Herxheimer und Walker Hall[17]) u. a.

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1) Israel, Über den Einfluß der Nierenspaltung auf akute und chronische Krankheitsprozesse des Nierenparenchyms. Mitteilungen aus den Grenzgebieten der Med. und Chir. 1901.

2) Edebohl, The cure of chronic Brights disease by operation. Medical Record 1901; Surgical treatment of Bright's disease. 1904.

3) Pousson, De l'intervention chirurgicale dans certaines variétés de néphrites médicales. Association française d'urologie 1899; De l'intervention chirurgicale dans les néphrites infectieuses aigues et dans les néphrites chroniques. Revue de Chirurgie 1901; Contribution à la physiologie pathologique de l'incision et de l'exstirpation du rein. Annales des maladies des organes génito-urinaires 1901; Discussion sur la néphrotomie dans les néphrites médicales. Gazette hebdomadaire des sciences médicales de Bordeaux 1902; De l'intervention chirurgicale dans les néphrites médicales. Annales des maladies des organes génito-urinaires 1902.

4) Ferguson, Surgical treatment of nephritis or Bright's disease. Medical Standard 1899.

5) Mongour, De la néphrotomie dans les néphrites médicales chroniques. Journal de Médecine de Bordeaux.

6) Scheben, Beitrag zur Wirkungsweise der Edebohlschen Operation. Münchner med. Wochenschr. 1906.

7) Wolff, Über die Erfolge der Nephrorrhaphie auf Grund der nach dem Verfahren von Herrn Professor Rose in Bethanien operierten Fälle. Deutsche Zeitschr. f. Chir. 1897.

8) Caillé, Chronic parenchymatosis nephritis in a child treated by renal decapsulation. Arch. of Pediatrics 1902.

9) Guitéras, The surgical treatment of Bright's disease. New York medical Journal 1902.

10) Stern, Beitrag zur Frage der chirurgischen Behandlung der chronischen Nephritis. 75. Versammlung der deutschen Naturf. und Ärzte 1903, Cassel.

11) Nouëne, Traitement chirurgicale des néphrites. Thèse Paris de 1903.

12) Henry, Nephropexy in a case of chronic nephritis. American Journal of the med. science 1903.

13) Korteweg, Die Indikationen zur Entspannungsinzision bei Nierenleiden. Mitt. aus den Grenzgebieten der Med. und Chir. 1901.

14) Wendel, Die Entwicklung der Nierenchirurgie in den letzten Jahren. Therapeutische Monatsh. 1899.

15) Bakes, Ein neues Verfahren zur operativen Therapie der chronischen Nephritis. Zentralbl. f. Chir. 1904.

16) Rovsing, Zur Behandlung der chronischen Morbus Brightii durch Nephrolysis und Nephrokapsektomie. Zentralbl. f. Chir. 1904.

17) Herxheimer und Walker Hall, Über die Entkapselung der Niere. Virchows Arch. 1905.

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Diagnose.

Der erste Fall ist in vieler Hinsicht interessant.

Wir sehen hier plötzlich ohne frühere Symptome Hämaturie in sehr heftiger Weise auftreten und jeder Behandlung trotzen. Dazu kam nur heftige Nierenkolik, weiter war nichts zu finden. Es war also ein klassischer Fall von essentieller Hämaturie oder Klempererscher Nierenblutungen aus gesunden Nieren. Dann konstatierte man einen Nierentumor, der sich erwies als eine Anhäufung von Fäzes. Wahrscheinlich war diese Koprostase entstanden unter dem Einfluß der Nierenkolik. Jenner doch sagt: „Nephritic colic will cause loss of power in the colon, and so induce constipation, thus favouring the idea that the patient has intestinal colic."

Nun jedoch war das bisher dunkle Krankheitsbild deutlich geworden. Eiweiß und Zylinder gaben das Recht, die Diagnose auf Nephritis zu stellen. Mit diesem Falle ist auch die Ansicht von Lecorché und Talamon[1]) widerlegt, daß Hämaturie bei Nephritis nicht heftig sein könne.

Soweit ist dieser Fall ein Analogon von dem von Schüler[2]). In diesem Fall bestand bei einer 49jährigen Frau seit 10 Monaten Hämaturie. Die freigelegte rechte Niere, woraus die Blutung stammte, war makroskopisch und auf dem Sektionsschnitt vollkommen normal, aber die mikroskopische Untersuchung von einem exstirpierten kleinen Stück Nierengewebes bewies die Anwesenheit einer Nephritis.

Überdies aber gab die Patientin Zustimmung, sich die Ureter katheterisieren zu lassen, und nun zeigte sich, daß Eiweiß und Zylinder nur aus der Niere, woraus auch das Blut kam, stammten.

Hiermit ist, sei es auch nicht mit absoluter Sicherheit, das Bestehen der einseitigen Nephritis bewiesen. An der absoluten Gewißheit fehlt, daß wir die Einwendung zugeben müssen, daß die Abwesenheit von Eiweiß und Zylinder in dem Urin der anderen Niere nicht sicher die Gesundheit derselben beweist. Die Wahrscheinlichkeit ist jedoch groß, daß es hier der Fall ist.

Auch in dem zweiten Fall ist unsere Diagnose Nephritis. Das eigenartige von dem Fall war, daß hier zyklische Albuminurie bestand, was in Zusammenhang mit der gleichzeitigen Anwesenheit von Zylindern ein Beweis ist, daß zyklische Albuminurie und Nephritis zusammengehören, wie Henoch, Heubner u. a. schon behaupteten.

Unglücklicherweise wollte der Patient sich nicht einer zystoskopi-

1) Lecorché et Talamon, Traité de l'albuminurie. 1888.

2) Schüler, Beitrag zur Lehre von den Blutungen aus anscheinend unveränderten Nieren. Wiener klin. Wochenschr. 1904.

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schen Untersuchung unterwerfen, wodurch nicht zu konstatieren war, ob die Hämaturie ein- oder doppelseitig war.

Morris passierte es, daß er die Doppelseitigkeit der Hämaturie erst nach der Exstirpation der einen Niere konstatierte, wonach die Blutung fortdauerte und der Patient sukkumbierte.

Der dritte Fall endete als eine gewöhnliche hämorrhagische Nephritis. Das wichtige von diesem Falle war aber das Debütieren unter dem ausgeprägten Bilde einer essentiellen Hämaturie. Wahrscheinlich bestanden hier im Anfang schon geringe nephritische Veränderungen in einer Niere. Später änderte sich das Bild. Akute Exazerbation des Leidens führte zu einer doppelseitigen hämorrhagischen Nephritis.

Auch der vierte Fall kennzeichnet sich durch das Debütieren unter dem Bilde einer essentiellen Hämaturie. Später, viel später erst bemerkte man das Bestehen von Tuberkulose von Blase und Niere. Die Untersuchung ergab, daß nur eine Niere erkrankt war, und wegen der Richtigkeit der Worte Zuckerkandls[1]): „Noch schwerer aber fällt ins Gewicht, daß bei langer Dauer des Prozesses die andere Niere niemals intakt bleibt", wurde das Exstirpieren der kranken Niere natürlich in Beratung genommen, aber wegen der gleichzeitig anwesenden Blasentuberkulose nicht verrichtet.

Auch hier werden wahrscheinlich in der ersten Zeit des Leidens, als Hämaturie noch das einzige Symptom war, wohl schon tuberkulöse Veränderungen in der Niere bestanden haben, und ist dieser Fall daher ein warnendes Beispiel, ja stets dem Rat Israels zu folgen und bei jeder lange dauernden Hämaturie ohne bekannte Ursache die Niere frei zu legen zur richtigen Diagnostik.

Doch sind auch dann Irrtümer nicht immer zu vermeiden, wie ein durch Israel selbst mitgeteilter Fall beweist.

Israel[2]) beschreibt einen Fall von Carcinoma renis, wobei er auch aufmerksam macht auf die Schwierigkeit der Diagnose eines Nierentumors wegen der Eigentümlichkeit des infiltrierten Karzinoms, keine Formveränderung, keine Vergrößerung der Niere erkennen zu lassen. Die linke Niere war hier angegriffen von Karzinom, aber aus der rechten Niere stammte eine Hämaturie, verursacht durch einen da anwesenden Nierenstein. Die zystoskopische Untersuchung hätte hier gewiß nicht die karzinomatöse, sondern die gesunde Niere, worin der Nierenstein, zur Operation angewiesen. Wir müssen daher nie vergessen, falls wir eine plausibele Ursache für die Hämaturie gefunden

1) Zuckerkandl, Über die Behandlung der Nierentuberkulose. Deutsche med. Wochenschr. 1906.

2) Israel, Erfahrungen über Nierenchirurgie. Arch. f. klin. Chir. 1894.

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haben, daran zu denken, ob nicht vielleicht ein anderes Nierenleiden Veranlassung derselben war.

Kümmel[1]) gab auf dem Kongreß der Deutschen Gesellschaft für Chirurgie im Jahre 1904 den Rat, in Fällen, wo Nierentuberkulose einseitig ist, die kranke Niere zu exstirpieren trotz der gleicherzeit bestehenden Blasentuberkulose. Eine einmal vorhandene Blasentuberkulose heilt man am besten durch Entfernung der die Infektion veranlassenden Niere. Eine Blasentuberkulose sollte man niemals operieren, dagegen die die Infektion veranlassende Niere möglichst früh entfernen.

Konklusionen.

1. Es gibt keine essentielle Hämaturie.

2. Renale Hämaturie muß stets als Symptom einer Nierenaffektion betrachtet werden.

3. Von den hier gemeinten Nierenaffektionen kommt in erster Linie Nephritis in Betracht. Die Hämaturie kann dabei sehr profus sein.

4. Einseitige Nephritis existiert nicht nur, sondern sie ist aller Wahrscheinlichkeit nach im Anfangsstadium immer einseitig.

5. Eine Nephritis kann schon längere Zeit bestehen, ohne daß der Urin Zylinder oder Eiweißspuren aufweist.

6. Auch bei Nephritis treten Nierenkoliken auf, die den Nierensteinkoliken nicht in Heftigkeit nachstehen.

7. In jedem einzelnen Fall von langwieriger Hämaturie schreite man nach einer in jeder Hinsicht möglichst sorgfältigen Untersuchung zur Freilegung und Spaltung der Niere zur Feststellung der Diagnose. Sehr richtig sagt Grosglik: „Die chirurgische Behandlung aller dunklen Nierenblutungen schützt uns zweifellos vor unangenehmen Enttäuschungen, welchen wir bei exspektativem Verfahren ausgesetzt sein können."

8. Und aus noch einem weiteren Grunde schreite man zur vorerwähnten Operation, und zwar, weil die Erfahrung uns gelehrt hat, daß sich das Bild der Nephritis zufolge der Nierenspaltung in günstigem Sinne ändert.

9. Exstirpation der Niere soll möglichst umgangen werden. Richtig ist das Urteil Nepveus[2]) zu dieser Operation: Cette opération (Néphrectomie) aurait pu être évitée dans la plupart des cas où elle est pratiquée. Elle aurait pu être remplacée par des opérations plus sures et tout aussi bonnes. Jusqu'ici cette opération nous semble devoir être condamnée par la saine critique et par l'art.

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1) Kümmel, Die Frühoperation der Nierentuberkulose. Verhandlungen der deutschen Gesellschaft für Chirurgie. Kongreß 1904. Zentralbl. f. Chir. 1904.

2) Nepveu, De l'exstirpation du rein. Archives générales de Médecine 1875.

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10. Schreitet man trotzdem zur Exstirpation, so darf diese nur stattfinden nach vorheriger kryoskopischer Untersuchung.

Achard[1]) empfiehlt hierfür folgende Methode: Eine subkutane Injektion mit 0,05 g Methylenblau. Bei normal funktionierenden Nieren nimmt die Ausscheidung bereits nach 15—30 Minuten ihren Anfang, während innerhalb 35—60 Stunden alles entfernt ist. Ist dieses jedoch erst innerhalb 3—4 Tagen der Fall, so besteht Niereninsuffizienz.

11. Trotzdem verlasse man sich nie ausschließlich auf eine, wenn auch noch so vollständige kryoskopische Untersuchung. Der Stockmannsche Fall doch beweist den Stockmannschen Ausspruch: „Die Methode stimmt im allgemeinen, aber nicht im besonderen."

Außerdem nehme man die kryoskopische Untersuchung erst vor, nachdem die Diagnostizierung nach älteren Untersuchungsmethoden stattgefunden hat. Wiener sagt sogar in The New York Medical Journal vom 9. März 1901 (Blood in the Urin as a symptom and the Diagnosis of its source), daß man in dem Falle die Kryoskopie nicht nötig hat.

Der 48jährige Patient von Stockmann[2]) hatte heftige Schmerzen in der rechten Lendengegend und der Urin enthielt sowohl Eiter und Blut, als auch Blasenepithelien. Zystoskopie war in diesem Falle unmöglich. Der wachsenden Beschwerden wegen, zu denen sich auch heftiges Erbrechen gesellte, beschloß man die Exstirpation der rechten Niere, da die Diagnose rechtsseitigen Nierenstein oder Tuberkulose ergeben hatte. Da die Gefrierpunktsbestimmung des Blutes die normale Ziffer 0556 ergab, durfte man voraussetzen, daß die andere Niere richtig funktionierte. Die Operation fand jedoch nicht statt, da der Patient starb. Die Sektion ergab das Fehlen der rechten Niere, während die linke Niere sehr erweitert und tuberkulös war. Die anwesende Niere hatte natürlich bereits seit vielen Jahren die fehlende ersetzt und, seit der tuberkulösen Affektion, hatten die gesunden Gewebeteile die Arbeit des erkrankten Gewebes übernommen. Somit war der Gleichgewichtszustand trotz des tuberkulösen Prozesses nicht gestört. Dieser Fall stimmt also vollständig überein mit dem Tierexperiment von Tuffier, Wolff und Kümmel, nämlich daß man allmählich eine Niere ganz und von der anderen einen $^3/_4$ Teil entfernen kann, ohne daß dadurch der Tod des Tieres eintritt. Eine normale Gefrierpunktsziffer des Blutes bedingt also nur einen Gleichgewichtszustand, weiter nichts.

1) Achard, Die Diagnose der funktionellen Nierenstörungen. Prager med. Wochenschr. 1901.

2) Stockmann, Ist die Gefrierpunktsbestimmung des Blutes ein ausschlaggebendes Hilfsmittel für die Nierenchirurgie? Monatsber. f. Urologie 1902.

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504/07.

(Innere Medizin Nr. 149/52.)

Die klinische Diagnose der Pulmonalarteriensklerose.

Von

Privatdozent Dr. **Adolf Posselt,**

Innsbruck.

In der Pathologie der Pulmonalarterie im weiteren Sinne zieht der Kliniker in den Kreis diagnostischer Erwägung bestimmte angeborene Bildungsanomalien, Klappenfehler, Stenosen- und Aneurysmabildung, wobei seit je die außerordentlichen Schwierigkeiten der Diagnose betont werden, daher auch die so häufigen Fehldiagnosen leicht erklärbar sind.

Die Sklerose der Pulmonalarterie betrachtete man als Domäne des pathologischen Anatomen.

Zur Illustrierung der großen Seltenheit des Befundes findet man immer wieder die Häufigkeitsskalen der Arteriosklerose in den verschiedenen Gefäßregionen von Lobstein, Rokitansky, Huchard zitiert.

Die Atherosklerose der Lungenschlagader[1]) kann als primäres Leiden oder als sekundäre Erscheinung auftreten.

Von ersterem figurieren in allen Handbüchern, Herzpathologien und sonstigen Berichten stereotyp nur die Beobachtungen von Klob, Romberg und Aust (obwohl, wie hier nur nebenbei bemerkt sei, eine nicht zu unterschätzende, allerdings schwer zugängliche Literatur hierüber besteht und bei fast sämtlichen, auch die obigen einge-

1) Auf spezielle pathologisch-anatomische und histologische Verhältnisse bei der Arteriosklerose der Pulmonalis konnte in vorliegender klinischer Abhandlung nicht eingegangen werden.

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schlossen, der streng primäre Charakter der Kritik kaum standzuhalten vermag).

Unter den Beobachtern herrscht bezüglich der sekundären Pulmonalarteriensklerose so ziemliche Übereinstimmung, daß man bei Lungen- und Herzaffektionen noch relativ am häufigsten solche sklerotische Prozesse bei der Mitralstenose antrifft.

Zumeist werden in den Krankengeschichten ganz allgemein gehaltene Angaben über nicht charakteristische Symptome, die sich direkt oder indirekt auf das Herz beziehen, gemacht: Druck in der Herzgegend, Stechen in der Tiefe, Beklemmung, Herzklopfen, Schweratmigkeit. Von objektiven Befunden: Herzhypertrophie.

Die bekannten oft zitierten Fälle primärer Sklerose von Klob, Romberg, Aust und Laache boten das Bild eines kongenitalen Vitiums, wie auch zumeist die klinische Diagnose lautete.

Bisher wurde überhaupt kaum noch ein Versuch einer klinischen Diagnose der Pulmonalarteriensklerose gemacht.

Ich beschränke mich hier darauf, einige Ansichten und Bemerkungen verschiedener Kliniker und Ärzte wiederzugeben.

Nach Zehetmayer (1845) läßt die geringe Zahl der bekannt gewordenen Fälle in diagnostischer Beziehung bis jetzt keine gehaltvolle Ausbeute sammeln.

Der Zustand läßt sich, dem Ausdruck Bambergers (1857) zufolge, bis jetzt weder im Leben erkennen noch behandeln.

Nach De Buman (1858) setzt der vorliegende Prozeß keine spezifischen Erscheinungen.

Klob (1865) vermutet wohl, daß sein Fall möglicherweise einige Anhaltspunkte zu einer Diagnostik derartiger Erkrankungen bieten könnte.

Ganz ablehnend verhält sich Sauné (1877), wenn er schreibt: „Während des Lebens gibt es kein einziges Symptom, welches den Verdacht auf Bestehen eines Atheroms der Pulmonalarterie rechtfertigen würde."

Rombergs (1891) Ansicht geht dahin, daß die Pulmonalsklerose im allgemeinen klinisch symptomlos bleibe, es trete kein neuer Zug zu dem ursprünglichen Krankheitsbild. Sie bilde einen mehr zufälligen Befund bei der Autopsie. Klinisches Interesse rufe sie erst hervor, wenn sie — ohne das Bestehen eines Klappenfehlers oder hochgradiger Lungenveränderungen — zu starker Verengerung der Pulmonaläste geführt habe.

Da Rombergs und Austs (1892) Fälle unter den klinischen Symptomen eines angeborenen Herzfehlers verliefen, dürfte es nach letzterem schwer fallen, beide Erkrankungen klinisch auseinander zu

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halten. Eine klinische Symptomatologie dieser Krankheit ist nach Reichel (1894) seines Wissens noch nicht gezeichnet. Allerdings ist Schrötters (1901) Meinung nach die Möglichkeit, die Erkrankung auch im Leben zu erkennen, bei sorgfältiger Untersuchung nicht ausgeschlossen.

Schwartz (1907) kommt zu dem Schlusse, daß Sklerose der Arteria pulmonalis in den allermeisten Fällen symptomlos verlaufe. In wenigen Fällen sehr hochgradiger Sklerose könne klinisch ein besonderes Krankeitsbild zustande kommen, ähnlich dem Morbus coeruleus, stärkste Cyanose, Dyspnöe, Husten.

In allerjüngster Zeit glaubt Kitamura (1908) zu dem Ausspruch berechtigt zu sein, „daß die Kliniker so gut wie gar keine Erfahrungen über Sklerose der Pulmonalarterien gesammelt haben".

Das Resumee aus allem ist so ziemlich die völlige Ableugnung der Möglichkeit einer klinischen Diagnose der Atherosclerosis pulmonalis.

Diesem absoluten Pessimismus gegenüberzutreten und an der Hand von Eigenbeobachtungen zu zeigen, daß das Erkennen des Zustandes während des Lebens unter bestimmten Umständen zweifellos gelingt, sei Aufgabe unserer Darlegungen[1]).

Mehr als $1\frac{1}{2}$ Dezennien verfolgte ich an klinischen und ambulanten Kranken das abzuhandelnde Thema.

Um die ohnehin komplizierten und oft schwer zu deutenden Verhältnisse nicht noch mehr zu komplizieren, werden hier, wo es sich vor allem um klinische Semiologie handelt, eine Reihe von Prozessen ausgeschaltet:

Kongenitale Fehler und Mißbildungen, Pulmonalstenosen und Aneurysmabildungen am Gefäße, die zu derartigen atherosklerotischen Vorgängen Veranlassung bilden, resp. zum Teil mit diesen in Korrelation oder direkt von ihnen abhängig sein können.

Von den nachfolgenden zehn Eigenbeobachtungen (zumeist an der Innsbrucker medizin. Klinik gemacht) betreffen die ersten drei zufällige Obduktionsbefunde, die anderen sieben sind einer klinischen Analyse zugänglich.

Unter ihnen bestand zweimal Aortensklerose mit Insuffizienz der Semilunaren, die übrigen fünf Fälle beziehen sich auf Mitralstenosen.

Bei denen der letzten Kategorie wurde auf Grund der früheren

1) In einer vorläufigen Mitteilung (Münchener med. Wochenschr. Aug. 1908 Nr. 31) legte ich die Hauptpunkte dar nach einem am 26. Juni 1908 in der Innsbrucker wissenschaftlichen Ärztegesellschaft gehaltenen Vortrag.

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Beobachtungen und Epikrise zweimal eine Wahrscheinlichkeits- und einmal eine sichere klinische Diagnose auf Atherosclerosis pulmonalis gestellt.

Für die gütige Überlassung der Sektionsprotokolle bin ich Herrn Prof. Pommer, Vorstand des pathologisch-anatomischen Institutes, zu besonderem Danke verpflichtet.

Eigenbeobachtungen.

I. Fritz B., 67jähriger verheirateter Pensionist, Eintritt an der medizin. Klinik am 5. September 1902.

Klinische Diagnose: Cirrhosis hepatis, Hydrops ascites, Venenektasien im Ösophagus. Peritonitis chronica. Myodegeneratio cordis incipiens. Bronchopneumonia. Alkoholismus chronicus. Arteriosklerose.

Ausgesprochener schwerer Alkoholiker. Zu wiederholten Malen stärkere Diarrhöen. In letzterer Zeit öfters Erbrechen. Seit langen Jahren ist Pat. auffallend vergeßlich.

Seit mehreren Wochen Zunahme des Bauchumfanges, Atemnot, Urinverminderung. Öfters Schmerzen in der Leber- und Milzgegend.

Status: Großer, mäßig kräftiger, abgemagerter Mann mit deutlich schmutzig gelblichem Kolorit und dem sonstigen Habitus der Cirrhotiker. Zunge trocken, rissig, gelb belegt.

Deutliche ausgebreitete bronchitische Zeichen.

Mäßiges Lungenemphysem. Herzspitzenstoß nicht fühlbar. Töne abgeschwächt, rein, Puls im allgemeinen ziemlich kräftig. Mäßige Sklerose der Radialarterie. Übrige Arterien vielleicht etwas rigider.

Abdomen ausgedehnt (92 cm), namentlich in den seitlichen Partien; freier Flüssigkeitserguß deutlich nachweisbar.

Milzdämpfung stark verbreitert (über 10 cm) reicht nach vorne fast bis zum Rippenbogenrand.

Leberdämpfung von der achten Rippe an mit tympanitischem Beiklang. Leberoberfläche anscheinend uneben, Resistenz der Leber vermehrt. Im 24stündigen verminderten, hochgestellten, braunroten Harn kein Eiweiß, Spuren von Gallenfarbstoff.

Therapie: Kalomel. Entsprechende Diät.

14. IX. Erbrechen von dunkelbraunschwarzen flüssigen Massen in der Menge von 1100 ccm.

26. (Ausstrahlende?) Schmerzen im l. Arm.

Abdomen stark aufgetrieben. Singultus.

Von Mitte Oktober an Zunahme des Aszites. Ödem am rechten Rippenbogenrand.

Im Oktober wiederholt dumpfe brennende Schmerzen hinter dem Brustbeine, vermehrte Stauungssymptome. Zyanotisches Kolorit.

Universelle Stauungskatarrhe. Schweratmigkeit.

Unter Benommenheit, Herzschwäche und bronchopneumonischen Symptomen Exitus letalis am 5. November 1902.

Sektion: 7. November 1902 (Prot.-Nr. 6227/278).

Pathol. anat. Diagnose:

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Cirrhosis hepatis im Stadium vorgeschrittener Schrumpfung. Dilatatio cordis. Atrophie, parenchymatöse und fettige (?) Degeneration des Myokards. Hochgradige Sklerose der Brustaorta und z. T. auch der Arteria pulmonalis. Mächtige, hochgradig verkalkte, sklerotische Wucherungen an der Mündung der linken Arteria coronaria. Weitgehende Einengung der rechten Koronararterienmündung durch myokarditische verkalkte Bildungen. Ausgeprägter chronischer Speiseröhren-Magen-Darmkatarrh. Ödem und hypostat. Pneumonie der hinteren Lungenbezirke.

(Die Klappen des Ost. atr.-ventric. sin. etwas verdickt, Sehnenfäden kurz.)

II. Johann L., 76jähriger verwitweter Gärtner von Bozen (wegen Zystitis an der medizin. Klinik in Behandlung), kam am 17. November 1893 in das mediz. Ambulatorium (Ambul. Prot.-Nr. 1264) mit der Klage über Kreuzschmerzen, Schwäche in den Beinen und Schwindel. Die klinische Diagnose lautete auf: Vertigo; Arteriosclerosis, Hypertrophia cordis praecipue ventriculi dextri. Anamnestisch wurde eruiert, daß der Mann in früheren Jahren Blattern und Malaria zu überstehen hatte. Vor 3 Jahren Harn- und Stuhlverhaltung durch ca. 3—4 Tage, mit Schwächegefühl in den Beinen. Wegen Klagen, die auf ein Rückenmarksleiden und psychische Störungen hindeuteten, wurde Pat. an das Ambulatorium der neuropsychiatr. Klinik verwiesen.

An letzterer Klinik (Vorstand Herr Prof. Anton) erfolgte am selben Tage (17. November 1893) die Aufnahme (Prot.-Nr. 183). Im folgenden sei nur die Anamnese und die auf den uns hier interessierenden Befund der Thoraxorgane bezüglichen Daten gebracht. Vor 5 Wochen merkte der Kranke, daß seine Beine schwächer wurden und daß er sich nur mit Mühe fortbewegen konnte. Zeitweise heftige Schmerzen im Kreuze. Wiederholt Anfälle von Schwindel und Flimmern vor den Augen, so daß sich Pat. an der Wand halten mußte, um nicht umzufallen. Verschlechterung des Gesichtssinnes. Er müsse auch öfter urinieren als sonst und der Harn habe einen starken, unangenehmen Geruch.

Es wird auch eine auffällige Verschlechterung des Gedächtnisses angegeben. Ohne auf die näheren Befunde in neurol.-psych. Hinsicht einzugehen, genüge, daß die Diagnose auf Myelo-Meningitis chronica gestellt wurde. Es findet sich die Angabe: Herzdämpfung ziemlich innerhalb normaler Grenzen. Herztöne im allgemeinen rein. Es finden sich weiter keine besonderen, auf den Zustand der Brustorgane bezüglichen Angaben. Am 20. Januar 1894 gebessert entlassen.

Am 29. desselben Monates erfolgt jedoch gleich wiederum die zweite Aufnahme an derselben Klinik. Durch 3 Tage hatte sich Pat. ganz wohl gefühlt, am 4. trat wieder Zittern an den Beinen und Schwäche auf. Das Gehen verschlechterte sich, zugleich ging der Urin unwillkürlich ab.

Schmerzen und Stuhlbeschwerden fehlten. Während des Spitalaufenthaltes wechselten heftige Diarrhöen mit Verstopfung ab. Zustand sehr wechselnd.

Am 5. Mai 1894 Entlassung. Neuerliche (3.) Spitalsaufnahme am 10. Juni desselben Jahres, Aufenthalt bis 17. September.

In der Krankengeschichte finden sich keine besonderen Bemerkungen bezüglich des Verhaltens von Lunge und Herz. Einige Male Schwindelanfälle. Allmähliche Besserung des Gesamtbefundes und der Rückenmarksleiden-Symptome.

4. Aufenthalt vom 12. Oktober bis 11. Dezember 1894. Nach dem Austritt sofortige neuerliche Verschlechterung des Zustandes, so daß er sich am 12. Oktober wiederum aufnehmen ließ. Am 8. November bereitete ihm eine jüngst entstandene Hernie Schmerzen. Vom 11.—18. November Auftreten von Husten, der wiederholt bei Tag und Nacht eintrat, dann nachließ.

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Vom 30. November bis 8. Dezember Diarrhöen, am 3. Dezember etwas höhere Temperatur. Harn normal.

Die letzte Aufnahme erfolgte am 14. Januar 1895. Am 18. ist schlechter Schlaf und etwas unregelmäßiger Puls notiert.

In der Nacht des 20. stand Pat. auf und war unruhig, laut, und rumorte herum. Ist nicht gut orientiert.

Es findet sich folgender Status angegeben: Mittelgroßes Individuum von senilem Aussehen, schlechtem Ernährungszustand. Schläfenarterien geschlängelt, fühlen sich rigide an. Beide Infraorbital. auf Druck schmerzhaft. Gerontoxon an beiden Augen, Cataracta incipiens. Linke Pupille etwas weiter als rechte. Herztöne klingend. Deutlicher Tremor der Hände.

18. I. Pat. schwach, neigt wieder zu Diarrhöen. Während der Nacht Verwirrtheit. Transfer. auf die psychiatr. Abteilung.

24. I. starke Diarrhöen. 25. I. Pulsus irregul.

26. I. dreistündiger Schüttelfrost (Temp. 39,7). Puls 104.

Über dem l. Unterlappen bronchial. Exspir., vereinzelte Rasselgeräusche. 18. II. Zystitis.

Im weiteren Verlauf zunehmende Verworrenheit.

Hinsichtlich des Herz- und Lungenbefundes finden sich keine näheren Notizen.

Am 9. VII. 1895 Exitus letalis $11^1/_4$ Uhr nachts.

Die klinische Diagnose lautete:

Arteriosklerose. Myelitis chronica auf arteriosklerot. Basis. Dementia senilis.

Außerdem laut Protokoll der mediz. Klinik und Ambulatorium: Hypertrophia ventricul. dextr. cordis. Bronchitis chron. Cystitis chronica.

Sektion am 10. Juli 1895. Prot.-Nr. 3761/144.

Körper mittelgroß, kräftig, etwas abgemagert.

Beide Lungen frei, Pleuren zart. Im Herzbeutel ca. 30 g leicht molkig getrübter, gelblicher Flüssigkeit. Kleine Blutungen an den Pleuren. Epikard fettreich, stark venös injiziert, läßt die Arterien als verdickte geschlängelte Streifen durchschimmern.

Lungen überall lufthaltig, blutreich, die vorderen Ränder gedunsen.

Herz mittelgroß, rechter Ventrikel verbreitert und schlaff.

In beiden Höhlen lockere Blut- und Fibringerinnsel, sowie im linken Ventrikel auch flüssiges Blut.

Der rechte Ast der Pulmonalarterie ist bis auf einen kleinen oben noch freien Spalt völlig verlegt durch eine an der unteren lateralen und auch an der medialen Wand innig anhaftende Thrombusmasse, deren seitliches und unterstes Gebiet ins Weißgelbliche verfärbt erscheint und sehr schlottrig, wie ödematös ist, deren übriger größerer Teil bis auf ein 2 cm dickes Wandgebiet völlig untergegangen ist in einer mit brauner Flüssigkeit erfüllten Erweichungshöhle. Auch im untern Teile des Thrombus hochgradige, nur eine dünne Wandpartie übriglassende zentrale Erweichung. Der untere Teil ist ebenfalls wandständig fixiert.

Die Aorta ist hochgradig erweitert und arteriosklerotisch, sie mißt im Umfang ca. 12 cm.

Der bei der Durchschneidung der Pulmonalis zur Durchtrennung kommende Thrombus setzt sich bis auf eine Strecke von 3 cm oberhalb der Klappen in den Stamm der Pulmonalis fort und sitzt da an der hintern Wand, dieselbe auf eine Strecke von 5 cm des im ganzen 12—14 cm weiten Umfanges einnehmend. Der

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größere rechts- und nach vorn gewendete Teil des Lumens ist offen. Wand der Pulmonalis über 1 mm dick, Intima gerunzelt.

Im Bereiche der Fixationsstelle des Thrombus ein bis auf 3 cm dickes knorpeliges, hartes, sklerotisches Wandgebiet bemerkbar.

Die Zipfelklappen an den Rändern verdickt, ebenso die Aortenklappen, die letzteren sehr stark ausgedehnt. Die Konorarostien sehr weit. Linker Ventrikel etwa 1—1½ cm dick; Herzmuskel auffallend dunkel, gesättigt braun-blaurot. Trabekel im linken Herzen ziemlich mager. Die des rechten Herzens massig, dick. Konus der Pulmonalis sehr weit und 7 mm dick. —

In den peripheren Teilen des Zirkulationsapparates waren keinerlei thrombotische Veränderungen auffindbar.

Diagnose: Primäre Thrombose der rechten Arteria pulmonalis mit beinahe völliger Verlegung des Lumens. Arteriosklerose der Pulmonalis. Exzentrische Hypertrophie des rechten Herzens.

Atrophie des Gehirns. Chronische Ösophagitis. Hochgradige Bronchitis purulenta. Cystitis chronica mit exzentr. Hypertrophie der Blase, reichlicher Divertikel- und Steinbildung.

Das Präparat wird im Museum des pathol. anatom. Institutes unter der Signatur C 93 b (neue Signatur C 136) aufbewahrt. (Anfangsteil der Pulmonalis mit wandständigem, zentral erweichten Thrombus. Stamm der rechten Arteria pulmonalis mit fast vollständig obturierenden, zentral erweichten, gemischten Thromben.)

III. Am patholog.-anat. Institut (Vorstand Herr Prof. Pommer) kam am 13. Januar 1906 nachstehender Fall zur Obduktion (Prot.-Nr. 7254/10). Barbara B., 60jährige Hausmagd von Innsbruck.

Ärztl. Diagnose: Degeneratio cordis.

Fettreiche, stark ödematöse Leiche. Hirn atrophisch, Gefäße an der Hirnbasis hochgradig arteriosklerotisch. Stauung. Beiderseitiger mäßiger Hydrothorax. Überall Luft in den Lungen.

Herz stark fettbewachsen. Residuen von Perikarditis. Geringes Hydroperikard.

Venen des Ösophagus sehr weit. Epithel verdickt.

Im Kehlkopfeingang Blutungen und Ödem. In den Bronchien stark trübe Schleimflocken. Lungen stark pigmentiert und atrophisch.

Wand des linken Ventrikels beträchtlich hypertrophisch (2½ cm), Ostium atrioventriculare sinistrum nur für einen Finger durchgängig. Sehnenfäden stark verkürzt und verdickt. Bikuspidalis verwachsen. Wand des rechten Ventrikels ebenfalls hypertrophiert. Muskel fettdurchwachsen. Farbe des Herzfleisches fahlbraun, dasselbe brüchig.

Aorta ungewöhnlich weit, Klappen zart, schlußfähig. Intima der Arteria pulmonalis arteriosklerotisch verdickt, ebenso an der Aorta.

Milz etwas vergrößert.

Pathol. anatom. Diagnose:

Stenose des Ostium atrioventriculare sin. infolge abgeheilter Endokarditis der Bikuspidalis. Exzentrische Hypertrophie, besonders des linken Ventrikels. Trübe Schwellung des Myokards und Fettdurchwachsung desselben im rechten. Ödem der Lunge. Atrophie des Parenchyma. Arteriosklerose der basilaren Hirnarterien, Brustaorta und Arteria pulmonalis. Chron. Rachen-Ösophagus-Magen-Darmkatarrh. Allgemeine Adipositas. Potatrix.

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Von den Angehörigen und dem behandelnden Arzt konnte nur so viel in Erfahrung gebracht werden, daß die Kranke schon seit langem herzleidend war.

Wegen Zunahme der Beschwerden (Atemnot, Ödeme usw.) wurde am 11. Jan. H. Dr. P. gerufen, welcher die Kranke in fast präagonalem Zustand mit den Erscheinungen allerhöchstgradiger Herzinsuffizienz antraf.

Während des Lebens sollen öfters Anfälle von starker Blauverfärbung mit Atembeklemmungen bestanden haben.

IV. Zu wiederholten Malen stand vom Jahre 1891—1896 die 1891 im 39. Lebensjahr stehende Fabriksarbeitersfrau Maria F., geb. zu Matarello in Südtirol, wohnhaft Innsbruck, ambulatorisch und klinisch an der mediz. Klinik in Behandlung.

1. Aufnahme. 3. XI. 1891—16. I. 1892. Diagnose: Insuffic. valv. bicuspid. Insuffic. cordis. Es wurden verordnet: Digitalis, Kal. jod., Kalomel.

2. Aufnahme. 1. III. 1891—24. III. 1892. Diagnose: Stenosis et insufficientia valv. bicuspid. Degeneratio cordis.

3. Aufnahme. 3. IX.—1. X. 1893. Insuffic. cordis. Insuffic. bicuspidal. Cyanosis.

4. Aufnahme. 7. I.—24. II. 1895. Vitium cordis. Insuffic. et stenosis valvul. mitralis. Degeneratio cordis. Cyanosis. Albuminuria lev. grad.

5. Aufnahme. 30. XI. 1895—10. II. 1896. Stenosis et insuffic. valvul. mitralis. Degeneratio et insufficientia cordis. Cyanosis permagna. Polyarthritis rheumatica. Induratio cyanotica hepatis et renum. (Infarct. pulmon.?) Pleuritis obsoleta.

6. Aufnahme. 2. VI.—9. VII. 1896. Stenosis et insuffic. valv. mitral. Degeneratio cordis. Cyanosis permagna. Polyarthritis.

7. Aufnahme. 8. XI.—4. XII. 1896. Stenosis et insuff. valv. mitralis. Degeneratio et insuffic. cordis hypertroph. Cyanosis permagna. Induratio cyanotica hepatis et renum. Albuminuria. (Herzfehlerlunge.) Ad finem Hydrops.

Die Kranke wurde in ihrer Jugend wegen Drüsenabszessen am Halse operiert. Mit ca. 15 Jahren überstand sie eine sehr schwere, über 6 Monate dauernde Gliedersucht, Gelenksrheumatismus, welcher in Zwischenräumen von ungefähr 2 Jahren noch 2mal wiederkehrte. Während der nächsten Zeit hatte sie durch 1½ Jahre in der Schweiz an Bauchschmerzen zu leiden, worauf sie sich völlig erholte. Im Jahre 1883 heiratete die Pat.

Nach der ersten Entbindung, Mai 1885, bekam sie Herzklopfen und Mattigkeit, litt an Schweratmigkeit beim Gehen, besonders beim Stiegensteigen.

Der Urin verminderte sich und es traten kurze Zeit geringe Anschwellungen der Füße und des Bauches auf. Es erfolgten noch 3 Geburten (2mal Forceps). Nach jedesmaliger Entbindung hatte sie die gleichen Erscheinungen und es verschlechterte sich immer der Zustand. Es traten auch leichte vorübergehende Ödeme in der Knöchelgegend auf.

Während ihrer beiden ersten Spitalsaufnahmen gingen die Beschwerden auf entsprechende Medikation immer sehr rasch zurück.

3. Aufnahme 3. Sept. 1893: Seit ihrer letzten Entbindung, am 16. Aug. 1893 liegt die Kranke immer zu Bette und klagt über Herzklopfen, Schweratmigkeit und Schwäche. (Nach der Entbindung sei auch das Handgelenk etwas angeschwollen gewesen.) Ihre 4 Kinder sind alle in sehr frühem Alter gestorben.

Status: Pat. ist von schwächlichem Körperbau, Muskulatur schlaff, Ernährungszustand schlecht. Dieselbe befindet sich stets in sitzender Stellung im Bette, ringt nach Atem; ihr Benehmen ist ängstlich, ihre Stimme schwach und tonlos.

Die allgemeine Hautdecke und die sichtbaren Schleimhäute blaß, livid; geringes Ödem an den Beinen und der Abdominalhaut angedeutet. Puls klein, arhythmisch.

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Beschleunigte, dyspnoische Respiration. Expektoration gering. Leicht eitrig-schleimiges Sputum.

Kopf klein. Gesichtsfarbe livid mit einer Spur subikterischer Verfärbung. Hals mäßig lang, an der linken Seite unter dem Unterkieferwinkel 2—3 unregelmäßige, etwas vertiefte alte Narben (von Drüsenabszessen aus der Kinderzeit herrührend).

Thorax ziemlich breit, flach. Lungengrenzen normal, überall voller Lungenschall und vesikuläres Atmen, reichlich trockene und feuchte Rasselgeräusche über den unteren Lungenpartien.

Das Herz zeigt nach oben und rechts etwas erweiterte Grenzen. Beginn der absoluten Dämpfung im 3. I. R. Nach rechts reicht dieselbe bis zur Sternalmitte. Spitzenstoß ziemlich schwach unter der Mammilla im 5. I. R. Epigastrische Pulsation. Die Auskultation ergibt ein über der Herzspitze am deutlichsten wahrzunehmendes, den 1. Herzton begleitendes, jedoch nicht ganz verdeckendes, pfauchendes Geräusch von wechselnder Stärke. Die übrigen Töne rein. 2. Pulmonalton akzentuiert. Leichtes diastolisches Vibrieren im 2. l. I. R. —

Abdomen: In der Höhe des Rippenbogens, vom 10. Brustwirbel beiläufig nach vorne zu bis genau zur Medianlinie eine gürtelförmig angeordnete Reihe von flachen erbsen- bis bohnengroßen, ziemlich unregelmäßigen, doch rundlichen, im Zentrum heller, am Rande dunkelbraun pigmentierten Narben, an welchen früher heftiges Jucken auftrat. Dieselben sollen aus Blasen, welche 2 Tage vor der letzten Entbindung auftraten, entstanden sein (Herpes zoster). Bauchdecken ziemlich stark gespannt, Bauch aufgetrieben.

Lebergrenzen innerhalb gewöhnlicher Ausdehnung.

Beine ödematös. Therapie: Bettruhe, Digitalis, Milchdiät.

Während ihres 5. Aufenthaltes stellten sich wieder Gelenkschwellungen und die Erscheinungen eines hämorrhagischen Lungeninfarktes ein. Durch einige Tage auffallende Dyspnöe und höchstgradige Zyanose, Verfallensein.

Ebenso war während des 6. Spitalaufenthaltes ein neuerlicher Anfall von Polyarthritis rheumatica vorhanden. (2. Juni 1896.) Vor 5 Tagen habe sie sich wiederum erkältet. In der Früh konnte sie nicht mehr aufstehen. Pat. hatte Schmerzen in der linken Schulter, in beiden Ellbogengelenken und beiden Kniegelenken.

Die Gelenke waren gerötet und geschwollen. Bei der Aufnahme sind auch Schmerzen im linken Hüftgelenk aufgetreten, die bis zur Wirbelsäule ausstrahlen. Die Kranke klagt über Beklemmung, Herzklopfen, Husten, Auswurf und Atemnot.

Es besteht sehr starke Zyanose, ausgesprochene Dyspnöe, jedoch keine Ödeme. Urin leicht vermindert, sauer, Chloride vermindert, Diazoreaktion negativ, reichlich Albumen. Auf Bettruhe, Milchdiät, Natr. salicyl., Salol, Bäder ließen die Schmerzen bald nach.

In der Zusammenfassung der Befunde während ihrer letzten Spitalsaufenthalte wurde nachstehendes notiert:

Es handelt sich um eine ziemlich kleine, abgemagerte, enorm zyanotische Frau. Dieselbe kam immer höchst zyanotisch und mehr weniger dyspnoisch zur klinischen Aufnahme, wobei die Blausucht graduell stets stärker war wie die Atemnot.

Die Stauung in den Lungen und parenchymatösen Organen, die kolossale Zyanose beherrschte immer das ganze Krankheitsbild dieses Vitium cordis. Ödeme, früher nur in sehr geringem Grade vorübergehend vorhanden, traten vollkommen in den Hintergrund.

Herzbefund. Verbreiterung der Herzdämpfung nach rechts bis gegen die rechte Sternallinie, im 2. linken I. R. knapp am Sternum eine ca. $1^1/_2$ cm betragende Ver-

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kürzung des Perkussionsschalles, etwas Druckempfindlichkeit daselbst, speziell bei vorgebeugtem Oberkörper.

Systolisches und mitunter dieses an Stärke und Schärfe übertreffendes diastolisches Geräusch an der Herzspitze.

Akzentuierung des 2. Pulmonaltones, manchmal undeutliches, inkonstantes, leicht schwirrendes Geräusch über der Pulmonalis.

Puls klein, weich, öfters sehr unregelmäßig. Zuweilen stärkere Tachykardie und hochgradige Arhythmie. Hie und da anfallsweise stechende Schmerzen in der Oberbrustgegend mit Angst verbunden.

Im Vergleich zu den früheren Befunden am Herzen, traten nach und nach die Symptome der Insuffizienz der Mitralklappe gegenüber denen der Stenose immer mehr zurück, so daß schließlich von diesen beiden Zuständen am Ostium atrioventric sin. weitaus die Stenosierung das Übergewicht bekam.

Auf Gebrauch von Digitalis, Spartein, Strophantus und Milchdiät besserte sich immer ziemlich rasch der Zustand. Manchmal wurden Digitalispräparate schlecht vertragen, weshalb zu den angeführten andern Mitteln und zu Adonis vernalis, Diuretin, Koffein gegriffen wurde. Während die Atemnot verhältnismäßig rasch zu beeinflussen war, ging die Zyanose nicht zurück.

7. und letzter Aufenthalt vom 8. November bis 4. Dezember 1896. Pat. schlecht genährt, abgemagert. Gesicht (besonders Nase und Wangen), dann die Endphalangen enorm zyanotisch. Dabei zeigt die Frau außerdem einen Stich ins Ikterische (subikterische Verfärbung) Dyspnöe. Die Kranke sitzt aufrecht im Bette, klagt über Herzklopfen, Präkordialangst, zusammenschnürendes Gefühl, Schmerzen in der Herzgegend und Lufthunger. Auswurf bei quälendem Husten unregelmäßig, manchmal spärlich und mehr zähe, hie und da etwas reichlicher. Herzgegend leicht vorgewölbt. Herztätigkeit ungemein lebhaft, sehr arhythmisch, frequent. Über der ganzen Herzgegend ausgebreitetes pulsatorisches Wogen. Von Zeit zu Zeit sieht man systolische Einziehungen im 4. und 5. I. R. zwischen Sternal- und Papillarlinie. Die erregte, weithin sichtbare, wogende Herzaktion steht im auffallenden Gegensatz zum schwachen, weichen, leicht komprimierbaren Puls.

Die perkussorische Untersuchung des Herzens ist etwas erschwert, jedoch kann eine wesentliche Verbreiterung der Herzdämpfung nach rechts bis über den rechten Sternalrand, fast bis zur rechten Parasternallinie konstatiert werden.

Im 2. linken Interkostalraum an der oben angegebenen Stelle findet sich eine kleine Zone gedämpften Schalles. Pulsatorische Phänomene daselbst nicht nachweisbar, vielleicht etwas Druckempfindlichkeit, die übrigens auch weiter nach abwärts zu manchmal auftritt.

An der Herzspitze besteht ein weiches, ziemlich langgezogenes, undeutliches systolisches, hie und da präsystolisches Geräusch von mehr schabendem Charakter. Bei gewissen Stellungen tritt ein viel lauteres, jedoch kürzeres, pfauchendes, diastolisches Geräusch hervor, das im weiteren Verlauf das Dominierende wird und sich mitunter am deutlichsten im 3. linken I. R. am Sternalrand findet.

Akzentuierung des 2. Pulmonaltones, dabei über der Pulmonalis inkonstante, nicht recht zu lokalisierende, und nichts Charakteristisches darbietende Geräusche verschiedenen, einige Male schabenden Charakters, zeitweise mit Andeutung von Spaltung. Keine Reibegeräusche. An der Herzspitze zumeist diastolisches, leicht rieselndes Schwirren palpabel.

Bei der Betastung der Lebergegend fühlt man eine erhöhte Resistenz, auf Druck leichte Empfindlichkeit.

Abdomen mäßig aufgetrieben. Flüssigkeitserguß erheblicheren Grades nicht

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vorfindlich. Es bestehen keinerlei Ödeme. Die auffallend dünnen Beine zeigen trockene, abschilfernde, leicht in Falten aufhebbare Haut.

Urin vermindert, 800 ccm, mäßig getrübt, saturiert, zeigt mäßigen Eiweißgehalt und hyaline Zylinder. Die mikroskopische Untersuchung des Auswurfs läßt Eiterkörperchen und sehr zahlreiche typische Herzfehlerzellen erkennen.

Die Blutuntersuchung ergibt über 6 Mill. rote Blutkörperchen im Kubikmill.

Bei einer Visite klagte die Frau plötzlich über krampfhafte in die Tiefe gehende Herzschmerzen, zugleich zeigte sie Schweißausbruch, Blässe und äußerst ängstlichen Gesichtsausdruck. Nach einigen Minuten hörte der Anfall auf und es trat wieder das gewöhnliche Bild starker Blausucht auf.

Während des letzten Spitalsaufenthaltes besteht auffällige Idiosynkrasie gegen Digitalispräparate, weshalb die oben erwähnten Mittel zur Anwendung gelangen. Es macht sich immer mehr und mehr hochgradige Herzinsuffizienz merklich, welche sich ganz besonders in der Richtung der höchstgradigen Blausucht und Atemnot dokumentiert.

Das Herz arbeitet ungemein angestrengt, der Puls aussetzend, sehr unregelmäßig, im höchsten Maße weich und komprimierbar.

Gegen die Anfälle von Asthma cardiale findet wiederholt Nitroglyzerin mit gutem symptomatischem Erfolg Anwendung, späterhin gegen die Kollapszustände Reizmittel.

Anfangs Dezember erst machen sich leichte Ödeme in der Knöchelgegend bemerkbar. Harnmenge gering. Eiweißgehalt zugenommen.

1. XII. Arhythmie und Schwäche des Pulses höchstgradig.

Unter Zunahme der Dyspnöe und der tiefschwarzblauen Verfärbung Eintritt von Kollaps und Erstickungsanfällen. Äther- und Kampferinjektionen. Reizmittel. Wachsen der Ödeme, Benommenheit, exzessive Herzschwäche. Exitus letalis am 4. Dezember 1896 um 6½ Uhr abends.

Die klinische Diagnose lautete: Stenosis et insufficientia valvulae mitralis subsequ. hypertrophia ventric. dext. cordis. Degeneratio et insufficientia cordis hypertrophicae. Cyanosis permagna. Induratio cyanotica hepatis et renum. Albuminuria (Nephrit. chron.). Hochgradige Stauungs(Herzfehler)lunge. Zum Schluß Ödeme.

Es wurde der Verdacht auf eine Perikardialsynechie ausgesprochen. Hinsichtlich der Arteria pulmonalis mußte die Diagnose in suspenso bleiben.

Für eine aneurysmatische Erweiterung waren zu wenig Symptome da.

Daß aber die Pulmonalarterie in Mitleidenschaft bei diesem eigenartigen und schweren Herzfehler gezogen wurde, wurde bei jedem Aufenthalt klarer; nur konnten die diesbezüglichen Symptome nicht recht in eine bestimmte Kategorie eingereiht werden. Nicht undenkbar war eine infolge Verwachsung mit der Umgebung (vom Herzbeutel aus) aufgetretene Verzerrung, zumal Spuren obsoleter Pleuritis bestanden. Der Gedanke an eventuelle sklerotische Prozesse wurde wegen der exzessiven Stauung in der Lunge und der riesigen Zyanose auch rege (eine ausgesprochene Arteriosklerose an den peripheren Arterien fehlte). Es wurde bei dieser Kranken während der letzteren klinischen Aufenthalte bei verschiedentlichen Gelegenheiten (Demonstrationen in Kursen und Vorlesungen) wiederholt die Wahrscheinlichkeitsdiagnose, daß bei dem bestehenden Vitium cordis, speziell der immer stärker hervortretenden Mitralstenose, eine Sklerose der Pulmonalarterie vorliegen könne, ausgesprochen und die hierfür maßgebenden Zeichen analysiert.

Sektion 5. Dez. 1896. Prot. Nr. 4154/285.

Körper klein, schwächlich; untere Extremitäten ödematös, blaß. Zyanose des

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Gesichtes. Totenstarre an den unteren Extremitäten erhalten. Unterleib aufgetrieben.

Pachymeningitis interna haemorrh. Atrophia cerebri, chron. Ödem der Meningen.

Im Rachen und Luftwegen mit Blut vermengter Schleim. Schleimhäute blaß. Beide Lungen bis auf die unteren Partien gleichmäßig fixiert.

Diese von dicken Adhäsionssträngen, zwischen welchen etwa 1/8 l klarer, seröser Flüssigkeit angehäuft ist, an das Zwerchfell angeheftet.

Perikard in ganzer Ausdehnung mit dem Herzen verwachsen.

Die Lunge ziemlich voluminös, dicht, teils mit schaumarmer, teils mit feinschaumiger Flüssigkeit infiltriert, auffallend rot gefärbt, sehr zäh, jedoch schlaff. Bei Durchschneidung der rechten Lunge fällt auf, daß ein überdoppeltdaumenbreiter Pulmonalast zum größten Teil durch einen haftenden geschichteten Thrombus verstopft ist. An der Verwachsungsmembran des Perikardiums zerstreute Blutaustritte.

Herz sehr groß, stumpf, die Spitze zum größten Teil vom rechten Ventrikel gebildet, der Länge nach 12 cm, der Breite nach 14 cm, der Dicke nach über 10 cm messend, nicht kontrahiert. In den Herzhöhlen viel Fibrin und Blutgerinnungen. Das Ostium atrioventriculare sinistr. nur für die Spitze eines kleinen Fingers durchgängig.

Die Bikuspidalklappe sehr verdickt und verkürzt. Der Aortazipfel überragt von verkalkten, rauhen Erhebungen in der Ausdehnung einer Bohne. Der Papillarmuskel desselben stark ausgezerrt, die Sehnenfäden kurz, kaum 2/3 bis 1 cm lang, säulenähnlich verdickt. Der linke Vorhof sehr weit, sein Endokard verdickt. Der linke Ventrikel ziemlich weit, seine Wand bis 1 1/2 cm dick. Im hohen Grade erweitert ist der rechte Vorhof und Ventrikel.

Die Wand des Conus pulmonalis bis über 1 cm dick, die unteren Teile der Wand des rechten Ventrikels bis 1 1/2 cm dick, die Trikuspidal- und Pulmonalklappen zart. Die Trabekel und Papillarmuskeln fettig gelb, die peripheren Schichten des Herzmuskels dunkel rotzyanotisch. In der Aorta und deren Zweigen keine auffallend sklerotischen Veränderungen, ebensowenig in den Gehirnarterien, diese blaß.

Die Arteria pulmonalis hat im Bereiche der Klappen einen Umfang von 7 cm, die Aorta beiläufig 1/2 cm oberhalb der Klappen.

Im Gebiet der Teilung der rechten Pulmonalis, weit hineinreichend in die ersten Äste derselben, eine hochgradige Erweiterung, so daß der Umfang der Teilungsstelle der rechten Pulmonalis bis über 9 cm beträgt, dabei die Wandung hier und in den weiteren Ästen, sowie auch in der beiläufig 6 cm weiten linken Pulmonalis reichlich gebuckelt und uneben, mit weißlichen Höckerchen und Strichelchen bezeichnet. Die rechtsseitige und vordere Wandpartie der rechten Pulmonalarterie mit einem über 3 cm breiten und 1 cm dicken geschichteten Thrombus besetzt, der sich in den angegebenen Pulmonalarterienast der rechten Lunge fast obturierend fortsetzt. Von der zentralwärts gewendeten Spitze dieses Thrombus erscheint, zum Teil überdeckt, ein bis auf 1 cm oberhalb der Pulmonalklappen (Kommissur zwischen der rechten und linken) sich erstreckendes, 3eckig gestaltetes, bis 3 cm langes, unter dem Thrombus 1 cm breites Feld, beiläufig 1/2 cm unter der übrigen Intimafläche der Pulmonalis gelegen.

Dieses vertiefte Feld ist mit einer blassen, glatten Bekleidungsschicht ausgestattet, die Ränder desselben sind abgerundet, wie etwas gewulstet und leicht nach außen verzogen.

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Eine ähnliche 4 cm lange, überwiegend gleichmäßig 1—⅔ cm breite Auseinanderweichung der innersten Wandschichte der Pulmonalis erstreckt sich von dem Teilungssporn des Hauptstammes der Pulmonalis in das Gebiet der erweiterten rechten Pulmonalis hinauf. Auch die Ränder dieser Zusammenhangstrennung sind abgerundet und etwas nach außen gekrempelt. Die Basis leicht wellig, faltig, graurötlich, durch dieselbe schimmern die dunklen Substanzen der benachbarten Lungen- und Lymphdrüsengebilde zum Teil durch. Die faltige Fläche dabei nirgends rauh, zerfasert, sondern ebenfalls mit einer glatten aber durchscheinend dünnen Schichte überzogen. Dieser Auseinanderweichung schließt sich ein halb bohnengroßer, haftender, welliger Thrombus an. An der Basis der Auseinanderweichung selbst nur einige Spuren von Blutgerinnung.

Geringgradiger Aszites; hochgradiger Stauungskatarrh des Magens und Darms; zyanotische Induration der Milz (14:9:6).

Atrophische Muskatnußleber. Zyanose der durch interstitielle Nephritis granulierten und amyloid entarteten Niere.

Pathologisch anatomische Diagnose:

Exzentrische Hypertrophie des Herzens, besonders des rechten, infolge Stenose des Ostium atrioventriculare sinist. Insuffizienz der Bikuspidalis. Adhäsive Perikarditis. Chron. interstitielle Nephritis mit Amyloid. Beiderseitige adhäsive Pleuritis.

Hochgradige Sklerose und Erweiterung der Pulmonalarterien, besonders der rechten und unvollständig abgeheilte Ruptur der letzteren. Thrombose derselben. Rote Induration der Lungen. Zyanose der Leber Nieren, Milz. Allgemeiner Hydrops.

V. Peter M., 34jährig., led. Bauerntaglöhner von Völs, stand wiederholt ambulatorisch und klinisch an der mediz. Klinik des Innsbrucker allgem. Krankenhauses in Behandlung.

16. IX. 1890 Ambulat. Diagnose: Insuffic. mitral. Stenorkardie?

Am 29. Januar 1892 Prot. Nr. 64/307 wurde er wegen Husten, Auswurf, leichter Dyspnöe aufgenommen.

Diagnose: Vitium cordis. Insuff. et stenosis valvul. bicuspid. Phthisis pulmonum incip.? Intumescentia glandul. lymphat. colli. Am 5. Februar wurde er in wesentlich gebessertem Zustande entlassen.

Später stellte er sich im Ambulatorium vor und zwar im Mai und mehrmals im September desselben Jahres. Als Diagnose ist Stenosis et insufficientia valvulae mitralis vermerkt.

Am 17. November 1892 kam Pat., unter Prot. Nr. 482/2793 der mediz. Männerklinik neuerdings zur Aufnahme und machte folgende Angaben:

Der Vater starb mit 78 Jahren, die Mutter mit 77. Krankheit unbekannt.

Hereditäre Verhältnisse ohne Belang.

Patient will bis ungefähr — eine genaue Angabe läßt sich nicht erzielen — zum Jahre 1874 vollständig gesund gewesen sein, um welche Zeit ihn nach einer Erkältung ein Gelenkrheumatismus befallen habe, von 3wöchentlicher Dauer, mit Beteiligung fast aller Gelenke. Nach 2 Jahren, 1876, wiederholte sich die Polyarthritis rheumatica, 7—8 Wochen dauernd. Schon nach der ersten Attacke, ganz besonders aber im Anschluß an die zweite, bemerkte er das Auftreten von Herzklopfen bei stärkeren Bewegungen und daß er damals als junger Burche seinen Altersgenossen bei weiteren Ausflügen, beim Laufen usw. nicht habe nachkommen können. Im Jahre 1878 akquirierte er angeblich einen Typhus, weswegen er in fast 2monatlicher Spitalsbehandlung verblieb.

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Die Nachforschung im Aufnahmsprotokoll ergab jedoch, daß der damals 23jähr. Kranke im Jahre 1878 vom 24. Oktober bis 30. November an der mediz. Klinik, Prot. Nr. 402/1582, wegen Endokarditis (und Perikarditis?) in Behandlung stand wobei sich der Vermerk findet, daß Pat. mit einer Insufficientia valv. bicuspidalis hinausging.

Nach dieser Zeit haben sich zuerst schwach auftretende, dann immer mehr sich steigernde Atembeschwerden bemerkbar gemacht, erst später mehr oder minder starkes Anschwellen der Füße in der Knöchelgegend, manchmal auch solches der Waden. Seit anfangs 1892 steigerten sich die Beschwerden so, daß der Mann arbeitsunfähig wurde. Neben Atemnot, Herzklopfen leidet er an Husten und Stechen in der rechten Seite, auf welcher er allein nur zu liegen imstande ist, und an Schmerzen in der Herzgegend.

Status: Pat. mittelgroß, mäßig gut genährt, klagt über starke Atemnot, Herzklopfen, Husten und geschwollene Füße.

Gesicht zyanotisch. Hals dick. Rechts, hinten und außerhalb des M. sterno-cleido-mastoideus eine weiche, pulsierende, verdrängbare, fast hühnereigroße Anschwellung. In der linken Supraklavikulargrube, wie auch in der rechten, einige vergrößerte Lymphdrüsen fühlbar.

Thorax gut gewölbt. Respiration symmetrisch, angestrengt, beschleunigt. Die Herzgegend vorgewölbt.

Die Perkusion ergibt in der rechten Supraklavikulargrube und den gleichseitigen Interkostalräumen bis zur 6. Rippe relative Dämpfung, von dort nach abwärts absolute Dämpfung bis 4 Querfinger unter den Rippenbogen in der Papillarlinie. Links oben voller Lungenschall. Die absolute Herzdämpfung beginnt im 4. Interkostalraum. Die Herzdämpfung überschreitet nach links die linke Papillarlinie um $1^1/_2$ Querfinger und reicht nach rechts bis zur Medianlinie.

Hinten über der ganzen rechten Thoraxhälfte relative Dämpfung, links voller Lungenschall. An der Herzspitze systolisches Schwirren fühlbar. Spitzenstoß im 6. I. R. palpabel.

Auskultation: Über der rechten Lunge unbestimmtes Atmen und beiderseits reichliche feuchte Rasselgeräusche. An der Herzspitze, namentlich im 6. I. R. ein systolisches und diastolisches Geräusch, welch ersteres weit nach aufwärts zu hörbar ist. Im Epigastrium pulsatorische Erschütterung sichtbar.

Abdomen ausgedehnt und freie Flüssigkeit nachweisbar.

Milzdämpfung vergrößert, namentlich im Längsdurchmesser. Die unteren Extremitäten stark ödematös geschwollen. Temp. normal. Puls frequent, ungleichmäßig, arhythmisch, sehr schwach. Harnmenge vermindert, spez. Gew. 1020. Reakt. sauer, Eiweiß reichlich.

19. XI. 1 pro mille Albumen. Nach Gebrauch von Digitalis, Strophantus und Koffein nimmt die Diurese zu, vorübergehende Besserung. Am 27. Nov. Puls kräftiger, gleichmäßiger, normale 24stündige Harnausscheidung.

Am 5. Dezember stellen sich wieder sehr starke Zyanose, dann Ödeme und Atembeschwerden ein, welche nach Digitalisgebrauch bald zurückgehen.

Am 20. Dezember waren die Ödeme ganz geschwunden, die Respiration verhältnismäßig leicht. Der Ernährungszustand läßt jedoch viel zu wünschen übrig. Von Zeit zu Zeit Anfälle von Zyanose und Oppression auf der Brust mit Schmerzen in der oberen Herzgegend in die Tiefe zu.

Die klinische Diagnose lautete:

Insufficientia et Stenosis valvulae mitralis. Hypertrophia ventriculi dextri cordis. Cyanosis. Hydrops universalis.

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Pleuritis dextra. Albuminuria.

Am 29. Dezember wird der Mann gebessert entlassen.

Nach kurzem Aufenthalt in seinem Heimatsdorf stellte sich Pat. jedoch bald wiederum an der mediz. Klinik ein und fand daselbst am 11. Januar 1893 abermals Aufnahme, Prot. Nr. 20/102 der med. Männerklinik. Die oben angeführten Beschwerden hatten unterdessen wieder ganz besonders zugenommen. Pat. leidet unter Beängstigung und Atemnot, Hydropsien haben sich abermals eingestellt, die Zyanose erreicht hohe Grade. Nachdem Digitalis 4 Tage ohne besonderen Erfolg verabreicht wurde, bekam der Kranke Jodkali, Strophantus und Kalomel, neben Milchdiät. Die Diurese erreicht dann fast die Norm. Wiederholte Schmerzanfälle in den obersten Herzabschnitten mit Angstgefühl und Unruhe. Variable rieselnd-schabende Geräusche über der Herzbasis gegen links zu. Schon während seines früheren Aufenthaltes war im Verlaufe des Dezembers bei der Auskultation des Herzens zu konstatieren, daß das diastolische Geräusch immer prägnanter wurde, ja zum Schluß prävalierte.

Auch jetzt herrschte bei der Auskultation weitaus das diastolische Geräusch, das in ziemlich weitem Umfang an der Spitze zu hören war, vor. Zeitweise war dasselbe an der Herzbasis (2. linker Rippenknorpel) am lautesten zu hören.

Stauungsbronchitis. Verkürzung des Schalles im 2. linken I. R. von der Sternalbis etwas außerhalb der Parasternallinie. Ein dumpfes, undeutliches, systolisches schwirrendes Geräusch an der Basis des Herzens, namentlich gegen den 2. linken I. R. zu, rasch wechselnd. Der Breitendurchmesser der Herzdämpfung hat nach rechts zu beträchtlich zugenommen.

Am 15. Januar Auftreten von stechenden Schmerzen L. h. und daselbst Dämpfung mit unbestimmten Atmen und feuchten Rasselgeräuschen links unten. Hämorrhagisches Sputum.

Rechts hinten unten ebenfalls Dämpfungszone mit abgeschwächten Atmen, unbestimmten Rasselgeräuschen.

Unter zunehmender Dyspnöe, hochgradiger Zyanose Exitus letalis am 23. Januar 1893 $^1/_2$2 Uhr nachts.

Die klinische Diagnose lautete:

Stenosis et insufficientia valvulae mitralis. Hypertrophia et dilatatio passiva ventriculi dextri cordis.

Cyanosis. Degener. et insuffic. cordis.

Hydrops universalis. Infarctus haemorrhag. pulmon.

Pleuritis obsol. dextr., rec. sin.

Sektion am 24. Januar 1893. Prot. Nr. 3150/16 path. anat. Diagnose:

Endocarditis chron. cum stenosi ostii atrioventricul. sin. et insuffic. valvulae bicuspidalis. Hypertrophia excentrica cordis dext. eximia. Dilatatio atrii sin. eximia, ventric. sin. lev. gradus. Infarct. suppurat. lob. inf. pulm. sin. subsequ. exsudato sero-fibrinoso sin. partim occluso. (Pneumothorax partialis.)

Thrombosis arteriae pulmonalis dextrae partim obtur.

Endarteriitis arteriae pulmonalis eximia.

Atelectasis indurat. partial. pulmon. dext. Induratio fusca pulmonum, cyanotica renum, hepatis, lienis.

Catarrh. cyanot. intestini praec. grassi et ventric.

Der größte Teil des linken Oberlappens fest angewachsen. Die rechte Lunge im ganzen Umfang ziemlich fest angeheftet.

Die rechte Lunge bis auf wenige teils rundlich fleckige, teils im Bereich großer, mit weißen Thrombusmassen verstopfter Arterien des Unterlappens strahlig

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in das Lungengewebe hineingreifender Züge, lufthältig, gedunsen, mit schaumiger Flüssigkeit infiltrirt, in den strahligen Partien im Bereich der thrombosierten Arterien teils nur atelektisch, teils fibrös verdichtet, derb.

Die thrombosierten Arterienstämme des Unterlappens, sowie mehrere erbsen- bis haselnußgroße, bräunlich pigmentierte schwielig umgrenzte Infarkte in ihrer Nachbarschaft lassen sich durch das Gewebe hindurch als Knoten fühlen.

Bronchien erweitert. Die Lunge im allgemeinen ziemlich blutarm, von eigentümlich, gelbrötlicher Färbung. Das Gewebe der Lunge durchwegs substanzreicher. Ähnlich verhält sich der Oberlappen der rechten Lunge und Teile des Mittellappens, welche lufthaltig, in ihrem Gewebe verdichtet, eigentümlich gelbrötlich gefärbt, hie und da atelektisch und fibrös verdichtet sind.

Das Perikard des rechten Ventrikels an der Vorderfläche sowie des rechten Vorhofes mit ausgebreiteten zum Teil netzförmigen Sehnenflecken überdeckt. Der Anfangsteil der Pulmonalis mit dem Herzbeutel durch feste bindegegewebige Membranen verwachsen; an der Hinterfläche des linken Ventrikels teils zottige Bindegewebsmembranen, teils strahlige Verdichtungen.

Daß Herz sehr groß, besonders breit, es mißt: 12 cm in der Länge, 15 cm in der Breite und 9—10 cm in der Dicke, ist in beiden Ventrikeln kontrahiert.

Das linke Herz erscheint als kleiner Anhang des mächtig vergrößerten rechten Herzens. Das Fettgewebe des Perikardium spärlich. Die linke Herzhöhle mit locker geronnenem Blute strotzend erfüllt. Das Ostium atrioventricul. nur für 1 Finger durchgängig. In den rechten Herzhöhlen mäßige Mengen lockerer Blutgerinnsel und Fibrin. Ostium atrioventriculare dext. sehr weit.

Valv. tricuspidalis bis auf feine warzige Exkreszenzen am Rande ohne wesentliche Veränderung. Die Valvula bicuspidalis hingegen ist in allen ihren Teilen verdickt, verkürzt, der Aortazipfel von knotigen verkalkten Einlagerungen eingenommen, die zum Teil auf der Oberfläche hervorragen; besonders im linken Teile feine warzige Exkreszenzen. Die Sehnenfäden zum Teil miteinander verwachsen, zum Teil verdickt. Die Ansätze der Sehnenfäden an der hinteren Fläche des Aortenzipfels zu einem nur durch eine kleine Spalte geteilten, 1 cm breiten, 2—3 mm dicken Seile vereinigt. Die Papillarmuskeln nicht auffällig verändert. Konus der Pulmonalis mächtig erweitert. Die Wand bis zu 1 cm dick. Die Trabekel bis zu fast kleinfingerdicken Strängen vorspringend. Die Klappe der Art. pulmonalis blaß, zart.

Aortaklappen schließen und sind bis auf geringe Verdickung und Herabzerrung nicht wesentlich verändert. Koronarostien weit.

Linker Vorhof sehr weit, das Endokard strahlig weiß verdichtet.

Der linke Ventrikel in den unteren Teilen kaum 1 cm dick, in den oberen $1^1/_2$ cm, während die Wand des rechten Ventrikels 12 mm dick ist. Herzmuskel braunrot.

Die oben angegebenen Thromben in den Arterien des rechten Unterlappens lassen sich in Form randständiger, fest fixierter, meist gemischter, ziemlich derber Thromben hinauf verfolgen bis an die Abgangsstelle der Äste II. Ordnung. Diese und die kleinen Pulmonalarterien zeigen strahlig-fleckige Sklerosierungen der Intima (Museumpräparat, alte Signatur C 93 d, neue C 135 des pathol. anatom. Museums).

VI. Georg L., 29 J., led. Schankbursche, stand vom 27. März bis 5. Mai 1902 an der medizin. Klinik wegen eines schweren Herzfehlers in Behandlung. Prot. Nr. 273/1324 der med. Männerklinik.

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Die Anamnese ergab, daß der Vater des Pat. mit 56 Jahren einem Herzleiden erlag, Mutter und 4 Geschwister leben.

Er selbst hat vor 3 Jahren Gelenkrheumatismus, vor 2 Jahren eine Rezidive überstanden, wobei sich Herzerscheinungen meldeten.

In der Folge alle Symptome eines sich entwickelnden Herzklappenfehlers.

Der Mann, von gutem Ernährungszustand, kräftig gebaut, zeigt schon in gewöhnlicher Bettlage beträchtliche Dyspnöe und ganz außerordentlich starke Zyanose. Leicht subikterische Verfärbung der Skleren.

Enorme Herzhypertrophie, speziell des linken Ventrikels. Wogend undulierende Herzaktion in verbreitertem Umfang, hebender Spitzenstoß. Schwirren in beträchtlicher Ausdehnung an der Herzspitze. Daselbst lang gezogenes systolisches Geräusch, das gegen den Sternalrand zu rasch abnimmt. An der Basis außerdem ein kurzes, scharfes, diastolisches, ziemlich rauhes Geräusch über der Aorta, das sich auch gegen die linke Sternallinie zu fortpflanzt. Pulsus celer. Arterienphänomene. Die der Palpation zugänglichen Arterien vielleicht eine Spur rigider, namentlich im Verhältnis zum jugendlichen Alter.

Die klinische Diagnose lautete: Insuffic. semil. aortae. Hypertrophia excent. ventric. sin. Insuff. valv. bicusp. (relat.?). Hypertrophia ventr. dext. Insuffic. cordis. Arteriosclerosis.

Während des Spitalaufenthaltes stellten sich wiederholt Schmerzen unter dem Brustbein einhergehend mit Angst- und Beklemmungsgefühl ein. Der Charakter derselben war zu wenig ausgesprochen, so daß es in suspenso bleiben mußte, ob es sich um wirkliche stenokardische Anfälle oder um Retrosternalschmerz bei Arteriosklerose der Aorta handelte. Die Zyanose zeigte periodische Verschlimmerungen.

Manchmal waren die Geräusche an der Herzbasis eigentümlich schnurrend, von förmlich musikalischem Charakter.

Kardiaka. Nach Bedarf Exzitantien.

Im weiteren Verlauf Zunahme der Oppression, Dyspnöe und Zyanose und der Stauungserscheinungen. Auftreten von Ödemen, Stauungsbronchitis. Infarzierung der Lunge.

Unter den Symptomen höchstgradiger Herzinsuffizienz Exitus letalis am 5. Mai.

Sektion am 6. Mai 1902. Prot.-Nr. 6066/108. Herzbefund (auszugsweise): Vernarbte Endoarteriitis der Aorta und ihrer Klappen mit Loslösung des Insertionsgebietes des Taschenrandes der hinteren Semilunarklappe der Aorta und Umstülpung des dadurch frei gewordenen mittleren Teiles der hinteren Klappe.

Exzentr. Hypertrophie, besonders des l. Ventrikels.

Stauungsinduration der Lunge. Infarkt im Mittellappen der r. Lunge.

Art. coron. sin. sehr eingeengt, rechte erweitert.

Im Wurzelgebiet der Pulmonalarterie eine die Intima in ihrem ganzen Umfang betreffende, sich auf $1^1/_2$—2 cm in das Gefäß erstreckende Trübung und weißliche endoarteriitische Verdickung der Intima.

VII. Anton P., 55 J., verh. Schlosser (Prot. Nr. 282/1388), am 2. April 1902 an der medizin. Klinik aufgenommen.

Familienkrankheiten kennt der Pat. keine.

Mit 19 Jahren überstand er Variola.

Seit ungefähr 1 Jahr verspürt er heftige Schmerzen in der linken Seite,

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speziell in der Herzgegend, und Atemnot. Herzklopfen fehlt. Mitunter Hustenreiz. Ödeme bestehen seit ungefähr 4 Wochen an beiden Beinen. Der Schlaf ist durch asthmatische Anfälle gestört.

Status: Sehr großer, kräftig gebauter Mann von entsprechendem Ernährungszustand. Allgemeiner Hydrops. Hochgradige Ödeme an den unteren Extremitäten.

Mäßige Anasarka der Bauchdecken. Gesicht anämisch, Lippen und Wangen zyanotisch, Skleren zeigen leicht gelblichen Stich.

Hals kräftig, Thorax breit, gut gewölbt. Herzgegend stark vorgewölbt, bei etwas kräftigerer Perkussion schmerzhaft. Die Herzdämpfung reicht nach rechts bis zur Parasternallinie, nach links $1^1/_2$ Querfinger über die linke Papillarlinie. Arhythmie. Leise Töne. Über dem oberen Teil des Sternum und dem Manubrium langes diastolisches Geräusch. Arterien rigid. Pulsus celer. Temp. subnormal.

Sensorium nicht ganz frei.

Klinische Diagnose: Insufficientia semilunarum aortae. Arteriosclerosis. Hypertrophia ventric. sin. cordis. Dilatatio cordis passiva. Degeneratio et insufficientia myocardii. Volumen pulmonum auctum. Bronchitis. Cyanosis. Intumescentia hepatis. Induratio cyanotica hepatis et renum. Stauungsalbuminurie. Hydrops universalis. Icterus.

Therapie: Digitalis, Diuretin, Kalomel, Agurin, Jodnatrium (im Verlaufe des ganzen Aufenthaltes) abwechselnd bei entsprechender Indikation zur Anwendung gelangt.

Druckgefühl, Beklemmung in der Herzgegend, Gefühl von Enge, Stechen, manchmal Zusammenschnüren, Brennen in der Tiefe am Sternum in den oberen Abschnitten. Keine Anfälle von Herzbräune.

Nach und nach übertrifft die bläuliche Verfärbung das anämische Aussehen. Wiederholt nach Körperbewegungen Attacken von Zyanose mit Angstgefühlen, Beklemmungen und Herzschmerzen, dabei sehr unregelmäßige Herztätigkeit. Das diastolische Geräusch in verbreitertem Umfange nach links im 2. I. R. bis über die Parasternallinie hörbar.

20. IV. Ödeme und Zyanose beträchtlich zugenommen.

Passive Dilatation des Herzens, l. Ventrikel reicht bis zur vorderen Axillarlinie. An der Herzspitze sehr dumpfe, undeutliche, verschwommene Töne. In der Papillarlinie ein aus der Ferne kommendes diastolisches Geräusch, das nach aufwärts zu immer deutlicher wird. Über dem Sternum ein kurzes, undeutliches, weiches, systolisches und ein sehr ausgesprochenes, langes, scharfes, diastolisches Geräusch. Am 21. wurde wegen den enormen Hydropsien die Kapillarpunktion an den unteren Extremitäten vorgenommen, durch welche am 21. und 22. über 10 l Flüssigkeit entleert wurden. Dieselbe zeigte ein spez. Gew. von 1007 und war nur mäßig eiweißhaltig.

Die Besserung war nur von geringer Dauer. Rasche Zunahme der Ödeme, Benommenheit. Hämorrhagien am Handrücken und am linken Unterschenkel.

Anfangs Mai zeigten sich sehr ausgebreitete Hauthämorrhagien, besonders am Fuß- und Handrücken und an den Unterschenkeln. Am 7. beträchtliches Infiltrat am l. Unterschenkel, Abhebung der Epidermis. Tags darauf Bildung zweier gaseigroßer, mit blutigem Inhalt erfüllter pemphigusartiger Blasen in der Gegend des linken Sprunggelenkes und des Fußrückens.

Ikterische Verfärbung ausgesprochen. Unter Zunahme des Ikterus, der Zyanose, des Hydrops, Somnolenz und Schwäche Exitus letalis am 12. Mai 1902.

Sektion am 4. Mai 1902. Prot.-Nr. 6072/116.

Diagnose: Hochgradige Aortensklerose und Dilatation mit sklero-

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tischer Schrumpfung der Aortenklappen. Insuffizienz derselben. Exzentrische Hypertrophie des linken und des rechten Herzens hohen Grades mit braunatrophischer und fettiger Degeneration.

Allgemeine Zyanose mit auffallenden Hämorrhagien der allgem. Decke besonders und pemphigusartiger Blasenbildung.

Kruppöse Pneumonie des r. Unter- und l. Oberlappens, hier kombiniert mit hämorrhagischer Infarzierung. Fettnekrose des Pankreas.

Herz mächtig vergrößert, schlaff, von abgestumpfter Form, mißt vom Ursprung der Pulmonalis bis zur Spitze 16, im Querdurchmesser 17 cm, der Dicke nach 8 bis 9 cm. Gewicht des Organs 850 g. — Atrioventric.-Ostien so weit, daß sie für 4—5 Finger durchgängig sind. Ihre Klappen groß, zart, ausgezerrt. —

Die Innenfläche der bis 3 mm dicken, hochgradig erweiterten Aorta, sowie deren große Äste von ausgebreiteten netzförmigen und streifigen Verdickungen eingenommen. An zahlreichen Stellen Verkalkungsplatten in die höckerig unebene sklerotische Intima eingelagert.

Die Koronararterien weit, jedoch nicht artiosklerotisch verändert.

Klappen der Pulmonalis zart, schließen. Konus weit.

In den größeren Ästen der r. Pulmonalis nur flüssiges und locker geronnenes Blut. Keine Thromben bemerkbar. Stammgebiet der Arteria pulmonalis stark fleckig sklerosirt. Beim Aufschneiden der mittleren Äste finden sich einzelne Trübungen und leichte sklerotische Verdickungen.

VIII. Emilie St., 43j. led. Bonne, kam am 13. November 1903 an der medizin. Klinik zur Aufnahme und machte folgende Angaben:

Der Vater starb an Darmtuberkulose, die Mutter an Gedärmverschlingung. Pat. machte mit 10 Jahren Blattern, mit 16 Jahren linksseitige Pneumonie durch; sie war immer sehr schwächlich und zu Lungenerkrankungen disponiert.

Seit Jahren leidet die Kranke an periodischen Kopfschmerzen, Erbrechen und Herzpalpitationen, Herzschmerzen und zeitweiligen stärkerem Angstgefühl. Früher will sie am Urin keine besonderen Abnormitäten bemerkt haben, erst seit ihrem ca. 6tägigen Aufenthalt in Innsbruck lasse derselbe eine beträchtliche Trübung und Verminderung erkennen. Gleichzeitig haben sich Herzklopfen, Kopfschmerzen und Schwächegefühl verschlimmert. Seit einigen Tagen Anschwellung der Beine und etwas schwererer Atem.

Aufnahme am 13. Nov. 1903.

Pat. groß, kräftig, gut genährt. Allgemeine Hautdecke anämisch. Ödem in der Fußknöchelgegend.

Livide Gesichtsfarbe, anfallsweise stärkere Zyanose. Zeitweise wieder mehr anämisch.

L. h. u. verkürzter Schall, trockene und feuchte Rasselgeräusche. Herz nach rechts und links verbreitert, Spitzenstoß außerhalb der Mammillarlinie. Herzaktion verbreitert, stürmisch, unregelmäßig. Nur mit Mühe hie und da undeutliche systol.? oder diastolische Geräusche wahrzunehmen.

Pulsationen im Epigastrium. Puls angedeutet celer, hochgradig arhythmisch (ca. 130 i. d. M.). Wiederholte Tachykardieanfälle bis 145. Übelkeit. Schwindel. Harn sehr stark vermindert, 300 cm. Albumen $4^1/_2{}^0/_{00}$. Spärliche Formelemente (Zylinder), Nierenepithelien und Eiterkörperchen. Stuhl unregelmäßig, eher angehalten.

Inf. digital.

14. XI. Puls 166! nur sehr schwer fühlbar. Natr. brom. Digital. Eisbeutel. Leichter Exophthalmus.

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In der Folge Zunahme der Kopfschmerzen, schlechtes Sehen, Schwindel, Tachykardieanfälle höchsten Grades. Enorme Arhythmie. Heiserkeit. Erbrechen. Oppression in der Herzgegend mit anfallsweisen dumpfen Schmerzen daselbst.

22. XI. L. h. u. gedämpfter Schall, verstärkter Pektoralfremitus. Blutiges Sputum. 23. Erbrechen, starke Ödeme an den Beinen. 24. Kältegefühl in den Beinen. Die linke untere Extremität fühlt sich kühl an. Verschlechterung des Sehvermögens. Benommenheit, weiterhin Bewußtlosigkeit. Kalte Schweiße, höchstgradige Pulsschwäche. Kampferinjektionen. Hochgradige Zyanose des Gesichts. Auch das r. Bein schwillt an. Zunehmender Kollaps. Am 25. XI. 1 Uhr nachts Exitus letalis unter Erscheinungen von Lungenembolie (?).

Die klinische Diagnose lautete: Vitium cordis. Stenosis (et insuffic.?) valv. mitralis. Myodegeneratio et insufficienta cordis hypertrophicae. Anaemia. Cyanosis. Nephritis parenchym. chron. Uraemia. Bronchopneumonia. Thrombos. crur. sin. Infarct. haemorrh. pulm. (?).

Die am pathologisch-anatomischen Institut am 26. November 1903 vorgenommene Sektion (Prot.-Nr. 6543/274) ergab nachstehenden Befund (auszugsweise).

Patholog.-anatomische Diagnose:

Stenose des Ostium atrio-ventric. sin. und Insuffizienz der Valv. bicuspidalis infolge chron. Endokarditis mit exzentr. hochgradiger Hypertrophie des r. Herzens, Dilatation der Arteria pulmonalis und ihrer Äste und Arteriosklerose derselben bei Synechien beider Lungen, insbesondere des Unterlappens mit Fixation der umgekrämpelten Ränder rechterseits nach vor 10 J. überstandener Pleuropneumonie.

Obturierende Thrombose der Art. pulmonalis des l. Unterlappens. Wandständiger erweichter Thrombus der rechten Art. pulm. mit ausgedehnten hämorrhag. Infarzierungen, besonders d. r. Unterlappens (ohne Hämoptöe-Erscheinungen).

Thrombose des l. Herzohrs. Embolie der Aorta abdom. an der Bifurkation und der Teilung der r. Iliaca comm. mit fortgesetzter frischer und älterer Thrombose in der l. bzw. r. Iliaca externa. — Arterioskler. nephrit. Narben in beiden Nieren bei hochgrad. Arteriosklerose der Ursprungsstellen der Äste der Aorta abdom., der Art. renalis.

Sekundäre Tracheo-Bronchiektasien in beiden Unterlappen. Lungenränder z. T. angewachsen an das mediast. Pleurablatt. Lungenränder berühren sich, r. 3 l klarer seröser Flüssigkeit. Links 2—3 l.

Herz:

Länge des Herzens 9,5 cm, r. Ventrikel 11 mm dick.

Dicke des l. Ventrikels 14 mm. Breite des Herzens 10 cm.

Umfang der Aorta 9,5—10 cm.

Umfang der Arteria pulmonalis oberhalb der Klappen 8 cm.

R. Ventrikel sehr verdickt, beide Ventrikel kontrahiert. Ost. atrioventric. sin. für den kleinen Finger durchgängig.

Die Ränder der Klappen besetzt mit rauhen verkalkten Besätzen. Die Sehnenfäden auf 1½ cm Kürze reduziert. Hintere Klappe in ganzer Ausdehnung verdickt. Stamm der Art. pulmonalis frei. Der Durchmesser sehr stark bis auf 9 cm und darüber erweitert.

Die Art. pulm. enthält in ihrem Stamme und Teilungsstelle nur Leichengerinnsel. Aus dem r. ersten Aste und aus dem großen unteren Aste der linken Lunge ragen thrombot. Massen vor, welche den linken unteren Ast total obturieren und z. T.

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ziemlich innig an der Arterienwand angeheftet sind. Neben dem Thrombus der r. Pulmonalis bleibt noch ein ziemlich großer Teil des Lumens offen.

Dieser Thrombus ist ebenfalls fixiert, dabei aber bis auf eine dünne Randschichte zu einer chokoladebraunen dünnen Flüssigkeit erweicht. In der Fortsetzung dieses Thrombus die unteren Äste des r. Unterlappens durch Thrombusmassen total obliteriert.

Thrombose des l. Unterlappens nur in beschränkter Ausdehnung. In beiden Ästen der Pulmonalarterie außer der hochgradigen Erweiterung und relativen Dünnheit der Wände z. T. vorragende linsengroße, gelbe Flecken bemerkbar. Solche Felder auch verfolgbar in die Arterien des linken Oberlappens, wo sie frei von Thromben sind. Die oberen nicht thrombosierten Äste der r. Pulmonalarterie zeigen sehr auffällige sklerotische Flecken.

Über einen weiteren Fall von hochgradiger Sklerose der Pulmonalarterie bei Stenose und Insuffizienz der Valvula bicuspidalis (und geringgradiger Insuffizienz der Aortenklappen), ebenfalls ein noch jugendliches Individuum betreffend, kann in Kürze nachstehendes berichtet werden.

IX. Johann P., 22j. lediger Schneider von Telfs, aufgenommen an der medizinischen Klinik am 17. November 1902 (Prot.-Nr. 807/4871).

Schon im Jahre 1897 suchte er ambulatorisch Hilfe wegen seiner Herzbeschwerden (18. Mai 1897), wobei ihm schon damals Spitalsaufnahme empfohlen wurde. Er verblieb bis 3. Juli 1897 in klinischer Behandlung. Diagnose: Stenosis et insufficientia valvulae bicuspidalis. Cyanosis permagna.

Auch in der Folge frequentierte er ab und zu das Ambulatorium, wobei sich stets deutlich die Zeichen des genannten Herzfehlers mit starker Zyanose, jedoch ohne Dyspnöe und sonstigen auffälligeren Stauungssymptomen ausgeprägt fanden.

Aufnahme 1902. Anamnese:

In der Kindheit habe Pat. Freisen gehabt. Sonstige Krankheiten sollen keine bestanden haben. Im 12. Lebensjahr stellte sich Herzklopfen bei rascheren Bewegungen, namentlich beim Laufen ein, zu welchem sich in späteren Jahren erschwertes Atmen hinzugesellte. Schwerere Arbeiten brauchte er nicht zu verrichten. Anfangs lernte er Buchdruckerei, später wurde er Schneider.

Nach der Spitalsbehandlung 1897 (s. o.) fühlte er sich einige Jahre besser. Seit 1 Jahr verschlechterte sich sein Befinden. Er bekam öfters Schwindelanfälle, es wurde ihm dann schwarz vor den Augen. Diese Anfälle steigerten sich manchmal bis zur Bewußtlosigkeit. Eine Anschwellung der Füße bildete sich auf medikamentöse Behandlung wieder zurück.

Seit Monatsfrist verschlimmerte sich sein Zustand derart, daß er wieder Spitalshilfe aufsuchte.

Seine Beschwerden bestehen in Atemnot, Schwäche, heftigem Herzklopfen, Ödemen der Beine. Außerdem klagt er über ein spannendes Gefühl im Abdomen, Schmerzen in der Herz- und Lebergegend, Husten.

Status: Mittelgroßer, kräftig gebauter, ziemlich gut genährter Mann mit kräftig entwickelter Muskulatur. Zeitweise Dyspnöe. Sehr starke Zyanose der Wangen, Ohren, Nase, Lippen. Diese Teile, ebenso die Finger, Knie und Zehen fühlen sich kühl an. Thorax ziemlich breit, mittellang, mäßig gewölbt.

Perkussionsschall h. und namentlich rechterseits etwas verkürzt. Über diesen Partien sehr zahlreiches feinblasiges und Knisterrasseln, über den übrigen Lungenpartien mittelblasige unbestimmte Rasselgeräusche.

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Mäßiger Herzbuckel. Spitzenstoß im 5. I. R. außerhalb der Papillarlinie. Kein Schnurren oder Schwirren bei der Palpation wahrnehmbar. Herzdämpfung beginnt am unteren Rand der 3. Rippe, reicht nach rechts etwas über den rechten Sternalrand hinaus, nach links bis zur Mammillarlinie.

Am linken Sternalrand von der Ansatzstelle des 2. linken Rippenknorpels bis in die Herzdämpfung übergehend eine ca. 1—1½ Querfinger breite Dämpfungszone angedeutet. Auf stärkere tiefere Perkussionsschläge hier eine gewisse Druckempfindlichkeit, die anscheinend bei vorgebeugtem Oberkörper ausgesprochener wird. Verbreiterte unregelmäßige Herzaktion sichtbar.

Bei der Auskultation kann man etwas unten und außen von der Mammilla ein lautes systolisches, blasendes Geräusch konstatieren, daneben öfters auch ein präsystolisches, selten auch deutlich diastolisches blasendes Rauschen. In der Ruhe die Geräusche nicht besonders stark; bei Bewegungen, namentlich bei Rumpfbeugen tritt das diastolische Geräusch stärker hervor und wird dann mehr nach oben gegen den linken Sternalrand zu bis zum Ansatz des 3. linken Rippenknorpels als schärferes, lauteres, hauchend-schabendes Geräusch wahrgenommen.

Während der Bettruhe nur geringe, bei selbst mäßigen Bewegungen ausgesprochene Arhythmie.

Die Zyanose steigert sich beim bloßen Aufrichten im Bett zu beträchtlicher Intensität. Mitunter direkte Anfälle von tief schwarzblauer Verfärbung, ohne gleichzeitige Steigerung der Atemnot.

Puls sehr klein, weich, leicht unterdrückbar, arhythmisch. Blutdruck 70 mm Hg.

Periphere Arterien kaum, Arteria radialis vielleicht eine minimale Spur rigider.

Die Leberdämpfung reicht in der Mammillarlinie gut 1½ Querfinger über den Rippenbogen. Die Lebergegend bei der Palpation etwas schmerzhaft. Milzdämpfung sowohl im Breiten-, wie im Längsdurchmesser bedeutend vergrößert.

Sputum schaumig-schleimig. In demselben hie und da kleine bräunliche Stellen bemerkbar, die mikroskopisch Herzfehlerzellen aufweisen.

24stündige Harnmenge verringert. Harn hochgestellt, leicht getrübt, braunrot, enthält Eiweiß in mäßiger Menge.

Die tägliche Harnausscheidung ziemlich variabel.

Stuhlgang etwas unregelmäßig. Temperatur normal.

Respiration 25—28. Puls 80—90.

Kardiaka. Agurin. Worauf die Harnmenge vorübergehend etwas steigt, um nach einigen Tagen wieder zu sinken.

Im weiteren Verlauf rasche Zunahme der Kompensationsstörungen. Die Herzdämpfung überschreitet fast die rechte Parasternallinie. Passive Dilatation und Hydroperikard. Deutlicher Flüssigkeitserguß im Abdomen. Die Hydropsien nehmen rasch zu trotz aller angewandten Mittel (Diuretin, Kalomel usw.).

Die Arhythmie nimmt zu. Beim Aufrichten des Oberkörpers und öftern Vorwärtsbeugen des Rumpfes erscheint das diastolische Geräusch von größerer Intensität am linken Sternalrand mehr gegen die Herzbasis zu.

Unter Zunahme aller Stauungserscheinungen erfolgt am 27. November 7 Uhr früh der letale Ausgang.

Als klinische Diagnose wurde vermerkt:

Stenosis et insufficientia valv. mitralis subsequ. hypertrophia cordis praecip. ventric. dext. Stauungsbronchitis. Oedema pulm. chron. Cyanosis. Stauungskatarrh des Magens. Induratio cyanot. hepatis et renum. Stauungsmilztumor. Hydrops universalis.

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Der Umstand, daß der Kranke schon in sehr weit vorgeschrittenem Zustand zur klinischen Aufnahme kam und nicht ganz 2 Wochen bis zum letalen Ende vergingen, erschwerte die Analyse. In den epikritischen Notizen finden die enorme Zyanose, die eigenartigen Anfälle Würdigung.

Nachdem bereits vor 5 Jahren die ausgesprochensten Zeichen der Mitralstenose vorlagen und auch bei den ambulatorischen Vorstellungen diese auffallenden Symptomenkomplexe bestanden, dachte man daran, ob nicht hier auch ein derartiger Prozeß zugrunde lag. Die kurze Beobachtungszeit und der desperate Zustand, in welchem er zur Aufnahme kam, erlaubten jedoch keine genauere Präzisierung.

Sektion 1. Dezember 1902 (Prot.-Nr. 6240/290).

Patholog.-anatomische Diagnose:

Hochgradige exzentrische Hypertrophie des r. Herzens bei Stenose und Insuffizienz der l. Atrioventricular-Klappe.

Geringradige Insuffizienz der Aortenklappen infolge vernarbter Endokarditis. Geringgradige Endocarditis verrucosa der Pulmonalklappen, hochgradige Sklerose der Arteria pulmonalis. Hochgradige Stauungsinduration der Lunge, der Milz, der Leber und der Niere.

Stauungskatarrh in den Bronchien und im Digestionstraktus. Ödem und Anasarka. Hydroperikard und Hydrops ascites. Adhäsive Pleuritis, Perihepatitis und Perisplenitis. Adhäsive Pachymeningitis mit mäßiger Hyperostose der Tabula vitrea.

Allgemeines Ödem und hochgradige Zyanose.

Die r. Lunge im ganzen Umfang, die linke besonders in den unteren und seitlichen Teilen angewachsen, im frei gebliebenen Teile der l. Thoraxhöhle etwa 1 l klare seröse Flüssigkeit.

Im Herzbeutel 500 g klare, seröse Flüssigkeit von rötlicher Färbung.

Herz sehr groß, namentlich stark verbreitert. Spitze vom r. Ventrikel gebildet. Herz bei 15 cm lang, 16 cm breit, im r. Ventrikel ca. 12 cm dick. Gewicht des Herzens 627 g. Epikard im Bereiche der vorderen Koronararterienäste des r. Ventrikels mit Verdickungsstreifen besetzt.

Solche finden sich auch an der Pulmonalis. Umfang der Aorta an der Basis in der Gegend des Ansatzes der Klappen 6,5 cm. Umfang der Pulmonalis an der entsprechenden Stelle 9,5 cm.

Das Herz schlaff, in seinen Höhlen strotzend erfüllt mit flüssigem und locker geronnenem Blut und Fibringerinnseln.

Das Ostium atrioventric. sin. kaum für die Kuppe des Zeigefingers durchgängig. Das Ostium atrioventr. dextr. bequem für 4 Finger durchgängig. Die beiden Segel der Bikuspidalis miteinander verwachsen und außerdem die Sehnenfäden zumeist untergegangen in einem dicken, kurzen, kaum 0,5 cm langen Strange, welcher im verdickten unteren Gebiet des Klappensegels inseriert. Der obere Teil des im ganzen 3 cm langen Aortensegels dünn, durchscheinend, wie ausgezerrt. Papillarmuskeln ziemlich dick. Dagegen die Wand des l. Ventrikels kaum 11—12 mm dick. Der ziemlich stark verdickte linke Vorhof mit weißem Endokard ausgestattet. Die Trikuspidalis zart und nicht besonders verändert, vorderer Zipfel etwas stark ausgezerrt, wie vergrößert.

Die Wand des erweiterten r. Ventrikels mißt im Konusteil 1 cm, im Spitzenteil bis 14 mm.

Pulmonalarterie, auch im Ursprungsgebiet mit sklerotischen Flecken ausgezeichnet, hat einen Umfang von 9 cm. Die Aorta hingegen auffallend enge, zeigt nur einen Umfang von 5,5—6 cm.

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Klappen der Aorta, zwischen denen eingegossene Flüssigkeit allmählich abfließt, erweisen sich im Durchmesser und Längsrichtung verkürzt, besonders in ersterem. In ihren Kommissuren verwachsen, bis auf die ziemlich weiten Fundusgebiete der Taschenbuchten höckerig verdickt und eingekerbt. Hier und da kleine rötliche Fleckungen an denselben bemerkbar. In größerer Ausdehnung eine feine Auflagerung von Hellergröße geronnenen Blutes im Bereiche feinwarziger Erhabenheiten an der linksseitigen Pulmonalarterienklappe bemerkbar. Hochgradige Sklerose der Intima der Pulmonalarterie. Herzohren enthalten Fibringerinnsel. Die Koronarostien von gewöhnlichen Dimensionen.

Die Aorta in ihrem Bereiche und auch im Bogenteil nur mäßig diffus sklerosiert. In den großen Ästen zerstreute sklerotische Flecken. Der Umfang der Brustaorta beträgt 4 cm.

(Präparat des pathol.-anatomischen Museums Signatur C 122.)

X. Vom Jahre 1897—1904 stand zu wiederholten Malen in ambulatorischer und klinischer Behandlung der medizinischen Klinik Marie S., damals (1897) 28jährige Malersfrau.

Mehrere Jahre magenleidend. Zugleich mit Magenschmerzen habe sich Herzklopfen eingestellt, namentlich bei anstrengenden Arbeiten. Nach und nach hätten sich die Herzbeschwerden verschlimmert. Mitunter stechende Schmerzen in der Herzgegend mit Oppressionsgefühl.

Gelenkrheumatismus hat die Kranke nie gehabt, Lues wird negiert.

Die wiederholten eingehenden Untersuchungen ergaben als klinische Diagnose: Insufficientia valvulae mitralis mit mäßigen Stenosenerscheinungen. Hypertrophie des rechten Ventrikels. Auffallende Zyanose bei Fehlen stärkerer Stauungserscheinungen, Ödemen und Dyspnöe.

Ende der 90er Jahre gelang es des öfteren, die mäßigen Kompensationsstörungen durch Ruhe, entsprechendes Regimen und Medikation wieder zu beheben.

Bei jedesmaliger Neuaufnahme traten allmählich die Stenosensymptome an der Mitralklappe in den Vordergrund, so daß während der klinischen Aufenthalte und der häufigen ambulatorischen Behandlung seit 1901 weitaus die Stenose im klinischen Bilde überwog.

Während eines klinischen Aufenthaltes im November 1901 trat vorübergehend Ikterus mit Leberschwellung auf. Die ersten Zeichen von Ödemen machten sich erst im Januar 1902 bemerkbar, gingen jedoch auf Digitalis rasch zurück.

Aus dem Status im November 1901 sei nachstehendes hervorgehoben: Pat. sehr klein, von schlechtem Ernährungszustand, enorme Zyanose, Wangen und Lippen tief dunkelblau.

Jetzt auch zeitweise etwas stärkere Dyspnöe.

Herzdämpfung im 3. I. R. bereits absolut bis zum oberen Rand der 6. Rippe; nach rechts bis zur rechten Parasternal-, nach links überschreitet sie um 1 Querfinger die Papillarlinie.

Eine Spur kürzeren Schalles in Streifenform am linken Sternalrand von der Ansatzstelle des 2. linken Rippenknorpels abwärts von ca. 1—1½ Querfingerbreite.

An der Herzspitze systolisches und ausgesprochenes diastolisches Schwirren mit der aufgelegten Hand fühlbar. Spitzenstoß in der Breite einer Fingerkuppe knapp außer der 1. Papillarlinie im 5. I. R.

An der Herzspitze undeutlich kurzes, weiches, systolisches und ausgesprochenes, schärferes, langgezogenes, mehr schabendes diastolisches Geräusch bis außerhalb der Papillarlinie zu verfolgen. 2. Pulmonalton etwas akzentuiert.

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Epigastrische Pulsation angedeutet. Herzperkussion unten nicht schmerzhaft. Am oberen linken Sternalrand auf Druck kaum, auf rasche, tiefe Perkussionsschläge leicht empfindlich.

Periphere Arterien zeigen keinerlei Schlängelung oder Rigidität. Puls klein, weich, leicht zusammendrückbar, mäßig arhythmisch.

Die Kranke wurde zu wiederholten Malen in der Vorlesung, den klinischen Visiten und in Kursen vorgestellt, wobei speziell das allmähliche Überwiegen der Stenose über die Insuffizienz der Mitralklappe und das Bestehen auffälliger Zyanose hervorgehoben wurde. Gerade die Differenz zwischen der anfallsweise hochgradigen Zyanose und der verhältnismäßig sehr geringen Dyspnöe, Fehlen von Ödemen und sonstigen Stauungssymptomen war durch Jahre hindurch immer wieder zu konstatieren.

Zum ersten Male gab ich im November 1901 meiner Vermutung, daß hier bei der Kranken mit hochgradiger Bikuspidalstenose und Insuffizienz und konsekutiver Hypertrophie des rechten Ventrikels eine Endarteriitis und Arteriosklerose der Pulmonalis bestehe, Ausdruck.

Im weiteren Verlaufe der eingehenden klinischen Beobachtungen der Kranken, die in der Folge noch oft klinische und ambulatorische Hilfe suchte, wurde diese Vermutung durch allmähliches Deutlicherwerden aller Zeichen, die ich für diese klinische Diagnose verwerten zu können glaubte, immer mehr befestigt, so daß ich diesen Krankheitsfall in den klinischen Aufnahmsprotokollen und im Ambulatorium stets mit folgender Diagnose führte: Stenosis et insufficientia valvulae mitralis, subsequ. hypertrophia ventriculi dextri cordis.

Cyanosis permagna. Endarteriitis et arteriosclerosis pulmonalis. Unter welcher Diagnose die Frau wiederholt demonstriert wurde.

Während der klinischen Aufnahme im Sommer 1902 (14.—19. VII.) machte sich, neben der hochgradigen Blausucht, Dyspnöe und geringes Ödem an den Fußknöcheln bemerkbar.

Dabei war der Puls verhältnismäßig nicht schwach, jedoch leicht unregelmäßig. Die Herzdämpfung überschreitet jetzt nach rechts die Parasternallinie.

Das kurze, dumpfe systolische Geräusch an der Herzspitze tritt immer mehr an Stärke zurück, während das diastolische Geräusch mit seiner größten Intensität mehr gegen den linken Sternalrand zu rückt und viel lauter, rauh, schabend wird.

Über dem Pulmonalostium ein leises, leicht rieselndes Geräusch, das sich an keine bestimmte Phase hält. Akzentuierung des 2. Pulmonaltones.

Auf Digitalis, Brom, Bettruhe, Eisbeutel usw. gehen in Bälde die Ödeme zurück, der Atem wird freier, der Appetit besser. Die Zyanose zeigt am wenigsten einen Nachlaß. Bei rascheren Körperbewegungen oder psychischen Momenten Anfälle von stärkerer Blausucht mit Angstgefühl, Beklemmungen und Schmerzen in der Oberherzgegend.

Die Kranke verlangte nach jedesmaliger Erleichterung ihres Zustandes schon nach kurzem immer wieder ihren Austritt.

Beim Verlassen des Spitals am 19. Juli waren die Geräusche viel weniger intensiv, zeitweilig fast verschwunden, dafür der 1. Ton an der Spitze akzentuiert, und der 2. unreine Ton mit leisem diastolischen Geräusch verdoppelt.

Bei Körperbewegungen tritt auf kurze Zeit das diastolische Geräusch weiter nach oben zu am Sternalrand (3. I. R.) stärker und schärfer hervor.

Im Verlaufe der Jahre 1902 und 1903 erfolgte noch mehrmals ambulatorische und klinische Behandlung. Das Befinden verschlechterte sich allmählich. Dyspnöe und Ödeme nahmen etwas zu, gingen jedoch nach entsprechender Behandlung

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wieder zurück. Einige Male überfielen die Kranke Angstzustände mit Erblassen und heftigen Schmerzen an der Herzbasis, zugleich kleiner, unregelmäßiger Puls.

Die ganz besonders hochgradige Zyanose steigerte sich periodisch, wobei unter Auftreten stärkerer Arhythmie dumpfe Schmerzen in der Herzgegend bestanden. Die Kranke klagt dann über ein zusammenschnürendes Gefühl und in die Tiefe der linken Seite ausstrahlende Schmerzen, die in der oberen Herzgegend ihre höchste Intensität erreichen. Gewöhnlich stemmte sie sich dann mit den Ellbogen rückwärts an und hielt sich vollkommen ruhig, wobei sie eine Erleichterung wahrnahm. Es besteht ausgesprochene Hypertrophie und Dilatation des rechten Ventrikels (voussure).

Von den in ihrer Intensität wechselnden Geräuschen prävaliert weitaus das diastolische, das sich ganz besonders schon nach geringfügigen Körperbewegungen, namentlich bei raschem Rumpfvorbeugen am linken Sternalrand, in seiner größten Stärke entsprechend dem Ansatz des linken 3. Rippenknorpels und noch etwas nach aufwärts zu als rauhes, schabend kratzendes, lautes, meist etwas geteiltes (wachtelschlagartiges) Geräusch kenntlich macht.

Ein leiseres, nicht streng an die Herzphasen gebundenes, auch in die Systole fallendes, schwirrend rieselndes Geräusch pflanzt sich noch etwas weiter nach oben links zu fort.

Der Dämpfungsstreifen (s. o.) etwas ausgesprochener und breiter. Bei kurzem, starken Perkussionsschlag Empfindlichkeit daselbst. Leichte Albuminurie.

Im Sommer 1902 besserte sich das Allgemeinbefinden und die subjektiven Beschwerden. Der Blutdruck stieg auf 120—130 (Tonometer von Gärtner).

Im Verlaufe des Jahres 1903 mehrmalige ambulatorische Vorstellung, klinische Behandlung zu wiederholten Malen.

U. A. vom 16.—29. Juli 1903. Die Kranke ist wieder sehr stark zyanotisch. Wiederholte Schmerzattacken vom Brustbein nach links gegen Parasternal- und Mammillarlinie zu, einhergehend mit Schwäche, Übelkeiten und Blaßwerden, bald darauf wieder ganz auffällige Zyanose.

Arhythmie, Dyspnöe und Stauungssymptome stärker. Jugularvenen strotzend gefüllt. Andeutung von Bulbuspuls, hier und da auch Spur von venösem Leberpuls. Symptome vorübergehender relativer Trikuspidalinsuffizienz (?) angedeutet. Blutdruck vermindert.

1904: Aufnahme Januar und Juli. 5. I.—26. I.: Auf Digitalis Ansteigen der Diurese (von 400 auf 1400 ccm), um bald darauf wieder abzusinken. Zunahme der Ödeme an den unteren Extremitäten, Bildung von Hydrops ascites. Myodegeneratio cordis hypertrophicae. Wiederholt Koffein und Digitalis.

Ebenso während der 2. Aufnahme vom 30. VI.—10. VII. 1904.

Am 5. VII. Dränage ($1^1/_2$ l).

Exitus am 10. Juli 1904.

Sektion 12. Juli 1904 (Prot. Nr. 6739/155): Hochgradige Stenose und Insuffizienz der Valvula mitralis mit bedeutender Hypertrophie und Dilatation besonders des linken Vorhofes und des rechten Herzens. Hydroperikard.

Lobulär pneumon. Herde in den hinteren Anteilen des linken Oberlappens, neben Ödem und Induration der Lunge. Hydrothorax. Fettige Degeneration der Nieren, neben trüber Schwellung und alten nephrit. Absumptionen. In der linken Niere an der Vorderfläche ein vernarbter Infarkt. Starke groblappige Cirrhose und Stauung in der Leber. Chron. Gastroenteritis.

Hydrops ascites 5—6 l.

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Die Valv. mitralis in ihren Zipfeln stark verdickt, dieselben miteinander verwachsen, so daß sie nur noch für einen Finger passierbar; die Chordae tendineae fehlen fast vollständig. Der linke Ventrikel klein, zusammengezogen, wie atrophisch. Der linke Vorhof dagegen weit, ausgedehnt, besonders auch das linke Herzohr. Aorta von gewöhnlichem Umfang. Intima glatt und eben.

Pulmonalarterie in ihrem Anfangsteil, oberhalb des Klappenringes, gleichmäßig erweitert, in ihrer Wand beträchtlich verdickt, zeigt eine fast allgemeine Verdickung, Verhärtung und Verkalkung der Intima bedeutenden Grades.

Die arteriosklerotischen Ein- und Auflagerungen und Verkalkungen reichen bis in die Hauptäste des Gefäßes.

Im folgenden sei eine übersichtliche Darstellung der Literatur gebracht und zwar nur solcher Beobachtungen, bei denen auch klinische Befunde vorliegen und deshalb in den Rahmen dieser Abhandlung passen.

Kompliziertere Bildungsanomalien, gelegentliche zufällige Befunde oder solche von rein pathologisch-anatomischem und histologischem Interesse konnten hier nicht berücksichtigt werden.

Für die Einbeziehung der Literatur bewogen mich folgende Erwägungen: Fürs erste existiert eine Darlegung vom klinischen Standpunkt aus überhaupt noch nicht, zweitens fand eine Reihe solcher Mitteilungen bisher keinerlei Beachtung und Verwertung, drittens ist ein Großteil der hier einschlägigen Literatur sehr schwer zugänglich und zerstreut. Per parenthesin möchte ich bemerken, daß ich fast sämtliche der folgenden Arbeiten im Original nachgesehen.

Aus der ältesten Literatur existiert ein Beispiel von Blausucht infolge von Verknöcherung der Lungenarterienhaut bei Blancard, den auch Sénac anführt (Traité de la structure du cœur, de son action et de ses maladies. Paris. 2 t. 1749. [1774 éd. Portal.]).

In der heute noch lesenswerten Abhandlung von Kreysig (Die Krankheiten des Herzens. Berlin 1816. Bd. 3, S. 119) findet sich nachstehende Krankengeschichte:

(21. Fall.) Ein junger Mensch, 22 Jahre alt, von kleiner und magerer Statur, in seinem 18. Jahre noch ganz bartlos, hatte eine blaurote Gesichtsfarbe und stark aufgetriebene Venen der Haut. Jede Bewegung, vorzüglich in freier Luft, trieb ihm das Blut nach dem Kopfe, die Backen wurden dann blau, und bei starker Bewegung bluteten Lippen und Mund.

Das Atmen war immer beengt und durch Husten unterbrochen, besonders bei Körperbewegungen. Herzklopfen quälte ihn oft. Seit einiger Zeit litt er an heftigen Rheumatismen.

Ein Quacksalber behandelte ihn mit schweißtreibenden und erhitzenden Mitteln, worauf Fieber, große Beklommenheit des Atems, Heiserkeit und heftiges Herzklopfen eintraten. Die Zufälle verschlimmerten sich immer mehr, der Kranke verlor das Bewußtsein und starb.

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Bei der Leichenöffnung fand man die Brust an der linken Seite erhabener und aufgetriebener. Das Herz zeigte eine größere Breite als gewöhnlich, die Spitze war stumpfer, sonst erschien es wohlgebildet, ohne Veränderung seiner Substanz und ohne Klappenfehler.

Die Lungenpulsader war da, wo sie sich in ihre beiden großen Äste teilt, in ihrer Textur sehr verändert. Der rechte Ast war an einer Stelle so weich, daß er bei der Berührung sogleich zerriß. Daneben waren seine Häute verdickt und das Lumen sehr verkleinert. Die verdickte Stelle erschien äußerlich brandig. Mehrere Zweige waren fast ganz verschlossen, zum Teil faltig und eingeschrumpft und konnten kein Blut mehr geführt haben. Die Venen des Körpers waren sehr erweitert. (Leber groß.)

Nach Kreysig ist diese Verengung und Unwegsamkeit der Lungenarterie nicht angeboren, sondern er vermutet deren Entstehung in den ersten Kinderjahren. Durch die Behandlung mit reizenden Mitteln sei der Zustand verschlimmert und eine schleichende Entzündung der kranken Teile hervorgebracht worden, deren Folgen der Kranke erlag.

Ebenda wird (II. T. 1. Abschn. S. 430) eine alte Beobachtung von Kinglake (London med. Journ. 1789, vol. X, pars IV. p. 341 und Sammlung f. prakt. Ärzte 13. Bd., 3. St., S. 385) zitiert.

Bei dieser handelte es sich um ein zartes Frauenzimmer von 23 Jahren, bei welchem auf äußere Veranlassung Schmerzen im Herzen entstanden, schreckliches Klopfen und Gefühl von Erstickung sich beigesellten, welche Zufälle mit Ohnmachten abwechselten.

Bei der Sektion fand man innerhalb des rechten Herzohrs und der rechten Kammer einige polypöse Konkretionen von verschiedener Größe anhängen; die größten hatten den Umfang einer welschen Nuß, sie nahmen mehr als die Hälfte von den Höhlen des rechten Herzens ein. In den Häuten des Stammes der Lungenschlagader, einen halben Zoll über den Klappen derselben, entdeckte man eine harte steinigte Substanz, ungefähr ein halbes Quentchen schwer, welche die Höhle der Schlagader sehr verengerte. Lungen ohne Geschwüre. Es heißt dann: „Wer sieht nicht, daß die Zufälle dieser Kranken Folge der Verengerung der Lungenarterie waren, wie bei der Blausucht und bei Klappenfehlern."

Gelegentlich der ausführlichen und für seine Zeit geradezu epochemachenden Besprechungen über Angina pectoris kommt Kreysig (l. c. II. Th., 1. Abschn., S. 525) bei dem Abschnitte, der über Verwechslung der Angina pectoris mit Fehlern der Klappen und Verengerungen der großen Arterienstämme nahe am Herzen handelt, auf einen hier einschlägigen Befund von Stöller zu sprechen.

Stöller (Hufelands Journal der prakt. Heilk. 17. Bd., 2. Stück) führt einen komplizierten Krankheitsfall als Angina pectoris an. Anfälle von Angst und Schmerzen auf der Brust, die in die Arme ausstrahlten bei einem 56jähr. Manne, der wiederholt Pneumonien überstanden hatte und bei dem ein Lungengeschwür aufging.

Diese Zufälle legten sich nach Aderlässen. Nach 13 Jahren kehrten sie wieder, bis er einem erlag (Januar 1792). Bei der Sektion fand sich nach Stöllers eignem Bericht: die Lungen überall adhärent. „Das Herz von ungewöhnlicher Größe, aber schlaff und blutleer, sonderlich in der linken Höhle. Die halbmondförmigen Klappen

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der Lungenpulsader im Zustand der Verknöcherung, die großen Gefäße an ihren Anfängen steif und knorpelicht und voll von dickem schwarzen Blut. Die Kranzarterien und Venen wurden leider nicht besonders untersucht."

Huguiers (Mém. sur le diagnost. Arch. gén. de méd. févr. 1834) Beobachtung betrifft

eine 73jährige Frau. Seit 5—6 Jahren krank. Brustbeschwerden und Bronchitis. Herzklopfen. Sehr unregelmäßige Herztätigkeit. Schmerzen in der Herzgegend. Nekropsie: an der Trikuspidalklappe mehrere kleine verknöcherte Flecken. Die Mündung und eine Klappe der Pulmonalarterie zeigen verknöcherte Verwachsung. Die 3 Klappen etwas verdickt.

Die Arteria pulmonalis von ihrem Ursprung bis zur Bifurkation, die Aorta von der Konkavität des Bogens bis zum 2. Lendenwirbel fast vollständig verknöchert.

Die nicht verknöcherten Partien sind verdickt und viel leichter brüchig. Die ganzen verknöcherten Plaques finden sich in Intima und Media. An einigen Stellen die innere Haut destruiert, an anderen ulzeriert. Manche Ossificationen sind granuliert, über das Innere des Gefäßes vorspringend.

Bei Tiedemann (Von der Verengung und Schließung der Pulsadern in Krankheiten. Heidelberg, Leipzig 1843) findet sich eine Notiz über einen Fall der Literatur, der zweifellos als Thromboarteriitis pulmonalis zu gelten hat (S. 140) (obwohl derselbe nicht direkt hierher gehört bietet er doch einiges Interesse).

„Die Lungenpulsader kommt ebenfalls bisweilen entzündet vor und ihre innere Haut schwitzt gerinnbare Lymphe aus, wie aus folgender Beobachtung (L'Espérance 1835, 5. mart. no. 25) erhellt. Eine Wäscherin wurde nach einer heftigen Verkältung von Magenschmerz, großer Beklemmung des Atems und heftigem Fieber befallen. Plötzlich wurde ihr Antlitz aufgetrieben und blaurot. Die Augen traten vor und die Kranke fiel besinnungslos nieder. — Bei der Sektion fand man das Herz von großem Umfang und mit schwarzem dickflüssigem Blute gefüllt. Die rechte Kammer enthielt ein großes schwärzliches Blutkoagulum, welches ihren Wandungen fest anhing und sich in die Lungenpulsader und ihren größeren Verzweigungen fortsetzte. Die innere Haut dieser Arterie war rauh, flockig und zeigte hier und da kleine weißliche Flecken, an denen das Blutgerinnsel fest ansaß. An mehreren Stellen konnte man membranöse Schichten ablösen, welche Faserstoff oder plastischer Lymphe glichen. Die innere Haut ließ sich leicht stückweise ablösen."

Adlers (Nonnullae de morbis arteriae pulmonalis. Dissert. Vratislaviae 1855):

40jähriger Kranker zeigte Schmerzen in der Brust, Husten und Engatmigkeit. Hämoptöe. Anämie. Plötzlicher Exitus.

Dieser Fall von Pulmonalsklerose mit Thrombose ist dadurch ausgezeichnet, daß keine Klappenaffektionen und keine Sklerose der übrigens engeren Aorta vorhanden waren.

Bei Klinger (Beobachtungen über die Verstopfung der Lungenarterie durch Blutgerinnsel. [Dieselben wurden während der früheren Assistentenzeit des Verf. an der Würzburger med. Klinik gesammelt.] Arch. f. physiol. Heilkunde 1855, XIV. Jahrg., S. 362) fand ich einen hierher gehörigen Kasus, der ebensowenig wie die vorausgehenden Fälle in der Literatur berücksichtigt wurde.

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Aus der Krankengeschichte sei nur das Relevante gebracht (S. 371).

Fall 4. Kunigunde N., 40 Jahre alt. 21. XI. 1851 Aufnahme im Juliushospital. Diagnose: Atheroma arteriae pulmonalis[1]).

Pat. ist kräftig gebaut, will von Jugend auf brustleidend gewesen sein und oft an Blutspeien, Husten, Dyspnöe gelitten haben. Vor 7 Jahren soll dieses Übel sich so gesteigert haben, daß sie über 1 Jahr bettlägerig war, worauf sie sich einigermaßen erholte. Vor 3 Jahren gesellte sich zu ihren asthmatischen Zufällen Ödem der Füße, seit 6 Wochen war ihr Zustand sehr quälend und die hydropischen Erscheinungen nahmen mehr überhand.

Stat. praes.: Pat. von starkem Körperbau ist etwas abgemagert, Hautfarbe livid, Lippen blaurot, gedunsen, ebenso Wangen, Petechien auf Brust, Bauch, Extremitäten und Rücken: Intumeszenz der Halsvenen, leichte Undulation derselben ohne Geräusch. Hauttemperatur kühl. Puls nicht beschleunigt, schwach, Orthopnöe, pfeifende Respiration, 40mal in der Minute. Mäßiger Aszites und Ödem der Füße. Urin sehr spärlich, enthält keine fremden Bestandteile. Scrobiculum cordis gespannt, hervorgetrieben, schmerzhaft, besonders beim Druck, ebenso Schmerzen im rechten Hypochondrium (seit 6 Wochen).

Herzchok schwach, an gewöhnlicher Stelle fühlbar. Herzhypertrophie. Herztöne normal, schwach. Zweiter Pulmonalton akzentuiert. Verschiedene Diuretika und Drastika ohne Erfolg. Digestion gestört. Beständiges Würgen und Erbrechen. Letzteres durch Laudanum gestillt.

Anasarka tritt hinzu, die Schmerzen im rechten Hypochondrium nehmen zu, das Ödem der Füße ist enorm, hiezu kommt Ödem der Genitalien. Höchste Dyspnöe, besonders paroxysmenweise, Husten gering. Expektoration fehlt. Livor des Gesichtes ist bedeutend, die Conjunctiva bulbi wird ikterisch.

26. Dezember, deplorabler Zustand. Pat. ist am ganzen Körper geschwollen, Stickparoxysmen, verbunden mit Würgen und Erbrechen. Im Urin sind Spuren von Gallenfarbstoff nachweisbar. 28. Dezember letaler Ausgang.

Sektion: Allgemeiner Hydrops.

Perikardium stark ausgedehnt, hat die Lungen ganz zurückgedrängt, enthält ca. 18 Unzen einer hellen ikterischen Flüssigkeit, mehrere Sehnenflecke nebst fadenartigen Adhäsionen zwischen den beiden Blättern, Herz sehr ausgedehnt und groß, besonders der rechte Ventrikel stark dilatiert und hypertrophisch, im rechten Herzohr ein großes, altes Gerinnsel, die Ventrikel und Vorhöfe beiderseits gefüllt mit cruorreichen Gerinnungen. Klappen normal. Lungen an den Spitzen verwachsen, im linken Pleurasack etwas seröses Exsudat, im rechten ein großes, abgesacktes seröses Exsudat zwischen Wirbelsäule, Zwerchfell und Lunge, die in ihrem unteren Lappen luftleer komprimiert ist; die übrigen Partien der rechten wie linken Lunge sind lufthaltig, etwas hyperämisch und ödematös. Bronchien stark gerötet, besonders links, wo an einzelnen Stellen die Hyperämie bis zu Extravasation in der Schleimhaut gediehen ist.

Die Innenfläche der Arteria pulmonalis, besonders in den Verzweigungen ersten und zweiten Grades sehr dilatiert, fettig atheromatös degeneriert (die Aorta zeigt auffallend wenig atheromatöse Stellen), kleinere Pulmonaläste (von der Größe eines Gänsefederkieles) sind an mehreren Stellen durch Gerinnsel verstopft.

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1) Ob diese Diagnose „intra vitam“ gestellt wurde, ist nicht ersichtlich. Auch findet sich keinerlei epikritische Bemerkung oder Motivierung für sie.

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Lancereaux, Note relative a quelques faits d'obstruction des vaisseaux veineux et de l'artère pulmonaire; caractères non douteux de caillots emboliques; produits organisés (néo-membranes) au sein de l'artère pulmonaire et des veines, Comptes rendus de la Soc. de biologie 1861, XIII. ann. t. III, p. 119

berichtet 1. (S. 124) über einen 70jährigen Mann, Schuster, Alkoholiker mit Dyspnöe, gedunsenem blaurotem Gesicht. Diagnose einer Dilatation des rechten Herzens, Lungenödem. Gestorben 19. Mai 1861. Bei der Nekropsie: Herzvergrößerung, Erweiterung und Verfettung des rechten Ventrik. Thrombot. Verstopfung der Teilung des rechten Astes der Pulmonalis. Befund an der Art. pulmon.:

„Dilatation légère de l'artère pulmonaire à la surface de laquelle apparaissent de nombreuses plaques jaunâtres un peu dures et saillantes; assez fréquentes dans les grandes divisions, ces plaques deviennent beaucoup plus rares dans les petites." Hypertrophie und Dilatation des linken Herzens. Alteration der Koronararterien und der Arterien der unteren Extremitäten.

2. 57jährigen Mann, Juwelier, mit einem Herzleiden, speziell Mitralstenose. Ödem der unteren Extremitäten, Rasseln auf der Lunge. Exzessive Dyspnöe. Überstand Rheumatismus und Pneumonie. Gestorben 20. Juni 1861.

Autopsie: Rechte Lunge angewachsen.

Ausgedehnte Thrombose der Pulmonalis.

Befund an der Lungenarterie: „Cette artère est dilatée, sa paroi épaissie offre à sa surface interne, rougeâtre, injectée sur quelques points, d'abondantes plaques jaunâtres et graisseuses."

Das linke Ostium atrioventriculare verengt und kaum für den kleinen Finger durchgängig, das korrespondierende Herzohr hypertrophiert. In der Höhe der Botallischen Öffnung trifft man eine Membran von 1—2 cm Länge, am Gewebe adhärent, aus Bindegewebe bestehend.

Verf. glaubt, daß das organisierte Produkt (Endarteriitis) der fibrinösen Thrombenbildung vorausgegangen ist, daß es die Ursache und nicht die Folge war.

Den mikroskopischen Befund erbringt er in einer weiteren Mitteilung l. c. p. 162.

Weitere Berichte gibt

Martineau, Dégenérescence athéromateuse des artères pulmonaires droite et gauche; rétrécissement et insuffisance de l'orifice mitral; apoplexie pulmonaire; caillots dans les dernières ramifications des artères pulmonaires. Comptes rend. de la Soc. de biologie 1861, XIII. ann., p. 163.

39jährige Näherin. Herzpalpitationen, Oppression, unregelmäßiger Herzschlag. Insuffizienz der Mitralis, Hämoptöe (hämorrh. Infarkte).

Sektion: Hypertrophie der rechten Ventrikelwand. Aortenklappen normal. Einige atheromatöse Plaques an der Intima der Aorta, sonst aber an keinem andern Teil des arteriellen Systems. Stamm und beide Äste der Arteria pulmonalis ohne pathol. Veränderung.

Atheromatöse, fettige Entartung der Zweige der Pulmonalarterie, hochgradig in den kleinsten Ästen, besonders der zu den hämorrhagischen Herden führenden, in denselben fibrinöse, festhaftende, das ganze Kaliber verstopfende Pfröpfe.

Für das Entstehen dieser macht Verf. die fettige und atheromatöse Entartung der Pulmonalarterienäste und die Herzaffektion verantwortlich.

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Derselbe, Caillots dans les artères pulmonaires droite et gauche; dégénérescence athéromateuse de ces artères; apoplexie pulmonaire; rétrécissement et insuffisance de l'orifice auriculo-ventriculaire gauche. Comptes rendus des séanc. de la Soc. de biologie Paris 1861, XIII. Ann., p. 196 (Klinik Tardieu).

54jährige Taglöhnerin. Früher rheumatische Schmerzen. Seit 2 Jahren Herzklopfen. 5 Tage vor Spitalseintritt Ödem der Beine. Asthmatische Anfälle. Status: Lungenemphysem und Bronchitis. An der Herzspitze systol. Geräusch. Puls klein, weich, sehr frequent.

Später hämorrhagische Sputa, zunehmende Dyspnöe, Herzschwäche.

Autopsie: Die Äste der Lungenarterie sind der Sitz einer bemerkenswerten Veränderung, die sich jedoch in beiden nicht in gleicher Stärke zeigt.

In der linken Lunge, wo man weniger zahlreiche und weniger voluminöse hämorrhagische Herde trifft als in der rechten, sieht man in dem zum Unterlappen ziehenden Lungenarterienast da und dort an der Innenfläche des Gefäßes gelbliche sehr harte atheromatöse Stellen. Beim Aufschneiden begegnet man denselben hauptsächlich nur in solchen Zweigen, die zu den hämorrhagischen Stellen führen. Zugleich ist in einer gewissen Entfernung das Kaliber der Arteriolen völlig obliteriert durch einen schwärzlichen Pfropf, der an der Innenfläche des Gefäßes festhaftet. Die gleiche Veränderung trifft man in den andern zu den genannten Herden ziehenden Arteriolen.

In der rechten Lunge läßt sich die gleiche Läsion der Pulmonalarterie konstatieren. Nur ist hier der Prozeß in noch viel höherem Grade vorhanden.

Der zum Unterlappen ziehende Ast ist durch einen harten, an der Peripherie gelblich gefärbten Pfropf vollständig verlegt. Derselbe ist im Zentrum mehr schwärzlich und adhäriert an der Innenfläche.

Die Thrombenbildung setzt sich fort in die Verzweigungen der Arterien, vorzüglich der zu den hämorrhagischen Infarzierungen führenden.

Die atheromatöse Veränderung ist viel ausgeprägter als in der linken Arterie Die gelblichen Plaques sind sehr hart, viel ausgebreiteter und umgreifen das ganze Kaliber der Gefäße. Diese Veränderung trifft man jedoch nicht in den zu gesunden Lungenpartien führenden Zweigen des Gefäßes.

Das Mitralostium war der Sitz sehr schwerer endokarditischer Veränderungen. Die Aortenklappen intakt. An der Aorta mäßige Atheromatose.

Hinsichtlich seiner Beobachtung gelangt Martineau zu folgender Annahme: Die primäre Ursache der Lungenhämorrhagien wäre in der Veränderung der Lungenarterie gelegen, hierzu gesellte sich die hochgradige organische Herzläsion, durch Zusammenwirken dieser beiden Ursachen kam es im Verlaufe der Blutstauung in diesen gewissen Arterien zur Bildung der Thromben in den Pulmonalarterien.

J. Klob (Bericht über die Ergebnisse der in der pathol. anatom. Anstalt des k. k. Krankenhauses Rudolph-Stiftung vorgenommenen Obduktionen. Bericht der k. k. Krankenanstalt Rudolph-Stiftung in Wien vom Jahre 1865. Wien 1866) liefert die Beschreibung eines primären Falles:

S. 185. Endarteriitis pulmonalis deformans.

„Die Endarteriitis deformans in der Pulmonalarterie wird besonders

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dann das Interesse des Anatomen in Anspruch nehmen, wenn sie die gleichartige Erkrankung in der Aorta übertrifft, oder aber gar ohne diese vorkommt, wie dieses in einem Falle beobachtet wurde.

Soweit reicht nun vielleicht das Interesse des Anatomen allein, wenn nun aber infolge dieser Erkrankung so erhebliche Veränderungen in der Kapazität der Lungenarterienbahn entstehen, daß allgemeine Zirkulationsstörungen bis zum allgemeinen Hydrops auftreten, dann muß auch der praktische Arzt Notiz von einem Falle nehmen, dessen anatomische Veränderungen sich nicht nur in einen rein logischen Kausalnexus bringen lassen, welcher auch einigen Anhaltspunkt zu einer Diagnostik derartiger Erkrankungen liefern dürfte."

59jähriger Pfründner. Allgem. Hydrops und Zyanose. Anämie und leichtes Ödem der Lungen, exzentrische Hypertrophie des rechten Ventrikels mit starker Dilatation des rechten Vorhofes. Pulmonalarterie im Stamme weit, die Innenfläche derselben sehr ungleichförmig, stellenweise wie parallel runzelig, stellenweise in Plaques der unregelmäßigsten Form und Größe prominierend.

In ihren Verästlungen in der Lunge ebenfalls Verdickung der Intima, welche mitunter so bedeutend ist, daß ein querdurchschnittener Ast das Lumen stark exzentrisch erkennen läßt. Etwa von den Ästen 3. Ordnung an läßt sich im allgemeinen behaupten, daß das Lumen der Arterien auffällig enger wurde, so daß dasselbe am Durchschnitte von Ästen 5.—6. Ordnung kaum nadelstichgroß erschien, wobei das starre Gefäß eine Wanddicke von nahezu 1 mm (also 2 mm Durchm.) hatte, so daß diese Äste als feste weißgelbe Stränge die Lungensubstanz durchsetzten.

Es wurde also durch die Endarteriitis deformans eine Verengerung der Lungenarterienbahn in bedeutendem Grade hervorgerufen, aus welcher sich die übrigen anatomischen Veränderungen leicht erklären lassen. Die Lunge war entsprechend blutarm, doch hatte natürlicherweise ihre Ernährung nicht gelitten. Die Bronchialblennorrhöe ist vielleicht auch daraus zu erklären, daß infolge des abnehmenden Blutdruckes in den Kapillaren zwischen Lungenarterie und Venen eine Hyperämie des nutritiven Gefäßapparates der Lungen zustande kam.

In ähnlicher Weise wie die französischen pflegen die englischen Autoren bereits im umfangreichen Titel ihrer Mitteilung den Befund niederzulegen.

Conway Evans, Mode of causation of arterial atheroma; illustrated by a case of atheromatous disease of the pulmonary artery, in association with extreme contraction (congenital) of the left auriculoventricular orifice and hypertrophy of the right ventricle. Transact. of the pathol. Soc. 1866, vol. XVII, p. 90.

14jähriger Knabe. Hatte vor 3 oder 4 Jahren Husten und Beklemmung auf der Brust, speziell während des Winters. Vor Spitalsaufnahme beginnende Ödeme im Gesicht und den Füßen; konnte jedoch noch arbeiten. Niemals Polyarthritis oder eine andere schwerere Krankheit. — Gerötete Wangen, ängstlicher Ausdruck, schnelle Atmung, Orthopnöe. Sternum ungewöhnlich vorgewölbt, respirat. Thoraxverschiebbarkeit vermindert. Vesikul. Atmen mit lauten Rasselgeräuschen. Deut-

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lich systolisches Geräusch an der Herzspitze. Puls 80, regelmäßig. Ödeme und Aszites. Kein Albumen.

Diagnose: Mitralaffektion, Bronchitis und Emphysem. 3 Wochen nach der Aufnahme Tod. Sektion: Lungenkongestion und Emphysem, besonders des rechten Randes. Herzhypertrophie, speziell Dilatation und Hypertrophie des rechten Ventrikels, derselbe verdickt. Linker Vorhof und Aurikel erweitert. Rechtes Atrioventrikularostium stark erweitert.

Pulmonalarterie erweitert, ihre Auskleidung bis zur Teilung stark verdickt, Wandung dicker als die der Aorta. Die Innenfläche besetzt mit zahlreichen gelblichen Atheromplaques sowohl im Stamm als in den Verteilungen. Aorta von gewöhnlicher Dicke der Wand, aber verengert, kein Atherom in Aorta oder größeren Gefäßen. Alle drei Aortenklappen verkürzt und verdickt (kongenital?). Mitralostium verengt (trichterartig).

Ein kombiniertes Vitium betrifft nachstehender Kasus:

Deroye (Bullet. de la Soc. anatom. 1870, p. 166. Hôpital de la Pitié, serv. de M. le prof. Lasègue).

44jährige Näherin. Eintr. 11. Febr. 1870.

Nie rheumatische Beschwerden. Immer gesund. Seit vorigem Dezember Beine etwas angeschwollen und zwar gegen Abend zumeist in der Maleolargegend. Seit 2 Jahren hustet sie öfter und bekommt leicht Schnupfen. Der Atem war kurz, bei geringen Anstrengungen Herzklopfen. Vor 4 Tagen ohne besonderen Grund Hämoptöe, die noch andauert, dabei intensive Dyspnöe und rasche Zunahme der Anschwellungen. Status: Lupus im Gesicht, Respiration erschwert. Sehr starke Anasarka.

Verbreiterte Herzpulsation. Bei der Auskultation zwei Geräusche. Das eine, systolische, sehr deutlich an der Herzspitze, das andre, diastolische, an der Basis, aber weniger ausgeprägt, reichliches Rasseln. Jugularvenen ausgedehnt, jedoch kein Venenpuls bemerkbar. Links pleurit. Reiben. Reichliches Rasseln über den Lungen. L. v. o. kavernöses Rasseln. Exitus 24.

Sektion: Rechte Lunge angewachsen. Hepatisationen und Erweichungen. Im rechten Oberlappen bronchiektat. Kaverne. Embolien, Infarktbildungen, pneumonische Infiltrationen. Linke Lunge innig mit dem Perikard verwachsen. Concretio pericardii. Linker Oberlappen emphysematös. In den hinteren Partien Embolie und Blutungsherde.

Herz enorm, Aorteninsuffizienz, Stenose und Insuffizienz des Mitralostiums. Die Oberfläche der Aorta zeigt nirgends im Verlaufe Atherombildung, bloß zwei Semilunarklappen sind verdickt und atheromatös.

Die Pulmonalarterie dagegen zeigt an ihrer Innenfläche gelbliche, nicht ulzerierte atheromatöse Erhabenheiten. Obwohl die Klappen der Pulmonalarterie weich und zart sind, trifft man am freien Rand einer derselben einen kleinen, harten, hervorragenden Tumor von Erbsengröße. An einer andern Klappe sind kleine gelbliche Granulationen von gleicher Natur vorhanden. Vergrößerte harte Mußkatnußleber.

Einen fast analogen Fall weiß Cadet de Gassicourt (Bull. de la Soc. anat. 1872, p. 248. Hôpital Saint-Antoine. [VI. Beob. bei Sauné])[1] zu berichten.

1) Path.-anat. Bericht: Rosapelly, Etat scléro-athéromateux de l'artère pulmonaire droit; altérations des valvules pulmonaires, tricuspide; caillot autochtone

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37jähriger Tischler (April 1873) gibt an, Hämoptöe gehabt zu haben, vor einigen Monaten rechtsseitige Pleuritis. Rechtss. pleurit. Erguß zu konstatieren; über der oberen Hälfte derselben Seite ergibt die Auskultation Zeichen einer sehr vorgeschrittenen Phthise. Starke Dyspnöe, bis zur Orthopnöe.

Das abgemagerte Gesicht zyanotisch. Puls klein, unregelmäßig, heftige Herzaktion, Halsvenen turgescent. Blasendes Geräusch an der Herzspitze. Die Gegenwart der Mitralläsion zusammen mit der Lungenaffektion erklärt zur Genüge die Schwere des Allgemeinzustandes. Exitus 4. Mai.

Sektion: Bei Eröffnung des Thorax finden sich 2 l Flüssigkeit in der rechten Pleurahöhle.

Das Herz ist voluminös, links eher hypertrophisch, rechts dilatiert. Stenose und Insuffizienz des Mitralostiums und der Aorta. Im Bogen der Aorta einige kleine atheromatöse Flecken. Ebenso wenige erhabene atheromatöse Plaques in dem linken Herzohr.

Im rechten Herzen trifft man keine ähnlichen Veränderungen, als am Ursprung der Lungenarterie, wo zahlreiche atheromatöse Plaques vorhanden sind. In der rechten Lunge Stauung, im Mittellappen hämorrhagischer Herd. Der rechte Lungenarterienast erweitert, verdickt, die innere Oberfläche uneben infolge harter atheromatöser Plaques von hornartigem Aussehen.

Thrombotischer Pfropf in dem Pulmonalarterienast beider hämorrhagischer Herde.

Die Intima der Pulmonalarterie ist in ihrer ganzen Ausdehnung verdickt, sie zeigt eine große Menge von Plaques und Unebenheiten, deren Struktur analog ist denen, welche sich in der Aorta entwickeln.

Sie bestehen aus Lamellen von homogener Substanz, in deren Interstitien sich zellige Elemente finden mit Fortsätzen. In der unmittelbaren Nachbarschaft der Media sind diese zelligen Elemente ersetzt durch vereinzelte oder gehäufte fettige Granulationen in Form länglicher Haufen. Andre Plaques sind atheromatös in ihrer ganzen Dichte. Der Thrombus im rechten Zweige der Pulmonalarterie hängt an der Wand im Bereiche eines sklerösen Plaque und zeigt an jener Stelle in einer Ausdehnung von 2—3 mm den Anfang einer Organisation. Er enthält an dieser Stelle Elemente von bindegewebiger Beschaffenheit ähnlich der Intima.

Yeos (Disease of the pulmonary arteries; hypertrophy of the right ventricle. Dublin Qu. I. M. Sc. 1873, LV. May, p. 480) Bericht besagt folgendes:

Eine Frau, welche bereits schon vor längerer Zeit an Rheumatismusanfällen mit Endokarditis litt, zeigte ausgesprochene Erscheinungen von Mitralstenose. Es bestanden öfters Anfälle von Dyspnöe, Zyanose, Husten, Hämoptöe und Herzklopfen.

An der Spitze des Herzens hört man ein lautes systolisches, an der Basis ein leises, doppeltes Geräusch.

Zuletzt Ödeme und Aszites.

Obduktion: Hypertrophie des rechten Ventrikels; der rechte war in seiner Wand dicker als der linke, die Semilunarklappe intakt, die Trikuspidalklappe leicht

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dans le tronc de cette artère; infarctus pulmonaires. Mort, autopsie. Bull. de la Soc. anat. Paris 1872, XLVII, 2. s., t. XVII, p. 248.

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verdickt. Die Mitralklappe ist verdickt, verhärtet, ihr Orificium faßt nur die Spitze des kleinen Fingers. Die Aortenklappen sind ebenfalls erkrankt, die Aorta ist gesund, der linke Ventrikel etwas hypertrophiert. Lungen emphysematös, ihr vorderer Rand infiltriert; mehrere hämorrhagische Infarkte.

Die Pulmonalarterie ist von den Semilunarklappen angefangen bis in die feinsten Verzweigungen erkrankt. Sie ist unregelmäßig erweitert und hat ihre Elastizität verloren. Ihre Intima ist durchgehends von harten, vorherrschend gelben Flecken besetzt, von denen einige an ihrer Oberfläche rauh sind und das gewöhnliche Aussehen atheromatöser Ulzerationen haben.

Die Erkrankung scheint ihren stärksten Grad in den kleineren Ästen erreicht zu haben, einen geringeren jedoch in der unmittelbaren Nähe des Herzens. Die zu den Lungeninfarkten führenden Gefäße sind durch Fibrinpfröpfe obliteriert, welche fest an der Gefäßwand haften.

Wegen des klinischen Symptoms der Hämoptöe verdient nachfolgende Notiz mit anschließender Diskussion Beachtung.

Vignier, Bulletins de la Société anatomique, Paris 1873, p. 526, berichtet über den Herzbefund bei einem mit sehr starkem Herzklopfen und mit einem alten Herzleiden behafteten, plötzlich verstorbenen Kranken.

Man fand bei der Autopsie eine hochgradige Stenose der Mitralis, atheromatöse Plaques in der Pulmonararterie und infarktähnliche Herde in der Lunge. In der Nachbarschaft dieser Herde waren die Arterien erkrankt. Lancereaux, welcher die Präparate sah, erklärt diese Erkrankung als Arteriitis. Es existieren in ähnlicher Weise Infarkte in Nieren und Milz. Demnach der große und kleine Kreislauf in gleicher Weise erkrankt ist. Vignier vertritt den Standpunkt, daß das rechte Herz intakt und die Lungenarterie erkrankt, während das schwer erkrankte linke Herz das Blut in eine nahezu gesunde Aorta befördert.

Charcot glaubt, daß hier zwei Störungen zusammentreffen, die eine in den Lungenarterien, die andre im Parenchym der Lunge selbst, und daß es unmöglich ist, zu sagen, welche von diesen beiden vorausgegangen, ob die Arteriitis die Ursache oder die Folge der (hämorrhagischen) Infarktherde war.

Voisin, Bulletins de la Soc. anatomique 1874, p. 517. (Hôpital temporaire de la rue de Sèvres. Serv. de M. Damaschino.) 3. Beob. bei Sauné.

45jähriger Kunsttischler, eingetreten 2. April 1874.

Während des Krieges 1870 bekam er ein Erkältungsfieber, das den Winter über dauerte und sich in den folgenden Jahren während der kalten Jahreszeit wiederholte.

1873 Hämoptöe, angeblich 1 l Blut ausgehustet. Seitdem schwanden die Kräfte und er mußte die Arbeit aufgeben. Er bekam Atembeschwerden, Herzklopfen, Gesicht wurde zyanotisch. Nachtschweiße. 5 Tage vor Spitalseintritt Ödem der Beine, bis zu den Knien. 15. April auffällige Zyanose, Lippen blau. Jugularvenen ausgedehnt, Venenpuls. Herzstoß nicht fühlbar, leichte Voussure. Dämpfung vergrößert, im Längsdurchmesser 12—13 cm messend, bis zur Mitte des Sternums reichend. Verlängerte Systole an der Spitze. Systol. Geräusch am Angul. Ludovici, am Rand des Sternums das Geräusch sehr deutlich, manchmal verdoppelt. An der Basis der 2. Ton etwas klingend. Puls klein, unregelmäßig. Arterien atheromatös.

Über der Lunge Rasselgeräusche, über beiden Spitzen kavernöse. Eitriger Auswurf. Abends etwas Fieber. 20. April: Ödeme stärker, ebenso die Zyanose.

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Später Aszites. Rasseln in der Lunge sehr reichlich. Digitalis. Schlechtes Allgemeinbefinden. 25. Exitus.

Autopsie: Das Herz beträchtlich vergrößert. Trikuspidalinsuffizienz, jedoch keine Störung im linken Herzen. Die Aorta zeigt bloß einige atheromatöse Stellen. Die Lungenarterie dagegen weist zahlreiche atheromatöse Plaques auf, welche man bis zu den kleinen Endverzweigungen verfolgen kann. In der rechten Lungenspitze weite Kaverne im verdichteten Gewebe. Im übrigen zerstreute (Knötchen) Granulationen. In der linken Spitze Kaverne, 2 Finger breit, das Gewebe weniger verdichtet als rechts. Alte verkreidete Tuberkel und disseminierte Knötchen.

Sauné, De l'athérome de l'artère pulmonaire. Thèse de Paris 1877, no. 367. Beobachtung (Hôpital de Pitié, Prof. Péter).

38jähriger Diener, Eintritt 26. I. 1877, leidet an Herzklopfen beim Gehen, welches jede Arbeit unmöglich macht. Beträchtliche Dyspnöe und Ödem der Beine. Die Herzpalpitationen bestehen schon lange, denselben sind Attacken von Gelenksrheumatismus vorausgegangen, welchen er vor 12 Jahren das erste Mal hatte. Seit der Zeit leidend. Oppression, schlechte Verdauung. Anämisch. Subikterische Verfärbung der Skleren und der Haut. Wangen leicht zyanotisch. Puls klein, unregelmäßig. An der Aorta zweifaches Geräusch (Insuffic. et stenosis), das systolische ist viel stärker. Präsystolisches lang dauerndes, rauhes Geräusch über der Mitralis. Später Abdominalpunktion. Im weiteren Verlauf Zunahme der Ödeme und der Atemnot. Urinverminderung. Tod 20. März.

Sektion: Linke Lunge Lungenödem, besonders an den hinteren Partien. Verdickung der adhärenten Pleura. In den Bronchien der rechten Lunge schleimigeitriger Inhalt.

Das Herz ist enorm vergrößert. Das festangewachsene Perikard kann nur mit großer Mühe abgelöst werden. Diese Adhärenzen sind in der ganzen Ausdehnung des Perikards vorhanden. Die Aortenklappen sind schlußunfähig, sehr stark verdickt, geschrumpft, das Orificium ist leicht verengt. Unter den Klappen rechts von der Mitralis ist ein indurierter Plaque auf dem Endokard. Zwei Sehnen, ausgehend von der oberen Partie des Papillarmantels, inserieren sich an der Ventrikelwand.

Die Mitralis ist sehr deformiert und das verengte Orificium nimmt eben nur die Spitze eines Zeigefingers auf. Die rechte vordere Klappe ist geschrumpft und zeigt am freien Rande eine Dicke von $^1/_2$ cm. Die Sehnen, die sich an ihr inserieren, sind verdickt und verkürzt. Die hintere Klappe ist eingerollt und gleichmäßig verdickt. Der rechte Ventrikel stark ausgedehnt, leichte fettige Entartung. Die Pulmonalarterie zeigt atheromatöse Plaques in der Höhe der Klappen. Atheromatöse Plaques finden sich in ihrem Verlauf in der Lunge, sind unregelmäßig, durchscheinend, ein wenig vorspringend und sind namentlich deutlich am Sporn der Bifurkation. Die Aorta ist nicht sehr atheromatös, man findet nur sehr kleine Plaques in der Höhe des Aortenbogens. Hydrops ascites. Intumescentia hepatis. Milzschwellung, perisplenitische Flecke.

G. Crooke, Pulmonary endarteritis. Transact. of the Pathol. Soc. London 1888, XXXIX, p. 61.

34jähriger Mann. Als Kind Scharlach. Vor 5 Jahren Gelenksrheumatismus. Keine Syphilis. Vor 19 Monaten Atembeschwerden, Schmerzen in der linken Brust-

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seite und am Rücken. Während der ersten Hälfte dieses Zeitraumes konnte er noch arbeiten, allmähliche Verschlechterung. Bei der Aufnahme Dyspnöe, leichte Ödeme.

Herzspitzenstoß im 5. I. R. 3/4 Zoll außerhalb der Papillarlinie. Lautes systolisches Geräusch über dem Mitral- und Trikuspidalostium bis zur Axillarlinie. Leises über der Aorta, doch nicht über dem Pulmonarostium, 2. Pulmonalton stark akzentuiert. Basalbronchitis. Unter größter Atemnot Exitus.

Obduktionsbefund: Stenose des Mitralostiums mit ihren gewöhnlichen Folgen und außerdem ausgedehnte Atherombildung an den Zweigen der Pulmonalarterie, stellenweise Thrombenbildung und Bindegewebswucherung in der Umgebung der Gefäße; der Stamm der Arterie, sowie der Conus arteriosus dexter dilatiert. Die Arterien des großen Kreislaufes erschienen dickwandig, aber frei von Atherom.

Die ausführlich gegebenen Krankengeschichten der beiden primären Fälle, welche Romberg und Aust beobachteten, rechtfertigen eine eingehende Darstellung ihrer Befunde.

E. Romberg (Über Sklerose der Lungenarterie. [Aus der mediz. Klinik zu Leipzig.] Deutsch. Arch. f. klin. Mediz. 1891, Bd. 48, S. 197).

Der hereditär nicht belastete, 24jährige Pat., Gärtner, hatte als Kind die Masern, vor 1½ Jahren Muskelrheumatismus, der ohne Fieber verlief und bald heilte. Nie Gelenksrheumatismus. Keine luetische Infektion. Hat stets mäßig gelebt. Sein jetziges Leiden begann ganz allmählich vor ca. 1¼ Jahren mit Kurzatmigkeit, Druck in der Magengegend. Die Kurzatmigkeit nahm immer mehr zu. Zeitweise stellten sich Kopfschmerzen, öfters Schwindelanfälle ein. Gleichzeitig fiel der Umgebung des Pat. auf, daß seine früher gesunde Gesichtsfarbe bläulich wurde. Namentlich nach körperlicher Anstrengung will Pat. oft „blitzblau" ausgesehen haben.

Trotz seiner Kurzatmigkeit konnte Pat. bis vor wenigen Wochen seine allerdings nicht schwere Arbeit verrichten. Über Herzklopfen oder stärkere Anschwellung der Füße hat Pat. nie zu klagen gehabt.

Status (28. Juli 1890): Kräftig gebauter, leidlich genährter Mann. Mäßige Dyspnöe.

Resp. 24. Leichter Ikterus. Hochgradige Zyanose der Haut und der sichtbaren Schleimhäute. An den Wangen und der Streckseite der Vorderarme und Hände Haut dunkelblau. Keine Ödeme. Herzgegend im ganzen etwas vorgewölbt.

Eine zunächst als Spitzenstoß imponierende systolische Erschütterung findet sich im 4. l. I. R. 7,5 cm links von der Mittellinie, deutlich sichtbar und sehr resistent. Fortpflanzung in die Umgebung.

Neben dieser eine schwache systol. Erschütterung im 5. I. R. in der linken Mammillarlinie. Keine epigastrische Pulsation. Die Perkussion ergibt eine starke Verbreiterung der Herzdämpfung nach rechts und links. Die relative Dämpfung findet sich im 5. r. I. R. 7 cm rechts von der Mittellinie, links vom Sternum an der oberen 3. und im 5. l. I. R. 15 cm links von der Mittellinie. Im Gegensatz zu der relativen ist die absolute Herzdämpfung nur nach links hin verbreitert. Ihre Grenzen sind an den genannten Stellen der linken Sternalrand, obere 4., linke Mammillarlinie.

Bei der Auskultation hört man in der Gegend der starken Pulsation, am lautesten an der Stelle des scheinbaren Spitzenstoßes ein systolisches, weiches, den

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1. Ton verdeckendes Geräusch und am Ende der Systole ein kurzes, sehr scharfes Geräusch. Während das 1. nur im 3. und 4. I. R. hörbar ist, wird das 2. nach der Auskultationsstelle der Pulmonalis hin sehr deutlich fortgeleitet. Man hört hier vor ihm den 1. und nach ihm den akzentuierten 2. Pulmonalton. Über der Aorta sind die Töne sehr viel leiser als über der Pulmonalis. Venen des Halses nicht erweitert. Puls klein. Arterie wenig gefüllt, weich. Mäßiges Volum. pulmon. auct. Leber vergrößert. Harn konzentriert, eiweiß- und gallenfarbstoffhaltig. Subnormale Temperatur des Kranken höchst auffallend.

Vom 10. Aug. an Zyanose zunehmend, Puls schwächer. Dann starke den Atem versetzende Schmerzen in der linken Seite des Thorax unter dem Rippenbogen. Respirationsfrequenz stieg auf das Doppelte. Ängstlicher Gesichtsausdruck.

14. August Zunahme der Schwäche, Puls unfühlbar. Exitus letalis.

Annahme eines kongenitalen Herzleidens.

Die Sektion (Dr. Lochte) ergab eine außerordentlich verbreitete hochgradige Sklerose der Lungenarterie mit konsekutiver Hypertrophie der rechten Herzhälfte. Das Herz ist fast doppelt so groß als die Faust des Mannes. Seine der Brustwand zugekehrte Fläche ist gänzlich von dem stark vergrößerten rechten Ventrikel gebildet. Vom linken Ventrikel ist fast nichts sichtbar.

Am herausgenommenen Herzen erscheint von vorn gesehen der linke Ventrikel nur als ein Anhängsel des rechten, während auf der Hinterfläche beide Ventrikel annähernd gleiche Dimensionen zeigen. Die Vergrößerung des rechten Herzens betrifft also hauptsächlich den Conus arteriosus, wie auch aus den Maßen hervorgeht:

Größter Umfang des	ganzen Herzens	30	cm
„ „ „	linken Ventrikels	11	„
„ „ „	rechten „	19	„

Von diesen 19 cm kommen auf den Conus arteriosus (von der Mitte des Septums bis zur rechten Herzkante) 13 cm, auf die Hinterfläche (in gleicher Weise gemessen) nur 6 cm. Fast noch verschiedener als die Größe der Ventrikel ist die der Herzohren. Das linke ist kaum halb so groß als das rechte. Es erscheint sogar kleiner als normal. Rechter und linker Vorhof verhalten sich entsprechend. Am aufgeschnittenen Herzen fällt der enorme Unterschied zwischen rechter und linker Herzhälfte noch mehr in die Augen. Die Wand des rechten Ventrikels ist verdickt (z. B. am Ansatz des Herzohrs auf 6 cm). Die Trabekeln stark hypertrophisch. Das Foramen ovale ist am vorderen oberen Rande für einen Bleistift von 3 mm Durchmesser eben durchgängig. Die Öffnung wird durch einen 7 mm langen Kanal gebildet, ist also von links her durch die Valvula foram. oval. reichlich gedeckt.

Umfang des Ost. tricuspid. 10 cm. Klappen zart.

Der rechte Ventrikel ist besonders im Conus arteriosus stark dilatiert. Seine Wand verdickt. Auf der rechten Kante (2,5 cm unterhalb des Sept. atrio-ventr.) mißt sie 20 mm, von denen 9 mm auf die kompakte Muskulatur, 11 mm auf die enorm hypertrophischen Trabekeln kommen. Im Conus arteriosus ist die Wand ebensodick, aber es entfallen nur 5 mm auf die kompakte Muskulatur. Das Herzfleisch ist derb, graurot.

Mit bloßem Auge ist keine Verfettung, keine Schwielenbildung nachweisbar. Nur unmittelbar unter dem Ansatz der Pulmonalklappen zeigen sich unter dem Endokard zahlreiche feinste weiße Linien und Punkte. Mikroskopisch sind die

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Muskelfasern völlig intakt. An einzelnen Stellen wenig ausgedehnte Infiltrationsherde und kleine bindegewebige Schwielen. Die gut schließenden Pulmonalklappen sind zart, glatt. Die Lungenarterie zeigt unmittelbar über den Klappen keine Besonderheiten. Ihr Umfang beträgt hier 85 mm, am Klappenansatz 80 mm, unmittelbar darunter 90 mm. Die Venae pulmonales sind an ihrer Einmündung in den linken Vorhof eng, für einen dünnen Bleistift eben durchgängig. Der linke Vorhof ist eher etwas kleiner als normal. Die Dicke seiner Wand beträgt 2—3 mm, nur die Hälfte von der des rechten.

Umfang des Ostium mitrale 10 cm. Die Klappen zart, glatt, ihre Sehnenfäden nicht verdickt. Der linke Ventrikel ist ziemlich eng und besitzt nur wenig ausgebildete Trabekel. Das keinen Defekt zeigende Septum ventric. ist von rechts her etwas in den Ventrikel hinein vorgewölbt. Die Wanddicke beträgt im venösen Teil 12—15 mm, im arteriellen 10 mm. Aortenklappen zart, glatt.

Die Aorta ist auffallend eng, Umfang dicht oberhalb der Klappen 60 mm, am Ansatz des Ductus Botalli 50 mm.

Der Ductus Botalli ist in seiner ganzen Länge geschlossen. Koronararterien und Venen bieten nichts Besonderes.

Während die Aorta, von der Enge in ihrem ganzen Verlauf abgesehen, und ihre Hauptäste sich völlig normal verhalten, zeigt die Lungenarterie bis in ihre feinsten Verzweigungen hochgradige Veränderungen. Ihr Lumen ist bis zur 2. Teilung in der Lunge selbst ziemlich gleichmäßig erweitert (Umfang des rechten Astes 50 mm, des linken 40 mm), dann in den kleineren Ästen deutlich verengert. Die Verengerung wird um so hochgradiger, je kleiner das Kaliber der betreffenden Zweige wird. Sie scheint sämtliche Verzweigungen gleichmäßig zu betreffen. —

Die Intima sämtlicher Verästelungen zeigt mehr oder minder ausgebildete sklerotische Veränderungen. Zahlreiche, mäßig in das Lumen vorragende Erhabenheiten von verschiedenster Gestalt und Größe bedecken die Innenfläche, an einzelnen Stellen, z. B. im linken Hauptast, so dicht, daß kaum ein Fleck von der Erkrankung verschont ist. Die Prominenzen sind durchscheinend, gelb. Ihre Oberfläche ist glatt. Ihre Ränder gehen allmählich in die normale Umgebung über. Diese sklerotischen Veränderungen bedingen an den größeren Ästen eine nicht unbedeutende Verdickung der Wand, an den kleineren außerdem die erwähnte Verengerung.

Außerdem fand sich eine lockere, aber totale Verklebung der rechtsseitigen Pleurablätter ohne stärkere Schwartenbildung; Ödem und Hyperämie der wenig pigmentierten, völlig normalen Lungen. —

C. Aust, Kasuistischer Beitrag zur Sklerose der Lungenarterie. Aus dem Altonaer Krankenhaus. Münchener med. Wochenschr. 1892, September, S. 689.

25jähriger Arbeiter, hereditär nicht belastet, hat in den ersten Lebensjahren lange Zeit an englischer Krankheit und später vielfach an Lungen- und Magenkatarrh gelitten. Rheumatismus oder sonstige zu Herzaffektionen führende Erkrankungen will Pat. niemals überstanden haben, luetische Infektion und Potatorium stellt er entschieden in Abrede.

Von Profession Bäcker, gab jedoch diese Beschäftigung auf und war vom 16. bis 19. Lebensjahr Glasbläser, wobei er Einatmung heißer Luft bei anstrengender Lungentätigkeit ausgesetzt war. Später suchte er sich leichtere Arbeit Seit 1¼ Jahren schon bei geringer körperlicher Anstrengung Atemnot, zeitweise Präkordialangst. Periodenweise Durchfälle, Leibschmerzen.

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Aufnahme Aug. 1891. Blasse Gesichtsfarbe mit deutlicher Zyanose. Ödeme fehlen. Atmung etwas beschleunigt. Herzdämpfung beginnt im 2. I. R. reicht nach rechts $1^1/_2$ Finger breit über den r. Sternalrand hinaus und links bis zur linken Mammillarlinie. Der 1. Ton an der Spitze unrein und geht in der Richtung nach dem Proc. xiphoid. und dem mittl. Sternum allmählich in ein kurzes Geräusch über. Der 2. Ton an der Spitze leise, nach oben zu diastol. Geräusch im 2. linken I. R. von rauschendem Charakter. Im 2. r. I. R. Herztöne leise und dumpf, kein deutliches Geräusch.

Herzaktion regelmäßig. Puls klein. Digitalis. Im weiteren Verlauf stärkere Verbreitung der Herzdämpfung nach rechts. Später Zeichen von Infarkt. Ende Sept. geringe Ödeme an den unteren Extremitäten. Puls unfühlbar, Dyspnöe und Zyanose mit starken Exazerbationen.

Es konnte keine sichere klinische Diagnose gestellt werden, Insuff. semilun. aortae oder Insuff. semil. pulmon. waren mit einer Reihe von Erscheinungen nicht stimmend. Die ungewöhnliche Dyspnöe und Zyanose (trotz Digitalis usw.) legten bei dem Mangel an ausgesprochenen sonstigen Stauungserscheinungen (insbesondere an Ödemen) die Wahrscheinlichkeit eines bestehenden kongenitalen Herzfehlers nahe.

Sektionsbefund (25. IX.) (auszugsweise): Die Querschnitte der Lungenarterie auffallend weit, ihre Wandungen verdickt und starr. Herz enorm vergrößert. Der stark dilatierte rechte Vorhof ist schwappend gefüllt mit leicht geronnenem Blut, seine Wand stark hypertrophiert (größte Wanddicke 0,6 cm).

Der rechte Ventrikel stark dilatiert, die Wand enorm verdickt (2 cm), die Papillarmuskeln vergrößert. Endokard zart. Trikuspidalklappe intakt.

Die Pulmonalarterie ist stark erweitert, der innere Umfang am Klappenansatz mißt 9,5 cm, während oberhalb der Klappen eine deutliche Tendenz zu aneurysmatischer Erweiterung besteht (größter Umfang 10,2 cm).

Die Lungenvenen sind an ihrer Einmündungsstelle in den linken Vorhof für den kleinen Finger kaum durchgängig. Die ganze linke Herzhälfte entspricht etwa der Größe eines Kinderherzens; der linke Vorhof kommt in seinem Rauminhalt etwa dem rechten Herzohr gleich. Muskulatur schwach entwickelt. Das Endokard zeigt eine grobe, strahlige Strichelung. Die Mitralklappe zart, intakt. Der kleine enge linke Ventrikel wird von dem rechten halbmondförmig umlagert, seine Wand ist dünn (1 cm); die Papillarmuskeln wenig entwickelt.

Die Aorta ist eng, für den kleinen Finger kaum durchgängig. Innerer Umfang dicht oberhalb der Semilunarklappen 6,6 cm, im Brustteil 4 cm, im Bauchteil 3,5 cm. Klappen zart, vollkommen intakt, die Intima weist einige wenige atheromatöse Einlagerungen auf. Ductus Botalli vollkommen obliteriert, Koronararterien ohne Besonderheiten.

Hochgradige Veränderungen zeigt die Arteria pulmonalis mit ihren Ästen. Vom Anfangsteile bis in die kleinsten makroskopisch noch erkennbaren Verzweigungen zeigt die Intima der im ganzen verdickten und starren Arterienwand dichte, teils plattenartige gelbe Prominenzen mit glatter Oberfläche und verwaschenen Rändern, teils feine, in der Längsachse des Gefäßes verlaufende, erhabene Strichelung. Während die großen und mittleren Äste eine erhebliche Erweiterung aufweisen, ist das Lumen der kleineren und kleinsten Verzweigungen deutlich verengt. —

Die Ätiologie dieses Falles ist völlig dunkel.

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Als schädigende Momente werden angesehen: das langdauernde angestrengte Einatmen heißer Dämpfe und die Obliteration der einen Pleurahöhle.

„Das am stärksten hervortretende Symptom der Erkrankung bildete die allmählich bis zu enormer Hochgradigkeit zunehmende Zyanose und Dyspnöe bei völligem Mangel von Ödemen, ein Verhalten, das bei dem relativ jugendlichen Alter des Pat. zunächst die Annahme eines kongenitalen Herzdefektes mit abnormer Mischung von venösen mit arteriellem Blute wohl rechtfertigte."

Folgende Beobachtung von subakuter und subchronischer Endarteriitis der Pulmonalis will ich hier vorzüglich aus dem Grund einreihen, weil der Autor auch in klinisch-diagnostischer Hinsicht verwertbare Momente zu erblicken glaubt. Infolge der kontinuierlichen Einschwemmung kleiner embolischer Partikelchen in die Lungenarterienzweige entstand ein subakutes, der Tuberkulose ähnliches klinisches Bild.

Reiche (Arteriitis pulmonalis. Jahrbücher der Hamburger Staatskrankenanstalten Jahrg. 1891/92, Bd. 3. Hamburg und Leipzig 1894, S. 287.)

1. 17jährige Blumenmacherin. Keine heredit., keine luet. Momente. Beginn des Leidens mit Herzklopfen, Husten und Appetitlosigkeit. Schmerzen in der linken Brustseite, in letzter Zeit Kurzluftigkeit. Keine Gliederschmerzen. Schmächtig gebaut, sehr abgemagert, anämisch. Starke Verkrümmung der Brustwirbelsäule. Vortreibung des Sternums. Pleurit. Reiben l. h. und r. M.

Normale Herzdämpfung. Über ihr Schwirren fühlbar. An allen Ostien ein diastolisches Geräusch. Frequente, regelm. Herztätigkeit.

Während der Folge irreguläre Temperaturen, vereinzelte Fröste.

Keine Bazillen im Sputum. Später Ödeme an den Unterschenkeln. Temperaturschwankungen, Milzschwellung. Große Euphorie. Gelegentlich Brustschmerzen und vermehrter Husten. Dämpfung über der rechten Spitze, bronchial. Exspirium.

Ödeme zunehmend, Spuren von Albumen. Urin vermindert, große Schwäche plötzlicher Exitus. (Krankheitsdauer 3 Wochen.) Sektion: Perikard durch Flüssigkeit ausgedehnt. Herz in toto vergrößert, quergestellt. Hypertrophie des rechten und linken Ventrikels.

Auf den Pulmonalklappen mehrere weißliche, knötchenartige kleine Gebilde, nicht über Stecknadelkopfgröße. Aorta glatt, unverändert. Das Anfangsstück der Pulmonalis zeigt in einer Höhe von mehreren Zentimetern oberhalb der Semilunarklappen an der Seite, an der es der Aorta anliegt, eine fast markstückgroße rundliche Region mit papillösen Exkreszenzen, an denen Thromben festhaften. Eine größere Zahl bis bohnengroße Infarkte speziell in den Oberlappen verschiedenen Alters und verschiedener Größe. Keine Tuberkulose. Mikroskop. Untersuchung der Effloreszenzen in der Art. pulmonalis auf Bazillen nach Weigert negativ.

2. 21jähriger Schneider. Im 7. Jahre Diphtherie. Nie Polyarthritis, Scharlach oder Lues. Vor $1^1/_2$ Wochen heftiges profuses Nasenbluten. Mattigkeit. Fiebergefühl, Durchfall. Durst. Appetitmangel. Dumpfer Kopfschmerz. Husten und Brustbeschwerden bestanden nicht. Blässe der Haut und Schleimhäute. Später leichte Schmerzen in der rechten Brustseite. Herzdämpfung normal. Spitzenstoß kaum fühlbar. (5. I. R.) — Töne an der Spitze rein. An der Herzbasis, am lautesten über der Art. pulmonalis ein systolisches Geräusch, bei der Diastole ein kurzer, reiner Ton. Kein Frémissement. Über beiden Jugularvenen leichtes Sausen. Halsvenen

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nicht geschwellt. Radialpuls kräftig, geringe Dikrotie. Über der rechten Spitze und r. h. u. verkürzter Schall. Im Sputum keine Bazillen. Temperaturanstieg. Meteorismus. Roseola?

Rechts über der Klavikel und links nach außen vom Herzen Rasselgeräusche. Roseola verschwunden. Diarrhöen. Gelegentlich schmerzloses Erbrechen. Blässe, keine Zyanose. Weiterhin beiderseits unter Klavikel tympanit. Beiklang. In den Spitzen spärliches Rasseln. Links seitlich umschriebenes pleuritisches Reiben. Herztöne rein, frequent. 2. Pulmonalton leicht akzentuiert. —

Über dem oberen Sternum und besonders deutlich nach links von demselben hört man (seit 3 Tagen) ein lautes, in seiner Lautheit aber variierendes Geräusch (sausend, an- und abschwellend), das nicht absetzt, aber in diesem An- und Abschwellen den Herzphasen isochron, also systolisch und diastolisch erscheint. Es wird nicht zum Rücken fortgeleitet. Kein fühlbares Frémissement. Jugularvenen kaum sichtbar; über ihnen ein leises Nonnengeräusch. Keine Zyanose, Blässe. Wenig schmerzloser Husten. Schüttelfrost. Meteorismus. — Über dem oberen Sternum und am lautesten über dem linken 2. Rippenknorpel ist dauernd jenes weiche, mehr oder weniger deutlich an die beiden Herzphasen gebundene Geräusch hörbar. Milz vergrößert. Auswurf weißschaumig, zum Teil mit blutigen Streifen untermischt. Atmung weniger frequent. Sehr geringe Zyanose. Unruhe, Benommenheit. Exitus. (Krankheitsdauer 3 Wochen.) Sektion: Rechter Ventrikel mäßig dilatiert. Endokard intakt. Klappen zart. Aorta glatt. In der Art. pulmonalis 3—4 cm oberhalb der Semilunarklappen mit breiter Basis aufsitzender Thrombus. Nach dessen Fortnahme liegt in der Pulmonalarterienwand eine ungefähr 5pfennigstückgroße leicht erhabene Fläche zutage mit unregelmäßigen, welligen Rändern. Bronchopneumonische Herde. Embolus. Staphylococcus pyog. aureus.

Wenn auch nachstehender Kasus wegen der ätiologischen und speziellen pathologischen Verhältnisse etwas abweicht, verdient er doch der klinischen Erscheinungen halber kurze Erwähnung.

Gotthardt (Ein Fall von Endarteriitis verrucosa der Arteria pulmonalis. Inaug.-Diss. München 1896).

18jähriges Mädchen, seit früher Jugend häufig Herzklopfen, seit 2 Jahren kränklich. Abgemagert, zyanotische Gesichtsfarbe.

Verbreiterung der Herzdämpfung nach rechts und links. Im 2. und 3. linken Interkostalraum starke sichtbare Pulsation und Dämpfung. Über den Ostien systolische und diastolische Geräusche. Die Diagnose wurde gestellt auf: Dilatatio cordis dext. et sin. Aneurysma aortae; Pericarditis adhaesiva (Cor villosum). Phthisis apicis sinistri. Später Ödeme, welche wiederholt auf Digitalis zurückgingen. Exitus. Sektion: Hochgradige Hypertrophie und Dilatation des Herzens, Endocarditis verrucosa der Pulmonalklappen; Endarteriitis verrucosa der Arteria pulmonalis. Pericarditis fibrinosa. (Primäre Erkrankung: Tuberkulose der linken Lunge, Pericarditis.)

Laache (Om sklerose af arteria pulmonalis og erhervet „morbus caeruleus". Norsk Magazin for Laegevidenskaben 1899, no. 1) verfügt über zwei Eigenbeobachtungen der seltenen primären Arteriosklerosis der Pulmonalis.

Beim 1. Fall, 50jährige Frau, mit den klin. Erscheinungen einer idiopath. Herzhypertrophie zeigte die Sektion außer dieser erhebliche Arteriosklerose der Aorta, ferner, daß die Intima der Pulmonalis bedeckt war mit „zerstreuten, weißlichen, bis bohnengroßen, etwas erhabenen Flecken ohne Verkalkung". Während die Skle-

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rose der Pulmonalarterie hier eine Nebenrolle spielte, hatte sie in dem 2. von Laache beobachteten Falle eine prädominierende Stellung, wobei das klinische Bild des Morbus caeruleus bestand.

Ein 59jährige, etwas neuropathisch veranlagte Frau war in ihrem 36. Lebensjahre, etwa 1 Jahr nach einer etwas rätselhaften Vergiftung mit Apfelkuchen am ganzen Körper zyanotisch geworden. Häufig sehr starke Beklemmungen. Sie litt dann später viel an Husten. Sonstige Stauungserscheinungen fehlten. (Hatte auch eine vorübergehende Parese des rechten Armes und Beines.) Ödeme erst kurz vor dem Exitus, dann aber rasch zunehmend. Im Status erwähnt: außerordentlich starke Zyanose, verbreiterte Herzdämpfung, besonders nach rechts, Spitzenstoß an normaler Stelle, 2. Pulmonalton akzentuiert, allgemeiner Hydrops; keine Kolbenfinger. — Die Autopsie ergab: erhebliche Vergrößerung des rechten Ventrikels und Atriums, weniger des linken, geringes Atherom der Aorta, der Stamm der Pulmonalis bis zu 10 cm Umfang erweitert, auch die peripherischen Verzweigungen dilatiert, überall zahlreiche gelbe, konfluierende, prominente Plaques ohne Verkalkung. In der rechten Lunge einige Infarkte. Muskatnußleber und Nierenzyanose. — Die Ursache blieb dunkel, Alkoholismus und Syphilis auszuschließen.

Für unsere klinischen Besprechungen gewinnt nachstehende Publikation, die ebenfalls den deutschen Autoren vollkommen unbekannt geblieben ist, eine besondere Bedeutung.

J. H. Bryant, Functional pulmonary incompetence, and dilatation and atheroma of the pulmonary arteries, as complications of mitral stenosis. Guy's Hospital reports 1901, vol. LV, p. 83 berichtet über funktionelle Pulmonalinsuffizienz und Dilatation und Atherom der Pulmonalarterien als Komplikation von gleichzeitiger Mitralstenose bei verschiedenen gesammelten Fällen ärztlicher Beobachtung.

Es werden die ausführlichen Krankengeschichten von 16 diesbezüglichen Fällen mitgeteilt, von denen 9 obduziert und bei 7 klinische Diagnosen gemacht wurden (7mal war Zyanose notiert, 6mal Ödeme der Füße).

1. 36jähriges Fräulein. Mit 16 Jahren Typhus, mit 21 Jahren Scharlach. Zyanose. Mitralstenose. Annahme einer Pulmonalarterien-Erweiterung. Keine Ödeme. Sektion: Verzweigungen der Pulmonalarterie atheromatös, speziell in der rechten Seite und rechten Oberlappen. Klappen normal, Nodulus Arantii verdickt. Freie Ränder herabgezogen. Arteria pulmonalis erweitert und dicker als normal, fast so dick wie die Aorta.

2. 40jährige Frau. Mit 15 Jahren rheumat. Fieber. Herzpalpitationen. Später leichte Ödeme.

Anämische Schmerzen. Pulsat. im 2. I. R. links. Epigastr. Einziehung. Abdominalpunktion. Sektion: Pleuraladhäsionen. Frische Infarkte in den Lungen. Pulmonalarterien verdickt, erweitert und atheromatös. Thromben in denselben. Die Pulmonalarterie selbst erweitert.

3. 41jährige Frau. Schweratmigkeit und Ödeme. Stets schwere Arbeit. Rheumatismus.

Diagnose: Mitralstenose. Trikuspidalinsuff. und -stenose.

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Puls irregulär und beschleunigt. Sehr starke Zyanosis. Pulsation der Halsvenen. Mäßiges Ödem der Beine. Sektion: Zyanose. Rechte Oberlappenpleura verdickt, ebenso links.

Trikuspid. leicht verdickt. Vegetationen auf der Mitralklappe, sie selbst stark verdickt und verkalkt.

Pulmonalarterie verdickt (fast so stark wie die Aorta) und erweitert. Beide Aste stark verdickt und atheromatös, ebenso die kleineren.

4. 32jährige Frau. Anasarka und Dyspnöe. Rheumatismus in der Familie. Widerholt Polyarthritis. Zyanose. Pulmonarthrombose.

Sektion: An den Pulmonarklappen (11,4 cm Umfang) zweifellose Schlußunfähigkeit. In der Arterie atheromatöse Flecken.

5. 20jähriger Mann. Vor 5 Jahren Polyarthritis. Später Pleuritis sicca. Mitral- und Trikuspidalinsuff. Kleine Blutaustritte in der Haut.

Sektion: Zahlreiche Petechien und Ekchimosen. Pleuraadhäsionen.

Pulmonalverzweigungen verdickt und atheromatös. Erweiterung des Stammes. Mitralis verdickt und verkalkt, Stenose. Aortenklappen verdickt und adhärent, doch schlußfähig. Hämaturie.

6. 22jähriger Mann. Herzpalpitationen und Schmerzen in der Brust. Vater und Mutter herzkrank. Mit 8 Jahren Scharlach. Attacken von Herzzuständen und Schwäche.

Linke Seite der Brust in der Nähe des Sternums vorgetrieben.

Sektion: Beträchtliche Arteriosklerose und Verdickung der Pulmonalarterien.

Arteria pulmonalis beträchtlich erweitert. Atherom begann von der ersten Teilung an.

7. 27jährige Frau. Wiederholte akute Rheumatismen. Mitralstenose und Perikarditis. Später wiederholte Polyarthritis. Blässe und Zyanose. Weicher und schneller Puls. Weiterhin trockene Pleuritis. Plötzlich Dyspnöe, Exitus. Sektion: Leichtes Ödem der Beine. Alte und frische Pleuritis. Totale Perikardsynechie. Verdickung der Trikuspidalis. Erweiterung der Pulmonalarterie, bedeutend weiter als die Aorta. Intima verdickt, die größeren Äste atheromatös.

8. 25jährige Frau. Vor 6 Jahren Rheumatismus. Mitralstenose und Trikuspidalinsuff. Blaß und dyspnoisch. Keine Zyanose. Ödem der Beine und vielfach Blutaustritte. Im weiteren Verlauf zunehmende Zyanose und Orthopnöe.

Sektion: Hochgradige Zyanose. Ödem der Beine. Pleuraadhäsionen.

Braune Induration und Lungeninfarkt. Perikard verdickt. Die Säume der Aortenklappe verdickt und eingerollt. Trikuspidalis verdickt und insuffiz.? Pulmonalarterie weiter als Aorta, bedeutend dicker und zeigt atheromatöse Flecken.

9. Mann. 2mal rheumatisches Fieber. Ödeme. Schwerer Atem. Blutiger Auswurf. Pleuritis sicca. Dyspnöe. Sektion: Empyem der rechten Pleura. Perikard überall verwachsen. Am rechten Ventrikel verkalkte Masse.

Erweiterung der Pulmonalis und ihrer Äste. Intimaverdickungen. Kein Atherom. Zahlreiche verkalkte und fibröse Flecken in der Aorta, speziell im Beginn.

Brünings Untersuchungen über das Vorkommen der Angiosklerose im Lungenkreislauf, Zieglers Beitr. z. path. Anat. 1901, Bd. 30, S. 457, betreffen eingehende pathologisch-anatomische und histologische Befunde und kommen für klinische Zwecke kaum in Betracht.

Nachdem in den meisten Publikationen nachstehende Arbeit voll-

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ständig unberücksichtigt blieb, sei sie etwas ausführlicher gebracht, zumal in bezug auf unser spezielles Thema die klinischen Befunde genauer detailliert erscheinen.

Percy Kidd, Sequel to a case shown at the society in 1901 as congenital morbus cordis; diffuse endoarteriitis of the pulmonary arterial system. Clin. society of London 1904.

21jähriges Mädchen, litt von Kindheit an an Herzkrämpfen und Dyspnöe. Hatte weder Muskel- noch Gelenksrheumatismus. Pat. war leicht zyanotisch, die Endphalangen der Finger zeigten jedoch keine kolbige Auftreibung.

Herz hypertrophisch, namentlich im rechten Anteil. Ictus cordis im 5. linken I. R. in der Mammillarlinie, starke Pulsation im 3.—5. I. R., die bis zum rechten Sternalrand reichte. Die Herzdämpfung begann in der linken Parasternallinie an der 3. Rippe und reichte etwas außer die Mammillarlinie nach links und etwas innerhalb des rechten Sternalrandes nach rechts.

Auskultatorisch lauter, kurzer, knackender Ton über der ganzen Herzgegend hörbar, ähnlich dem ersten Ton bei Mitralstenose. Das Maximum fand sich über dem Pulmonalostium, gelegentlich konnte man hier auch ein kurzes diastolisches Geräusch nach dem 2. Ton hören; der 1. Ton war überall rein, weich, von keinem Geräusch begleitet. Lungen und die übrigen Organe gesund. Nach Anamnese, Symptomen und Aussehen der Kranken und bei der Hypertrophie des rechten Herzens schien es ganz klar, daß es sich in diesem Falle um eine angeborene Herzerkrankung handelte. Als solche stellte ihn auch Kidd früher in der med. Gesellschaft vor. Plötzlich bekam Pat. Erbrechen, starke Dyspnöe, Leberschwellung, Ödeme an den Knöcheln und andere Stauungserscheinungen. Exitus. Obduktionsbefund: Das ganze Herz, Ventrikel und Vorhof beträchtlich erweitert und hypertrophiert, namentlich schien der Ventrikelanteil des Trikuspidalostiums dilatiert, die Pulmonalklappen waren an ihren Zipfeln und in ihrem Zentrum mit kleinen Flecken besetzt, welche den Eindruck frischer endokardialer Wucherungen machten. Sonst waren die Klappen sämtlich gesund. Weder die Aorten- noch die Pulmonalklappen waren schlußunfähig. Nichts fand sich, was die Hypertrophie und Dilatation des rechten Herzens erklären konnte. Myokard und Kranzgefäße intakt. Die Aorta zeigte sehr leichte Arteriosklerose oberhalb der Klappen. Die Pulmonalarterie war deutlich atheromatös in ihrem Stamm und ihren großen Verzweigungen, selbst die kleinen Arterienäste in den Lungen wiesen Arteriosklerose höheren Grades auf. Die Gefäße, welche die einzelnen Lungenläppchen versorgten, waren mit zahlreichen gelblichen Flecken besetzt, die Wandungen zeigten breite atheromatöse Plaques. Verkalkungen waren nicht vorhanden. Mikroskopisch fanden sich selbst in den feinsten Lungenarterien-Verästelungen alle Zeichen chronischer Endarteriitis. Die Tunica intima war verdickt, die Kapillaren in den Lungen geschlängelt, ihre Wandungen etwas verdickt.

Kidd vermutet, der schon aus frühester Jugend her datierenden Beschwerden wegen, daß die Arterienerkrankung wahrscheinlich schon aus dem intrauterinen Leben stamme.

Torhorsts (Die histologischen Veränderungen bei der Sklerose der Pulmonalarterie. Zieglers Beitr. z. pathol. Anat. 1904, Bd. 36, S. 210) Mitteilungen umfassen die pathologisch-anatomischen, speziell histologischen Veränderungen in einer Reihe von Pulmonalsklerosenfällen, bei denen nur in aller Kürze klinische Diagnosen ohne nähere Befunde beigefügt werden.

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Dasselbe läßt sich von der Arbeit Ehlers (Zur Histologie der Arteriosklerose der Pulmonalarterie. Inaug-Diss. Bonn, Jan. 1905 und Virchows Arch. 1905, Bd. 178, S. 427) sagen.

Im Comité médical, 1900, S. 2. Februar, berichteten Boinet und Poesy (Athérome de l'artère pulmonaire. Marseille médical 1906, 43. ann., no. 5, p. 151) über:

eine 57jährige Kranke. Mittlerer Ernährungszustand. Zyanose des Gesichtes, Lippen violett. Sehr zahlreiche Venenausdehnungen an den Wangen und der Nase. Beträchtliche Dyspnöe. Keine infektiösen oder rheumatischen Affektionen vorher.

Seit 8 Monaten Ödeme der Beine. Schweratmigkeit mit Exazerbationen. Beklemmungen, nervöse Anfälle mit Schwäche, Aufgeregtheit, Krämpfen.

Schwacher Herzschlag. Doppeltes Geräusch während der Systole in breiter Ausdehnung bis zur Axillarlinie. 2. Ton an Aorta und Pulmonalis schwach.

Kein Venenpuls an der Iugularis. Schwacher, leicht unterdrückbarer, unregelmäßiger Radialpuls. Emphysem speziell der Oberlappen.

Bronchitis. Im Auswurf blutige Streifen.

Sektionsbefund: Im linken Oberlappen in den größeren Gefäßen schwarzbraune Infarktpfröpfe, ebenso im rechten. Im Unterlappen Stauung.

Herz vergrößert, linke Aurikel ausgedehnt.

Mitralinsuffizienz. Aortenatherom mit Erweiterung des Bogens.

Trikuspidalinsuffizienz. Atheromatöse Plaques von 5 mm Länge, bis 1 cm Umfang vom Ursprung der Pulmonalarterie. Klappen der Pulmonalis normal.

Rechtes Herzohr sehr erweitert und hypertrophiert. Die Ränder der Trikuspidalklappe infiltriert und fibrös verdichtet.

Mönckeberg (Über die genuine Arteriosklerose der Lungenarterie. Deutsche med. Wochenschr. 1907, 1. Aug., Nr. 31, S. 1243) verfügt über 2 Erkrankungsfälle der primären Art.

1. Fall: 33jährige unverheiratete Dame, die schon vor der klinischen Aufnahme längere Zeit in ärztlicher Behandlung stand.

Erkrankte Januar 1906, nachdem sie vorher immer gesund gewesen, mit Zeichen von Anämie und allgemeiner Nervosität.

Innere Organe ohne abnormes Verhalten. Im Februar leichte Verbreiterung der Herzdämpfung nach rechts, an allen 4 Ostien Geräusche, am lautesten über der Mitralis. Puls klein und stark beschleunigt.

Wegen leichtem Fieber und Albuminurie wurde damals die Diagnose auf Endokarditis gestellt. Ende März Verschlimmerung. Erbrechen und heftige Schweiße, Urinverhaltung und Ödeme traten zeitweise auf. Die Haut bekam eine bräunlich livide Färbung.

Leichte Protrusio bulbi und Graefes Symptom in geringem Maße, jedoch keine Schilddrüsenschwellung. Aufgeregtheit und schlechter Schlaf.

5. April klin. Aufnahme. Starke Zyanose, geringer Exophthalmus. Herz beträchtlich vergrößert: $\frac{3+12}{15.3}$.

An der Spitze ein systolisches Geräusch konstant, ein diastolisches zeitweise. Tachykardie, jedoch regelmäßiger Puls. Tremor.

Urin vermindert, Spuren von Eiweiß. Blutdruck 125, später 117. Temp. normal. Atmung und Puls beschleunigt.

Man dachte an eine Forme fruste des Morbus Basedow, doch wurde auch eine Myokarditis in Erwägung gezogen. Herzschwäche. Exitus 22. IV.

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Vom Sektionsbefund sei nur das Wichtigste gebracht. Keine Zeichen für Morb. Basedowii und Addisonii. Herz bedeutend vergrößert und zwar nur im rechten Ventrikel und Vorhof. Ersterer derb und starrwandig, während die Wandung des linken außerordentlich schlaff war. Auffallende Weite des Stammes und der beiden Hauptäste der Pulmonalarterie.

Beträchtliche Erweiterung des rechten Ventrikels, seine Wand bis zu 8 mm verdickt. Venöses Ostium erweitert, Trikuspidalis und Pulmonalklappen zart, frei beweglich, von entsprechender Größe.

Der Klappenapparat des engen linken Ventrikels zeigte nichts Pathologisches, ebenso Endo- und Myokard ohne Besonderheiten. Während die normal weite Aorta eine völlig glatte Innenfläche darbot, sah man an der Pulmonalarterie und ihren Hauptästen einzelne fleckige und streifige, wenig erhabene gelblichweiße Prominenzen auf der Innenfläche. Lungen lufthaltig. Die größeren Zweige der Lungenarterie auffallend starr und dickwandig. Starke Stauung an den Organen des großen Kreislaufes. Bei mikroskopischer Untersuchung zeigte es sich, daß die sklerotischen Prozesse in den Lungengefäßen eine weit größere Ausdehnung hatten. Die Intimawucherung war an den kleineren und kleinsten Ästen relativ noch viel hochgradiger.

2. Fall: 56jähriger Mann. Klagen über Hinfälligkeit und Atemnot, Klopfen und Unruhe des Herzens. Die Untersuchung ergab eine Mitralinsuffizienz im Stadium gestörter Kompensation, starke Herzvergrößerung, besonders in der Breite, systol. Geräusch an der Spitze, starke Leberschwellung, Ödeme der Beine. Auf Koffein und Diuretin Besserung auf 4 Wochen. Gegensatz zwischen guter Herztätigkeit und Puls einer- und großer Leber und Ödemen andererseits. Weiterhin stärkere Ödeme und Atemnot. Darreichung von Digitalis versagte.

Befund an der Poliklinik: Starke Zyanose, Ödeme an Rumpf, Beinen und Händen. Radialispuls nicht zu fühlen. Starker Venenpuls. Herz vergrößert. $\frac{5:11.5}{18}$. An der Basis ein nicht besonders lauter 2. Ton. Leber vergrößert, derb. Leberpuls (Aszites?). Links Hydrothorax. Pat. starb bereits am selben Morgen nach der Aufnahme im Bade. Diagnose: Insufficientia cordis. Insufficientia valvulae mitralis.

Aus dem Sektionsbefund: Linke Lunge seitlich oben und hinten flächenhaft, unten nur strangförmig verwachsen. Rechte in ganzer Ausdehnung teils flächenteils strangförmig verwachsen.

Herz im ganzen stark vergrößert. Rechter Ventrikel stark erweitert, Wand verdickt, starr. Endokard zart. Pulmonaltaschen groß, zart. Ost. ven. dext. für 4 Finger bequem durchgängig. Vorhof mäßig erweitert.

Linker Ventrikel nicht erweitert, Wand von entsprechender Dicke.

Myokard bräunlichrot, schlaff, Endokard zart, Ansatzrand der Aortentaschen und am vorderen Mitralsegel leicht verdickt. Mitralostium für 2 Finger bequem durchgängig. Klappensegel groß, freibeweglich.

Demnach Hypertrophie des rechten Herzens ohne organische Klappenveränderung. — Autochthoner Thrombus im Pulmonalarterienstamm, der das Lumen des Stammes und rechten Hauptastes nicht ganz verlegte, so daß eine Passage freiblieb, durch die ein wahrscheinlich aus der Vena cava inf. stammender Embolus in die zum rechten Ober- und Unterlappen gehenden Äste hineinfahren konnte. Der Thrombus war infolge ausgesprochener Arteriosklerose der Pulmonalis entstanden, wobei das Gefäß weit war, mit stark verdickten Wandungen. Die Erkrankung erstreckt sich nach der mikroskop. Untersuchung nicht so weit in die

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kleineren Gefäße wie beim früheren Fall, dagegen waren die größeren stärker befallen.

Einengungen der Gefäße durch Intimawucherungen, dabei vielfach erweiterte kleinere Äste ohne Intimawucherungen.

Da es sich auch hier um ein jugendliches Individuum handelt (wie in den Fällen Romberg und Aust), möchte der Verf. möglicherweise eine angeborene Alteration der Lungengefäßwände annehmen, die zu einer vorzeitigen Schwächung mit Schädigungen der Gefäßwand und reparatorischen Wucherungen der Intima geführt haben.

Beim folgenden Krankheitsfall standen die Lungenblutungen im Vordergrund des klinischen Bildes.

Schwartz (Über einen Fall von abundanter Lungenblutung bei Mitralstenose und hochgradiger Sklerose der Arteria pulmonalis. Münchner med. Wochenschr. 1907, Nr. 13).

30jähriger M. Kein Potus, keine Geschlechtskrankheiten. Mit 24 Jahren Auftreten von Herzklopfen und Engigkeit bei raschem Gehen und Treppensteigen.

Vor 3 Jahren Bluthusten und wesentliche Verschlimmerung der Engigkeit. In der Folge mehrmals Hämoptöe. Dez. 1904 plötzlich heftiger Anfall von Atemnot mit krampfartigen Schmerzen in der Brust, 1 Tag lang sehr viel Blut ausgeworfen. Weiterhin mehrmalige solche Anfälle.

Vom Status sei hervorgehoben: Haut sehr anämisch. Lippen zyanotisch, Dyspnöe. Herzverbreiterung nach rechts, weniger nach links. Spitzenstoß 6. I. R. 1 Querf. außerhalb der Mammillarlinie.

Sehr verstärkter 1. Ton an der Spitze mit typischem präsystolischen Geräusch und verstärkter 2. Ton. Herzaktion rhythmisch, beschleunigt.

Halsvenen pulsieren synchron mit dem Spitzenstoß. Fühlbare Arterien nicht sklerotisch. Während des klinischen Aufenthaltes fast täglich Hämoptöeanfälle. (Hemiplegie.) Exitus.

Klinische Diagnose: Vitium cordis valvulare (Mitralstenose und -insuffizienz, Trikuspidalinsuffizienz), Herzfehlerlunge, multiple Lungenembolien und -infarkte, pneumonische Prozesse im rechten Unterlappen. Embolie der linken Art. fossae Sylvii.

Anatomische Diagnose: Embolie der linken Art. fossae Sylvii, Mitralstenose, Trikuspidalstenose, braune Induration der Lunge. Blutungen in die Bronchien.

Arteriosklerose der Arteria pulmonalis. Milz- und Niereninfarkte. Blutungen in die Magenschleimhaut.

In einer Arbeit Rößles (Über Hypertrophie und Organkorrelation. Münchner med. Wochenschr. 1908, 25. Febr., Nr. 8, S. 377) fand ich zwei Fälle anscheinend primärer, nur mäßiger Sklerose, die jedoch für klinisch-diagnostische Zwecke nicht in Betracht kommen.

Kitamura (Über die Sklerose der Pulmonalarterie bei fortgesetztem übermäßigen Biergenuß. Ztschr. f. klin. Med. 1908, Bd. 65, S. 14) beschuldigt übermäßigen Biergenuß als Ursache.

Sein Fall betraf einen 33jährigen M. Potator. Fettsucht. Zyanose. Keine Ödeme.

Die Pulmonalarterien sowohl in den großen, als auch in den kleinen Ästen

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auffallend weit und klaffend. Die Intima zeigt schon in den großen Ästen, in zunehmendem Maße aber nach den mittleren und kleineren Ästen hin, zahlreiche gelbweiße, z. T. konfluierende beetartige Erhebungen.

Herz stark vergrößert. Verdickung der Ventrikelwände, speziell der rechten. Klappen und Endokard zart und intakt. Muskel nirgends von Fett durchwachsen. Die Intima der Aorta zeigt nur spärliche und kleine gelbweiße Verdickungen.

In allerjüngster Zeit berührte auf dem französischen Internistenkongreß als Korreferent Jaquet (Les formes cliniques de l'arteriosclerose. X. Congr. franç. de médec. interne, Genève. 3—5. Sept. 1908. La Sem.. méd. 1908, no. 37, p. 437) das Thema. Nach ihm sind die atheromatösen Veränderungen der Pulmonalarterie nicht ausgesprochen selten, sie bieten jedoch nur ausnahmsweise ein bestimmtes klinisches Bild. Manchmal indessen kann die Pulmonalarteriensklerose schwere Störungen hervorrufen, welche ungefähr an die Symptome der Pulmonalstenose erinnern.

Bei einer Eigenbeobachtung bestanden die Erscheinungen in starker Dyspnöe, Zyanose und Erweiterung des rechten Herzens, einem systolischem Geräusch an der Basis mit Verdoppelung des 2. Tones. (Außerdem Leberschwellung und leichte Albuminurie.)

Die Autopsie ergab ein Atherom des Stammes und der großen Äste der Pulmonalarterie, Dilatation und Hypertrophie des rechten Ventrikels.

Die Arterien des großen Kreislaufs waren mit Ausnahme der Nierenarterie frei von Sklerose.

Die Arteria pulmonalis ist das funktionelle Gefäß des Respirationsorganes; als korrespondierende abführende Gefäße dienen die Venae pulmonales, welche arterielles Blut führen.

Die zwei Arteriae bronchiales gelangen aus der Aorta zur Lungenwurzel; sie sind die Vasa nutritia für Bronchien, Pulmonalarterienwand und Lungenbindegewebe. Geringe Anastomosen bestehen mit der Art. pulmonalis.

Die zugehörigen Venae bronchiales führen das Blut nur von den größeren Bronchien zurück nach dem Hilus und münden in die Azygos oder die Anonyma. Die Venen der kleineren Bronchien gehen dagegen in die Venae pulmonales über, wodurch eine wichtige Beziehung zwischen diesen infolge des gemeinsamen Abflusses besteht.

Wird der Abfluß des Blutes der Venae pulmonales z. B. bei Mitralstenose erschwert, so werden auch die Bronchialvenen mächtig erweitert.

Das bis zu einem gewissen Grad mögliche vikariierende Eintreten der Bronchialarterien bei Lungenarterienverstopfung oder hochgradiger Einengung wird schon in der älteren Literatur gewürdigt (hierüber bringt unter anderen Tiedemann eigene und fremde Befunde), eingehender beschäftigte sich hiermit Küttner, worüber bei Hart (Über die Embolie der Lungenarterie. Deutsch. Arch. f. klin. Med. 1905, Bd. 84, S. 448) Näheres zu lesen ist.

Bei Atherosklerose der Lungenschlagader sind im Verhalten des ganzen Systems verschiedene Möglichkeiten vorhanden:

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Bei geringeren Graden beschränkt sich die Veränderung auf sklerotische Fleckungen, verteilt auf größere oder kleinere Gefäßbezirke ohne Beeinflussungen des Lumens.

Daß derartige anatomische, oft fast eher histologische Vorgänge nicht ins Bereich klinisch-diagnostischer Erwägungen fallen, ist selbstredend.

Bei höheren Graden stellt sich eine wesentliche Änderung der Konsistenz mehr weniger weiter Gefäßwandungsbezirke ein, Elastizitätsverlust, nach vorausgehender Erschlaffung, Starrheit, verschiedengradige Verengung der kleineren und kleinsten Gefäßverzweigungen, welche einerseits eine gleichmäßige, bis mehr oder weniger aneurysmatische Erweiterung des Stammes oder von Hauptästen, andererseits eine allgemeine ziemlich gleichmäßige Verengerung des gesamten Gefäßes vom Ursprung an bedingen kann.

In seltenen Fällen ist eine allgemeine gleichmäßige Erweiterung vom Stamm an bis in die feinsten Verästelungen möglich.

Daß die obliterierende Endarteriitis der feinen und allerfeinsten Lungenarterienäste (z. B. in den Fällen von Aust, Romberg, Kidd, Mönckeberg, Rößle) in bezug auf klinische vorzüglich „Lokalsymptome" ein differentes Bild zu der hochgradigen Sklerose des Stammes der Pulmonalis zustande bringt, mag hier gleich hervorgehoben sein. Letztere Endarteriitis höchsten Grades, die Arteriosklerose des Wurzelgebietes, der großen Äste und deren Verzweigungen, ohne besondere Beeinflussung des Kalibers mit ausgesprochener ausgedehnter Verhärtung und Verkalkung, haben wir bei unseren Besprechungen über die klinische Diagnose dieser Affektion bei der physikalischen Untersuchung in allererster Linie im Auge.

Thoma sieht die Ursache der Arteriosklerose in einer primären Erkrankung der Gefäßwand selbst und räumt der Veränderung der Vasa vasorum nur eine untergeordnete, sekundäre Stellung ein.

Während Huchard in eben dieser Alteration der Vasa vasorum das Primäre erblickt, bedingt durch eine erhöhte Wandspannung der Arterie, welche ihrerseits wiederum mit einem gesteigerten Tonus der Gefäßmuskulatur, mit einem Gefäßkrampf in Zusammenhang gebracht wird.

Ähnlichen Annahmen neigte bereits Köster zu. Zunächst leiden die Vasa vasorum, in welchen sich ein endarteriitischer Prozeß etabliert. Hierdurch wird das an ihrer peripheren Ausbreitung liegende Gewebe der Intima schlechter ernährt und Vorbedingungen für regressive Metamorphosen geschaffen. Nach reaktiver Bindegewebshypertrophie greift der Prozeß auf Media und Adventitia über.

Nach Josué (Pathogénie de l'arteriosclérose. Soc. biol. LXIII, 29. Oct. 1907) ist die Arteriosklerose das Resultat der Verteidigungsmittel, die der Organismus aufbietet, um die Funktionsfähigkeit der Arterien zu erhalten. Hierbei kommen jedoch außer den produktiven auch degenerative Veränderungen zustande, so daß man die Arteriosklerose als eine Verletzung zwecks Verteidigung auffassen kann.

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Wir dürfen nicht vergessen, daß der Vorgang in den Gefäßen sich nicht an ein allgemeingültiges Schema hält, daß es auf die Raschheit oder Langsamkeit der Entwicklung, den Ort, die Ausdehnung, den Grad ankommt, daß dabei die Beschaffenheit des Herzens bzw. das Stadium der ursprünglichen Herzaffektion, Zustand der Lunge, die Umgebung beider (Perikard und Pleura) ankommt.

Was die Zeitdauer betrifft, so wäre mit Thoma ganz besonders zu bedenken, daß eben die Wurzel des Übels die abnorme Dehnbarkeit in den Anfangsstadien ausmacht und erst in der späteren Entwickelung durch Verdickung der Intima eine Korrektur der Schädigung, dann aber durch Schwund der Elastizität, Verhärtungs- und Sklerosierungsprozesse, Kalkablagerung usw. eine völlige Starrheit eintritt.

Was nun die bei Mitralstenose obwaltenden Verhältnisse anlangt, so folgert Gerhardt (Über die Kompensation von Mitralfehler. Arch. f. experim. Pathol. 1901, Bd. 25, S. 186) aus seinen Versuchen, daß verstärkte Arbeit des rechten Ventrikels das durch eine Mitralstenose gesetzte Hindernis kompensieren kann, und deutet sonach die bei diesem Fehler regelmäßig vorkommende Hypertrophie des rechten Ventrikels als echte Kompensationserscheinung. Es ist jedoch eine verhältnismäßig starke Steigerung des Pulmonalarteriendruckes[1]) nötig, damit der Aortendruck beeinflußt wird. Damit werden aber an die Elastizität der Lungenarterien und -kapillaren erhöhte Ansprüche gestellt. Zur Mitralstenosenkompensierung müssen sie annähernd doppelt so großem Drucke standhalten wie in der Norm. In der menschlichen Pathologie drückt sich dieses in der starken Hypertrophie des rechten Herzens aus. Infolge der dauernd gesteigerten Belastung der Lungengefäße müssen diese ähnliche Veränderungen erleiden, wie die des großen Kreislaufs unter ähnlichen Bedingungen: Wandverdickung und Einbuße der Elastizität. Letztere bedeutet aber ihrerseits wieder eine Erschwerung der Arbeit des rechten Herzens, welche stärkere Hypertrophie erfordert.

Es bildet sich sonach ein Circulus vitiosus aus, der die Kreislaufsverhältnisse trotz anfänglicher Kompensation des Klappenfehlers dauernd verschlechtert. —

Für das Verständnis des ganzen Prozesses und einer Reihe klinischer Symptome ist nicht minder wichtig ein zweiter derartiger Circulus vitiosus, der sich in den Vasa nutritia, den Bronchialarterien abspielt. An verschiedenen Stellen wurde der schlechten Füllung des linken Ventrikels und der Umstände gedacht, die eine Füllung hier ganz besonders erschweren, infolgedessen auch die Blutversorgung in diesen Gefäßen leidet, was wiederum eine Steigerung des Prozesses in den Lungenarterienwandungen zur Folge haben muß. In diesen Stadien resultiert daraus und durch den mächtigen Überdruck von innen eine starke Gefäßüberdehnung, infolge deren die Vasa vasorum komprimiert und geschädigt werden.

Aus allem, aus der hier niedergelegten Literatur und unseren Eigenbeobachtungen geht hervor, daß unter allen Herzaffektionen die Mitralstenose weitaus die Prädilektion für die Entwickelung des Prozesses in der Lungenschlagader abgibt.

Ganz und gar über das Ziel schießt jedoch Rattone (Arch. per le scienze med. 1885, t. IX, no. 1), wenn er behauptet, daß das Atherom der Pulmonalis sich konstant bei Stenosen der Mitralis findet,

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1) Das Blutdruckverhältnis in der Pulmonalarterie zur Aorta geben unter anderen an: Beutrer und Marey mit 1:3, Goltz und Gaule mit 2:5.

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wenn er auch die Beschränkung beifügt: die eine bedeutendere Hypertrophie des rechten Ventrikels zur Folge hatten.

Beim Studium des vorliegendes Gegenstandes wurde ich veranlaßt, ein möglichst großes Material von Herzkranken mit Mitralstenose in den klinischen und Sektionsprotokollen, in sonstigen Publikationen, Statistiken, Krankenhausberichten, kasuistischen Mitteilungen, Sammelreferaten u. dgl. auf das Vorkommen dieses Prozesses in den Lungengefäßen durchzuprüfen, wobei ich zu dem Resultat gelangte, daß wirklich ausgesprochene Fälle von Atherosclerosis pulmonalis ganz entschieden trotzdem auch bei diesem Herzfehler selten sind. Dies gilt nicht allein für die an und für sich nicht allzu häufige reine Mitralstenose, sondern auch für die Kombination dieser mit Insuffizienz und Vorwiegen ersterer. Immerhin ist aber das von uns in obigem erbrachte Material reichlich genug, um auch für den Praktiker volle Beachtung zu verdienen.

Es sind zum Zustandekommen solcher Prozesse unzweifelhaft noch eine Reihe von Faktoren nötig, die sich recht kompliziert gestalten können.

Eine gewisse angeborene Kleinheit und Schwäche des linken Ventrikels mit geringerer Widerstandsfähigkeit der Lungengefäße (wofür das so häufig konstatierte jugendlichere Alter spricht), Enge der Pulmonalvenen, Hypoplasie der Aorta, wiederholte Einwirkungen schwerer infektiöser Prozesse besonders in der Jugend.

Abgesehen von dem Vorkommen bei Entwicklungsstörungen des Herzens wird der für manche Fälle geltende kongenitale Charakter durch Erscheinungen gekennzeichnet, die alle Zeichen einer kongenitalen Endokarditis tragen.

Um den Rahmen der vorliegenden Mitteilung nicht zu überschreiten, können nur einige Momente in gedrängtester Kürze gestreift werden.

So verdient hervorgehoben zu werden, daß bei beiden jugendlichen Individuen (24 und 25 Jahre) von Romberg und Aust mit anscheinend primärer Arteriosklerose sehr auffällige Enge der Aorta vorhanden war (beidesmal vollständige rechtsseitige Pleurasynechie).

Im Falle Rombergs bestand eine für die Auffassung des Prozesses ungemein wichtige Störung, auf die merkwürdigerweise weder er noch andere Bearbeiter des Themas Rücksicht nahmen, nämlich „eine ganz beträchtliche Verengerung der Pulmonalvenen". Es heißt: „Die Venae pulmonales sind in ihrer Einmündung in den linken Vorhof eng, für einen dünnen Bleistift eben durchgängig." Daß eine derartige Verengung nicht gleichgültig für den Blutkreislauf in der Lunge sein kann und daß dadurch eine Ursache für wesentliche Drucksteigerung in der Pulmonalarterie gegeben erscheint, ist ohne weiteres ersichtlich.

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Im schließlichen Endresultat dürfte sie dem durch Mitralstenose ausgelösten gleichkommen.

Demnach wird auch verständlich, wenn ich an anderer Stelle sagte, daß die „rein primäre" Natur einiger dieser Beobachtungen sehr cum grano salis aufzufassen ist.

Auch in unseren beiden Fällen IX und X von Mitralstenose mit hochgradiger Atherosis der Pulmonalartie zeigten die Pulmonalvenen auffallend enge Lumina, was um so mehr Berücksichtigung verdient, als ja bekanntlich als gewöhnliches Verhalten bei der Stenosierung des linken Atrioventrikular-Ostiums naturgemäß eine ganz außerordentliche Ausdehnung des linken Vorhofes, die sich auch stets auf die Mündungen der Pulmonalvenen erstreckt, resultiert.

Die Verengerung der Pulmonalvenen (vielleicht manchmal angeboren, zum Teil jedenfalls auch auf extrauterine Prozesse infektiöser Natur bei frühzeitiger Polyarthritis, Variola usw.) würde auch die direkte unmittelbare Beeinflussung der Zirkulationsverhältnisse in der Lungenschlagader und die daraus resultierenden Folgezustände recht plausibel erscheinen lassen und verdient jedenfalls in Zukunft vollste Beachtung.

Die Ätiologie soll wenigstens in dieser Mitteilung nur vom praktisch-klinischen Standpunkt aus berücksichtigt werden. Wir begnügen uns deshalb mit dem bloßen Hinweis, daß der Pulmonalsklerose eine separate Stellung gebührt, relativ unabhängig von dem Prozeß im sonstigen arteriellen System, daher nur äußerst selten als Teilerscheinung allgemeiner Arteriosklerose auftritt; sie kann auch mitunter bei gewissen Bildungsanomalien des Herzens und der großen Gefäße gefunden werden.

Nachdem Mitralstenose[1]) noch relativ am häufigsten diesen Prozeß im Gefolge hat, wird es uns gar nicht wundernehmen, daß wir des öfteren in der Anamnese Attacken von schwerer Polyarthritis rheumatica finden. Mehrfach läßt sich auch die Kombination von Pneumonien, Emphysem und Perikarditis mit solchen konstatieren. Daß letztere für die Ausbildung derartiger Befunde begünstigend wirken können infolge Zirkulationsstörungen und mechanischer Momente (Druck, Kompression, Verzerrung), bedarf wohl keiner weiteren Auseinandersetzung.

In akuter Weise kann aber auch die Intima und im weiteren Verlauf die ganze Wand des Gefäßes direkt durch bakterielle Wirkung

1) Auf das Auftreten von Pulmonalsklerose bei Mitralstenose wiesen zuerst und vor allem Dittrich (1850), Klinger (1855), Heschl (1855) und Bamberger (1857) hin.

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oder zum mindesten durch die Toxine bei Infektionen lädiert werden, wodurch als Resultat auch ulzerierende und sklerotische Veränderungen gesetzt werden können (Diplokokkeninfektion, Strepto- und Staphylokokken, gonorrhoische Infektion).

Ohne auf die diesbezügliche Literatur einzugehen, mögen hier einige Beispiele mit klinischen Erscheinungen Platz finden, ganz besonders deshalb, weil in den Lehr- und Handbüchern und den einschlägigen kasuistischen Arbeiten diesem Moment gar keine Rechnung getragen wird. Dieselben dienen demnach auch als Paradigma für akutes Entstehen solcher Prozesse.

Nyssens, Un cas d'endartérite pulmonaire végétante et ulcereuse. Infection streptococcique. La Presse Médicale Belge 12. Mars 1893, 45. ann., no. 11, p. 81.

Mädchen von 22 Jahren. Blässe, Erbrechen. Konstantes Geräusch an der linken Seite des Sternums. Milztumor. Später etwas Zyanose. Weiterhin Erbrechen, Fieber, Schweratmigkeit.

Sektion: Herz 430 g schwer.

„Autour des artères aorte et pulmonaires il existe de l'exsudat avec villosités sous forme de plaques d'epaissement, et surtout sur l'artére pulmonaire. Les valvules pulmon. sont transparents et souples mais à leur insertion elles présentent des points d'induration des trainées de sclérose.

Palleri, Un caso di endoarterite dell' arteria pulmonare. Riforma medica 1899, no. 254. Ref. Ztrlbl. f. inn. Med. 1900, Nr. 31, S. 787.

Eine Pneumonie des linken oberen Lungenlappens, die zur Induration geführt, eine frische des linken unteren Lappens, doppelseitige serös-fibrinöse Pleuritis, ulzerierende polypöse Endoarteriitis der Semilunarklappen der Pulmonalis, ausgebreitete fibrinöse Meningitis war der Krankheitsbefund, welchen Palleri beschreibt. Es gelang ihm, aus allen Krankheitsherden Reinkulturen von Diplococcus Fränkel zu erzielen; auch gingen alle mit diesen Kulturen geimpften Kaninchen prompt an Septikämie ein. Als Seltenheit betont Palleri, daß das Herz wie alle übrigen großen Gefäße sich vollständig gesund erwies und nur die Lungenarterie an dem Krankheitsprozesse Anteil nahm.

Miura (Virchows Arch. Bd. 111, und Mitteilungen der mediz. Fakultät der Universität Tokio 1899, IV, 5) beschreibt bei Kakkepatienten Veränderungen an der Pulmonalarterie, die als Vorstufen von Arteriosklerose betrachtet werden können.

Fürth und Weber, Maligne gonorrhöische Endarteriitis der Arter. pulmonalis. Edinburgh Medic. Journ. 1905, vol. XVIII, p. 33. Ref. Fortschr. d. Med. 1905, S. 891.

27j. M., seit einigen Monaten Gonorrhöe, unregelmäßiges Fieber mit Frösten, leichte Zunahme der Pulsfrequenz. Geräusch im 2. linken I. R. (Perikarditisverdacht). 4 Monate später neben einem systolischen ein lang gezogenes diastolisches Geräusch neben deutlichem 2. Pulmonalton. Hypertrophie des r. und l. Herzens. Albuminurie. Tod 7 Monate nach der Aufnahme im Spital.

Sektion: Herzklappen normal. In der Art. pulm. 2 cm oberhalb der Pulmonalklappe eine rauhe, ca. 1″ lange und 3 mm vorragende warzige Effloreszenz, in welcher zwar Streptokokken, aber keine typischen Gonokokken gefunden wurden.

Die Erweiterung der Pulmonalis bot für das diastolische Geräusch keine genügende Erklärung, jedenfalls nicht für die frühere Zeit des Auftretens.

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Dem chronischen Alkoholismus wird von französischen Autoren eine ätiologische Bedeutung bei der Pulmonalarterien-Sklerose zugeschrieben.

Nach Lancereaux (Caillots développés dans l'artère pulmonaire a la suite d'excès alcooliques. Compt. rend. de la Soc. de biologie, 1861, ann. XIII., p. 162) existiert eine Form der chronischen Arteriitis, welche von den Autoren noch nicht beschrieben ist und sich durch Produktion neuer Membranen an der Intima charakterisiert. Diese Arteriitis findet sich besonders in der Pulmonalis. Als ätiologisches Moment glaubt Lancereaux Alkoholismus beschuldigen zu können.

Einem anderen französischen Autor, Huchard (Causes et pathogénie de l'artériosclérose, 1889, und Maladies du cœur et des vaisseaux, 1892) zufolge wirke der Alkohol in erster Linie auf die Lebersubstanz ein und rufe weiterhin hauptsächlich arteriosklerotische Veränderungen in den Pulmonalarterien hervor, eine Anschauung, die, wie Brüning (Untersuchungen über das Vorkommen der Angiosklerose im Lungenkreislauf. Zieglers Beitr. z. path. Anat. 1901, XXX, S. 457) mit Recht bemerkt, wohl von keinem anderen Forscher geteilt wird.

Mehrfach fand ich in der Literatur einen Hinweis auf Münzinger. Aus dessen Arbeit (Das Tübinger Herz. Deutsch. Arch. f. klin. Mediz. 1877, Bd. 19., S. 449) ist jedoch keinerlei Ausbeute für klinisch-diagnostische Zwecke, unseren Prozeß anlangend, erhältlich.

S. 758: „Mannigfaltig gestaltet sich das Krankheitsbild (nämlich das vom Verf. geschilderte, infolge Überanstrengung auftretende Herzleiden der Tübinger Weinhauer), wenn von den Lungen aus (dem Emphysem, der atheromatösen Entartung der Pulmonalarterie), dem Gebiete der Arterien des großen Kreislaufs (Atherom), neue Bedingungen eingreifen, welche wohlgeeignet sind, ein verworren erscheinendes und schwer zu enträtselndes Ganzes zu gestalten."

Über besondere Arteriosklerose der Pulmonalis weiß dieser Autor jedoch nicht zu berichten. In einem Fall, 55j. M., wird allerdings angegeben: „Pulmonalis mäßig erweitert, schwach atheromatös"; ebendasselbe wird auch von der Aorta bemerkt.

In einem anderen, 50j. M., heißt es von Pulmonalis und Aorta: „Etwas erweitert, leicht fettig degeneriert."

In jüngster Zeit kommt Kitamura (Über die Sklerose der Pulmonalarterie bei fortgesetztem übermäßigen Biergenuß. Ztschr. f. klin. Mediz. 1908, Bd. 65, S. 14) auf das Thema zu sprechen, indem er reichlichen Biergenuß als ursächliches Moment beschuldigt. Es stelle sich bei diesem eine echte Plethora, eine absolute Vermehrung der Blutmenge mit konsekutiver Herzhypertrophie ein, wobei sich die Erschwerung der Zirkulation, besonders im Lungenkreislauf, geltend mache.

In unseren Fällen war eine derartige Ätiologie vollständig zu negieren. Nur Fall I betraf einen ausgesprochenen Potator mit Leberzirrhose und Herzdegeneration, bei dem der Prozeß in der Lungenschlagader als zufälliger Sektionsbefund angetroffen wurde.

Was speziell den übermäßigen Biergenuß anbelangt, so würde hier das kompetenteste Urteil in dieser Frage wohl außer jedem Zweifel dem Münchener pathologischen Institut zustehen.

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Auf eine diesbezügliche Anfrage hatte Herr Prof. Dürck von dem genannten Institut die Güte mitzuteilen, „daß solche Fälle sicher mit übermäßigem Biergenuß nichts zu tun hatten". Ein Fall der letzteren Zeit des Institutes betraf ein junges Mädchen.

Im Gegensatz zur Aorta spielt bei den Schädigungen dieses Gefäßes Lues ganz sicherlich nur eine sehr untergeordnete Rolle. Näher hierauf einzugehen, würde uns von unserem klinischen Thema zu weit abseits führen.

In letzter Zeit lenkte wiederum Wiesel in mehreren Mitteilungen[1]) die Aufmerksamkeit auf die Erkrankungen arterieller Gefäße im Verlaufe akuter Infektionen.

Für die Beurteilung und das Verständnis mancher Vorkommnisse, die im folgenden auseinandergesetzt werden, scheint mir von Wichtigkeit, daß auch Schädigungen der Vasomotoren durch infektiöse Prozesse behauptet werden (Romberg).

Bei Durchsicht der Literatur in bezug auf unser Thema muß einem die häufige Erwähnung durchgemachter Infektionskrankheiten auffallen, wobei ich gerade mehrfach Überstehen verschiedenartiger schwerer solcher antraf.

In der Anamnese unserer Kranken figuriert, abgesehen von zumeist sehr schwerer und wiederholter Polyarthritis rheumatica, die zu Endokarditis mit konsekutivem Vitium cordis führte, Typhus, Pneumonie, Perikarditis, bei 3 Fällen in jüngeren Jahren **„Variola"**.

Dieser schweren Infektionskrankheit bin ich geneigt, eine entschiedene Bedeutung in der Ätiologie des abzuhandelnden Prozesses beizulegen.

Verschiedenerseits wurden Befunde von Gefäßalterationen nach Blattern erhoben.

Tiedemann (S. 172) z. B. schreibt, daß Entzündung der inneren Haut der Pulsadern oft in Begleitung hitziger, mit Fieberbewegungen verbundener exanthematischer Krankheiten eintrete, namentlich bei Masern, Scharlach und Pocken.

Auch in sonstigen älteren deutschen Hand- und Lehrbüchern über Herzkrankheiten finden sich solche Hinweise.

Tanchon (Edinburgh Journal of medical sciences July 1816) fand bei einer großen Anzahl von an Pocken Verstorbenen die verschiedensten Grade von Entzündungen der inneren Fläche des Herzens und der großen Gefäße. Er beschreibt hierbei die verschiedensten Veränderungen der Gefäße, wobei es wiederholt zu beträchtlichen Verdickungen kam.

In dem bekannten Falle Gilewskis (Wien. med. Wochenschr. 1868, Nr. 33)

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1) Zeitschr. f. Heilk. 1905 und 1907. Über Schädigungen der Gefäßwand durch Infektionskrankheiten, die dann zu Sklerose führen, liegen Untersuchungen vor unter andern von Simnitzi, Seitz und Saltykow. Thayer, Beziehungen zwischen akuten Infektionen und Arteriosklerose. Deutsche mediz. Wochenschr. 1904. L. B. S. 1515.

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von kombiniertem Herzfehler mit Aneurysma und hochgradiger Arteriosklerose der Pulmonalis überstand der Kranke Blattern, Typhus, Wechselfieber, Lungenentzündung, Gelenkrheumatismus.

Nach Thoma leidet die Dehnbarkeit und Elastizität der Gefäße bei Diphtherie und Variola.

Speziell diese verursache, wie Huchard glaubt, unter besonderen Umständen eine akute Endarteriitis.

Schrötter läßt allerdings die Frage offen, ob dieses auch für die Arteriosklerose gelte.

Martin (Revue de Méd. 1881, t. I, p. 369) wies bei Infektionskrankheiten, namentlich bei Pocken, herdweise Veränderungen an den Gefäßen, Aorta usw. nach, welche herdweise Arteriosklerose auf eine Arteriitis der Vasa vasorum zurückgeführt wird.

Von Immermann (Variola. Nothnagels Spez. Path., Bd. IV, 2. H. 1896) wird unter Komplikationen und Nachkrankheiten nur Perikarditis, in einzelnen Fällen Endokarditis ulcerosa und marantische Thrombose angeführt.

Von einer Reihe von Autoren wurden nach akuten Infektionen, speziell Typhus und Variola, akute Arteriitis, die häufig in einen chronischen Zustand, Arteriosklerose, überging, beobachtet (Ducastel, Brouardel, Blachez, Landouzy und Siredey).

Anhangsweise möchte ich beifügen, daß in einem von mir beobachteten Falle von Pulmonalkonusstenose mit Septumdefekt[1]), bei dem schwere endokarditische Prozesse am Konus und Klappenapparat, endarteriitische Fleckungen an der Arterie selbst bestanden, in früher Jugend Blattern (mit 17 Jahren Pneumonie) durchgemacht wurden.

Die Möglichkeit des klinischen Erkennens des Prozesses ist von Haus aus an das Vorhandensein einiger Bedingungen geknüpft:

1. entsprechendes Stadium (je hochgradiger und fortgeschrittener die Erscheinungen der Kompensationsstörungen und Herzensinsuffizienz, desto weniger Aussicht bietet sich für sie).

2. genügende Ausbildung des Prozesses selbst, der Intensität und Extensität nach.

3. sehr lange Beobachtungsdauer.

(Bei unseren letzten zwei Patienten erstreckte sie sich auf 5 und 7 Jahre.)

In der klinischen Symptomatologie ist für das Auftreten der allgemein klinischen Symptome das Befallensein der kleineren und kleinsten Gefäße (Endarteriitis obliterans), für die Setzung der lokalphysikalischen Befunde das hochgradige Befallenwerden des Ursprungsgebietes, Stammes der Arterie und der großen Äste maßgebend.

Wir wollen von der Atherosclerosis pulmonalis bei Mitral-

1) S. Pommer, Wiener klin. Wochenschr. 1904. (s. u.)

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stenose ohne wesentliche Alteration der Kaliberverhältnisse des Gefäßes ausgehen.

Bei der physikalischen Herzuntersuchung wollen wir derjenigen Erscheinungen gedenken, welchen eine direkte oder indirekte semiologische und diagnostische Bedeutung zukommt oder durch das Zusammentreffen gewisser Umstände eine solche erlangen.

Bei der Inspektion weisen alle Zeichen (verbreiterte undulierendpulsierende Herztätigkeit, epigastrische Pulsation, Pulsationen in der Konusgegend) auf eine mächtige Hypertrophie des rechten Ventrikels hin.

Sichtbare Pulsationen im 2. linken Interkostalraum lassen wohl zumeist eher komplikative Verhältnisse vermuten: Adhäsionen, Retraktionen am Perikard oder der benachbarten Pleura, Erweiterungen und Aneurysmabildungen des Gefäßes.

Daß die Hergegend im allgemeinen meist recht beträchtlich hervorgewölbt, demnach die Herzbuckel(Voussure)bildung eine in der Regel ganz besonders auffallend starke ist, darf uns unter solchen Umständen nicht wundernehmen, zumal es sich ja so häufig um verhältnismäßig noch recht jugendliche Personen handelt.

Das mittelst Palpation bei Mitralstenosefällen wahrzunehmende diastolische Schwirren an der Herzspitze rückt manchmal im Verlaufe der Beobachtung sukzessive mehr nach aufwärts gegen den linken Sternalrand zu (vgl. Auskultation).

Wahrscheinlich dürfte das einige Male mit der aufgelegten Hand nachgewiesene leichte rieselnde Schwirren in der Pulmonalgegend auf Bildung von endarteriitischen Verdickungen und Rauhigkeiten zurückzuführen sein.

Durch die Herzperkussion läßt sich sowohl bei den genuinen als den auf Mitralstenose beruhenden Fällen eine ganz besonders auffällige Vergrößerung des rechten Herzens feststellen. Dieser höchst beträchtlichen Verbreiterung der Herzdämpfung nach rechts, die bereits schon nachweisbar ist zu einer Zeit, wo nach allem übrigen noch kein Grund für eine derartige das Maß des Gewöhnlichen weit überschreitende Kompensation vorzuliegen scheint, steht die Kleinheit des linken Ventrikels gegenüber.

In gleicher Weise, wie man bekanntlich einen Dämpfungsstreifen am obersten rechten Sternalrand auf die arteriosklerotische Aorta bezieht, möchte ich den wiederholt nachzuweisenden und im Verlaufe langer klinischer Beobachtung an Extensität der Ausdehnung und Intensität der Schallerscheinung immer deutlicher werdenden Dämpfungsstreifen am obersten linken Sternalrand (und angrenzenden

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2. I. R.) auf die atherosklerotische Pulmonalarterie zurückführen [1]).

Allerdings ist dieser Rückschluß erst dann erlaubt, wenn durch genaue differential-diagnostische Erwägungen anderweitige Prozesse in diesem Gebiete ausgeschlossen werden können.

Der Empfindlichkeit bei tiefen Perkussionsschlägen und der Haut in dieser Region, wie sie einige Male bestand, muß gedacht werden.

Unter bestimmten Verhältnissen dürfte auch die Untersuchung in stark vorneüber gebeugter Haltung (Zunahme der Breite des Dämpfungsstreifens und der Intensität desselben in diesem Gebiet, stärkere lokale Druck- und Perkussionsempfindlichkeit) brauchbare Aufschlüsse geben.

Dieser Abschnitt der physikalischen Herzuntersuchung würde des inneren Zusammenhanges entbehren, bliebe das Verhältnis der beiden Ventrikel zueinander unbesprochen, dem eben hier auch ein gewisser semiologischer Wert innewohnt. Das Verhalten des linken Ventrikels bei Mitralstenose hängt von der Blutmenge ab, welche er noch zu bekommen in der Lage ist. Bei geringen Graden tritt keine auffallende Erscheinung ein. Stellt sich aber ein Mißverhältnis zwischen dem durch die Stenose erzeugten Hindernis und der Kraft des rechten Ventrikels ein, so leidet die Füllung, die Arbeit sinkt und die Muskulatur atrophiert.

Bei der Mitralstenose wird der Druck in der Pulmonalarterie, wie die Tierversuche Gerhardts und die von Moritz am Modell dartun, wesentlich erhöht.

In der Aorta sinkt der Blutdruck, es macht sich eine schlechte Füllung und geringe Spannung bemerkbar.

Diese Verhältnisse machen sich in noch höherem Grade bei der gleichzeitigen Sklerose der Lungenarterie geltend und kommen physikalisch-diagnostisch in einem ganz besonders hohen Grad der Vergrößerung des rechten Ventrikels bei Kleinheit des linken zum Ausdruck. Die bei den Obduktionen erhobenen Befunde erhärten dies: der hochgradig vergrößerte rechte Ventrikel verdrängt den kleinen, unansehnlichen, geschrumpften linken nach hinten und umgreift ihn förmlich. Der sozusagen rudimentäre linke Ventrikel erscheint fast als unbedeutender Appendix.

Bei abweichendem Verhalten war die Ursache unschwer zu erkennen: gleichzeitige stärkere Aortenatheromatose, Insuffizienz der Semilunaren, beträchtliche Arteriosklerose peripherer Gebiete, interstitielle Nephritis, schließlich vorausgegangene Insuffizienz der Mitralis.

Eine wertvolle Bereicherung der Herzuntersuchung auf Verkalkungsprozesse liefert die Röntgendurchleuchtung. Man kann mittels dieses Verfahrens bei Schonung pathologischer Präparate verkalkte Stellen im Myo- und Perikard, an den Klappen, an den Koronararterien nachweisen. In dieser Hinsicht bietet auch der Nachweis von Atherosklerose der Pulmonalarterie, deren Sitz und Aus-

1) Eine bandförmige Dämpfung links neben dem Sternum im 2. I. R. wurde bereits von Hamernjk und Gerhardt auf eine Erweiterung der Arteria pulmonalis bezogen.

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breitung an solchen Präparaten großen Vorteil, weil dadurch derartige für Sammlungen wertvolle Objekte vollständig geschont bleiben.

Wesentlich schwieriger gestaltet sich naturgemäß, der Respirations- und Herzbewegungen halber, die Untersuchung am Lebenden. Die technische Unvollkommenheit der damaligen Apparate gestattete keine unzweifelhaften Schlüsse aus den Befunden zu ziehen.

Daß der allerdings etwas undeutliche, verwaschene Schatten im 2. linken Interkostalraum längs der Sternallinie bei der Röntgenuntersuchung auf die arteriosklerotisch veränderte und mäßig erweiterte Pulmonalarterie zu beziehen ist, wird um so wahrscheinlicher und sicherer, als Weinberger[1]) (Atlas der Radiographie der Brustorgane. Wien, Emil Engel) durch wiederholte, an Sektionen bestätigte röntgenographische Untersuchungen feststellte, daß ein so gelagerter Schatten durch die erweiterte Pulmonalarterie bedingt ist, worauf auch Hödlmoser (Ztschr. f. klin. Mediz. 1904, Bd. 54, S. 122) hinweist.

Die Verhältnisse des Röntgenogrammes bei erweitertem linken Vorhof und erweiterter Pulmonalis berührten anläßlich der Besprechung der Rekurrenslähmung bei Mitralstenose Schrötter, Ztschr. f. klin. Med., Bd. 43, Alexander, Berl. klin. Woch. 1904, S. 135, und Frischauer, Wiener klin. Wochenschr. 1905, S. 1383.

Nach Bittorf (Die Bedeutung des linken mittleren Herzschattenbogens. Fortschr. aus d. Geb. d. Röntgenstrahlen. Bd. 9, H. 1) ist der linke mittlere Herzschattenbogen normalerweise vorwiegend von der Arteria pulmonalis und zum geringen Teil vom linken Herzohr gebildet.

Während er die Sichtbarkeit des linken Vorhofes beim Gesunden und Kranken bestreitet, läßt de la Camp (Mediz. Klinik 1905, Nr. 53, S. 1381) denselben bei sehr starker Vergrößerung, z. B. Mitralstenose bei dorsoventraler Durchleuchtung, am Schattenrande teilnehmen.

Letztere Konstatierung ist gerade für unsere Besprechung von besonderer Wichtigkeit, weil sie zur Vorsicht der Deutung der auf die Pulmonalis bezüglichen Befunde bei Mitralstenose gemahnen muß.

Eine übersichtliche Darstellung der Verhältnisse bringen in allerjüngster Zeit Brugsch und Schittenhelm (Lehrbuch klinischer Untersuchungsmethoden. Urban u. Schwarzenberg 1908. S. 236 und 255).

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1) Weinberger, Über die durch Erweiterung der Pulmonalarterie im Radiogramme entstehende Schattenform. Ber. des Kongr. f. Radiologie 1902. Derselbe, Wiener klin. Wochenschr. 1903, Nr. 42: „Im Radiogramme, entsprechend dem zweiten Interkostalraum, eine Verbreiterung des der Pulmonalarterie zukommenden Schattens."

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Meines Erachtens ließen sich auch für manche der seltensten Fälle primärer Pulmonalsklerose (bei denen Verengung der Pulmonalvenenmündungen vorliegt) bei der Röntgenuntersuchung verwertbare Anhaltspunkte und zwar in folgender Weise finden: bei Mitralstenose resultiert infolge der Dilatation des linken Vorhofs und rechten Kammer eine Art Medianstellung des Herzens, die sich im Röntgenbild als solche ausdrückt (mitralkonfiguriertes Herz Holzknechts).

Falls die Ursache in dem Hindernis gelegen ist, das die Einengung der Pulmonalvenenmündungen liefert, wie in dem ersten Fall, entfällt daher die Dilatation des linken Vorhofes, was sich im Röntgenbild durch eine andere Konfiguration des linken oberen Randes aussprechen wird, wie bei der Mitralstenose mit ihrer konsekutiven mächtigen Vorhofserweiterung und dem daraus resultierenden Durchleuchtungsbild.

Von größtem Interesse und differential-diagnostischem Werte für die Erkennung dieser beiden Zustände wären meines Erachtens Aufnahmen von Elektrokardiogrammen (Einthoven) nach Kraus und Nicolai[1]).

Liegt eine Mitralstenose zugrunde, würde sich selbe in einer mächtigen Entwicklung der Vorhofszacke kund tun, der andere Fall (Pulmonalvenenverengerung) durch ein völliges Ausbleiben oder nur minimalste Andeutung einer solchen markiert sein.

Ebenso dürfte eine Förderung der Frage von der ösophagealen Kardiogramm-Methode[2]) zu erwarten sein.

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1) Kraus und Nicolai (Über das Elektrokardiogramm unter normalen und pathologischen Verhältnissen. Berliner klin. Wochenschr. 1907, S. 765 und 811) bringen die Kurve bei Mitralstenose mit präsystolischem Geräusch und Schnapp, (röntgenorthographisch starke Ausladung des mittleren Bogens des linken Herzschattenrandes).

Am Elektrokardiogramm entspricht der zugehörigen Hypertrophie und Erweiterung des linken Atriums eine Verlängerung, Verstärkung und bzw. eine Trennung der Vorhofsystole in 2 (oder selbst mehr) Zeiten. Demgegenüber ist der 2. Teil des Kammerelektrogramms auffallend kümmerlich. A. Hoffmann (Über das menschliche Elektrokardiogramm. Rhein. westphäl. Gesellsch. f. inn. Med. Düsseldorf, März 1908) hält allerdings eine genaue Deutung wegen der noch nicht genügenden Sicherstellung der Bedeutung der einzelnen Teile zurzeit noch nicht für möglich.

2) Vergl. Minkowski, Die Registrierung der Herzbewegungen am linken Vorhofe. Deutsche mediz. Wochenschr. 1906, Nr. 31, S. 1246.

Derselbe, Zur Deutung der Herzarhythmien mittels des ösophagealen Kardiogramms. Zeitschr. f. klin. Mediz. 1907, Bd. 62, S. 371.

Rautenberg, Neue Methode der Registrierung der Vorhofpulsationen vom Ösophagus aus. Dem. 19. Nov. 1906. Vereinsbeil. d. Deutschen med. Wochenschr. 1907, Nr. 9, S. 394. Berliner klin. Wochenschr. 1907, Nr. 21. Münchner med. Wochenschr. 1907, Nr. 50.

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Alle drei Untersuchungsmethoden, Elektrokardiogramm, ösophageales Kardiogramm und Röntgenaufnahme, erlangen namentlich dann einen Wert, wenn sich wegen nicht charakteristischen oder ganz fehlenden auskultatorischen Zeichen (s. d.) einer Diagnose auf physikalischer Basis besondere Schwierigkeiten entgegenstellen.

Die stets fortschreitende Technik der Röntgendiagnostik dürfte wohl den Nachweis erheblicherer sklerotischer und Verkalkungsprozesse an der Pulmonalis, speziell am Stamm des Gefäßes, auch für den Fall ermöglichen, daß keine gleichzeitige Dilatation besteht. Das theoretische Postulat hierfür wäre eine ungewöhnliche Verstärkung der Intensität des oberen Anteiles des linken mittleren Bogens im allgemein mitralkonfigurierten Herzbild. Letzteres in voller Ausbildung bei Mitralstenose, ohne die Zeichen der linken Vorhofsdilatation bei Pulmonalvenenverengung.

Das Röntgenogramm ist jedoch hinsichtlich eines Schattens an der angegebenen Stelle durchaus nicht eindeutig.

Es können sich in der genannten Gegend Pleuraschwarten, Perikardverdickungen, chronische Pneumonie, Tumoren usw. entwickeln, die ganz ähnliche Bilder liefern.

Es gilt eben hier das bei der Perkussion Gesagte.

In ganz speziellen Fällen wirkt, in weiterer Verfolgung des Themas der pathologischen Prozesse in der Umgebung des Gefäßes, der gleichzeitige Nachweis bestimmter solcher für die klinische Diagnose unterstützend. Vor allem gilt dieses für die in diesem Gebiete lokalisierten Perikardsynechien weiterer Ausbreitung und Mächtigkeit.

Die anatomischen Verhältnisse lassen es erklärlich scheinen, daß infolge der leichteren Möglichkeit des Übergreifens von der Aorta aus die Perikarditis mit ihren Folgezuständen: Synechien und Obliterationen, eher bei mit Aortenaffektionen komplizierten Fällen statthatte.

U. a. Deroye (1870). Aorteninsuffizienz, Stenose und Insuffizienz des Mitralostiums. Linke Lunge innig mit dem Perikard verwachsen. Concretio pericardii.

Sauné (1877). Insuffic. et Stenosis aortae. Mitralstenose. Das festangewachsene Perikard kann von dem enorm vergrößerten Herzen nur mit großer Mühe abgelöst werden. Die Adhärenzen sind in der ganzen Ausdehnung des Perikards vorhanden. — Perikardobliterationen ebenfalls bei Bryant.

Derselbe, Die Registrierung der Vorhofpulsationen von der Speiseröhre aus. Deutsches Arch. f. klin. Med. 1907, Bd. 91, S. 251.

Derselbe, Zur Physiologie der Herzbewegung. Zeitschr. f. klin. Med. 1908, Bd. 65.

Janowski, Berichte der Warsch. med. Gesellsch. 1907, H. 4, S. 442.

Derselbe, Über die Bedeutung des ösophagealen Kardiogramms für die genaue Diagnose der Stokes-Adamschen Krankheit usw. Wiener med. Wochenschr. 1908, 12. Sept., S. 2017.

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Mehrmals fand sich bei den Fällen unserer Eigenbeobachtungen Perikarditis, zuweilen schon anamnestisch, mehrmals auch klinisch nachweisbar.

Im Falle III mit Mitralstenose, hochgradiger Erweiterung der Aorta bei Arteriosklerose der Brustaorta Residuen von Perikarditis.

Es braucht sich jedoch durchaus nicht immer bei einem bestehenden Aortenprozeß die Perikarditis zu entwickeln.

So blieb Fall VII mit primärer hochgradiger Aortensklerose, Dilatation, Insuffizienz der Klappen trotz des hohen Grades dieser Prozesse frei von Perikarderscheinungen.

Ungleich wichtiger für die Pathologie und Klinik der Atherosclerosis pulmonalis ist der Nachweis, daß die Perikarditis von dieser Gefäßaffektion ihren Ausgang nehmen oder umgekehrt, wenn nicht alleinige, so doch sicherlich eine Mitursache für Pulmonalsklerose abgeben kann.

Bei Fall IV wurde intra vitam der Verdacht auf „Perikardialsynechie" ausgesprochen[1]). Mitralstenose und Insuffizienz. Es bestanden hier allerdings keine auffälligen Veränderungen an der Aorta, wohl aber ausgedehnte beiderseitige Lungenadhäsionen. Perikard in ganzer Ausdehnung mit dem Herzen verwachsen.

Eine überstandene Endo- und fragliche Perikarditis fand sich im alten Krankenprotokoll aus dem Jahre 1878 bei Fall V. vermerkt.

Auch bei diesem Kasus mit Mitralstenose und Insuffizienz war ohne auffällige Aortenveränderung der Anfangsteil der Pulmonalis mit dem Herzbeutel durch feste bindegewebige Membranen verwachsen, und es ist hier nach allem zweifellos, daß die „Perikardsynechie" im innigsten Zusammenhang mit dem sklerotischen Prozeß der Pulmonalis stand, der überhaupt die Tendenz zu bindegewebigen Sklerosierungen, Übergreifen auf die Nachbarschaft mit Adhäsionen und strahligen Einziehungen zeigte.

Die Ergebnisse der Auskultation weitaus am relevantesten erfordern wegen Mannigfaltigkeit der Beziehungen und ihrer allgemeinen und prinzipiellen Bedeutung eine eingehendere Darstellung und Besprechung.

Bei oftmaliger wiederholter gründlicher Auskultation an verschiedenen Punkten der Herzbasis und deren Nachbarschaft, vermochte

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1) Wegen der allseits anerkannten Schwierigkeit der klinischen Diagnose einer Perikardverwachsung, die auch unser Thema tangiert, sei darauf verwiesen, daß diese in letzterer Zeit eine wesentliche Förderung erhielt. Ich verweise auf: Ortner, Über Concretio et Accretio cordis, Mediastinitis fibrosa partialis und die Entstehung echter systolischer Herzeinziehungen. Medizin. Klinik 1907, Nr. 37, S. 1089; Derselbe, Zur Genese und Bedeutung echter systolischer Spitzenstoßeinziehungen und eines abnormen Hochstandes des Aortenbogens in der Incisura sterni. Deutsche med. Wochenschr. 1908, Nr. 15, S. 630.

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man nicht selten eigenartig rieselnde Geräusche, die sich zumeist an die Systole hielten, am linken Sternalrand in der Höhe des 2. Interkostalraums und von da weiter nach links hinüber zu hören.

Am Zustandekommen dieser an der Pulmonaliswurzel und deren näherer Umgebung zu hörenden, nach Körperbewegung manchmal deutlicher, lauter und rauher werdenden Geräusche dürften jedenfalls verschiedene Momente partizipieren: die Unebenheiten durch die Intimaverdickungen, Rauhigkeiten der Kalkauflagerungen dieser, die Starrheit und Derbheit des Rohres, Adhäsionen mit der Umgebung, die eine gewisse Zerrung ausüben.

Daß auch Wirbelbildung im Stamme der Lungenarterie mitwirken könne, wie Romberg für seinen Fall vermutet, ist sehr wahrscheinlich.

Die ungewöhnlich starke Akzentuierung des 2. Pulmonaltones erklärt sich ohne weiteres aus der hochgradigen Hypertrophie des rechten Ventrikels und der Drucksteigerung im Pulmonalkreislauf.

Wenn wir mit anderen Autoren annehmen dürfen, daß am 2. „klingenden" Aortenton bei schwerer Atherosklerosis des Gefäßes neben dem unter höheren Druck erfolgenden Klappenschluß die Resonanzverstärkung teilhat, die durch die Verdickung, Starrheit und Einlagerung von Kalkplatten am Gefäße ausgelöst wird, so dürfte wohl auch folgerichtig eine ähnliche Annahme für die Lungenschlagader zum mindestens in der Weise gestattet sein, daß der Prozeß modifizierend auf akustische Wahrnehmungen in dieser Gegend einzuwirken vermag.

Der Übersichtlichkeit halber und zum besseren Verständnis des Folgenden soll von den für die Klinik in erster Linie in Betracht kommenden, auf Mitralfehler beruhenden Krankheitsfällen ausgegangen werden.

Bekanntlich wird in manchen Fällen von Mitralinsuffizienz das systolische Geräusch am lautesten im 2. linken Interkostalraum gehört.

Die Fortpflanzung der Herzfehlergeräusche in der Richtung des Blutstromes ist ja eine recht typische Erscheinung.

Naunyn (Über den Grund, weshalb hin und wieder das systolische Geräusch bei der Mitralinsuffizienz am lautesten in der Gegend der Pulmonalklappe zu vernehmen ist. Berlin. klin. Wochenschr. 1868, Nr. 17, S. 189) interpretiert diese spezielle Lokalisation in folgender Weise:

Der Entstehungsort ist im linken Herzohr zu suchen, welches etwa 2 Zoll (5,4 cm) nach außen vom linken Sternalrande sich um die Pulmonalarterie herumschlagend der Brustwand am nächsten kommt. Am besten wird es eben mit dem systolisch rückläufigen Blutstrom, durch den es entsteht, in dessen Richtung fortgeleitet, weshalb es hier lauter ist als an der Herzspitze. Der Grund, warum jedoch dieses nicht regelmäßig zustande kommt, liegt, wie sich Naunyn durch Messungen

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überzeugen konnte, in der verschiedenen Länge des Herzohres. Kürzere treten nicht so nahe an die Brustwand.

Von Wichtigkeit für unsere Darlegungen ist die weitere Angabe dieses Forschers, daß bei gleichzeitiger Insuffizienz und Stenose, wenn das ersterer entsprechende Geräusch auch am bezeichneten Ort am lautesten, das diastolische dagegen, besser präsystolische, konstant am stärksten in der Gegend der Herzspitze zu hören ist.

Für die Lautheit des Geräusches in der Pulmonalgegend macht Sahli die zumeist sehr beträchtliche Erweiterung des linken Vorhofes an und für sich schon verantwortlich.

Nach Curschmann (Über eine eigentümliche Lokalisation des systolischen Geräusches, besonders bei frischen Mitralklappenfehlern. Arbeiten aus der mediz. Klinik Leipzig. 1893) findet sich diese Lokalisation vorwiegend bei frisch entstandenen Klappenfehlern, bei älteren rückt es wieder an die Herzspitze, wofür er folgende Erklärung gibt: Der linke Vorhof und mit ihm das Herzohr wird oft gleich anfangs erweitert und rückt damit der Brustwand näher. Durch die später einsetzende Vergrößerung des rechten Herzens wird das ganze linke Herz wieder von der Brustwand entfernt.

Bekanntlich ist die Prädilektionsstelle für akzidentelle, anorganische, anämische Geräusche im allgemeinen die Herzbasis, und wurden eben zur Erklärung dieser systolischen Geräusche eine Menge Ursachen herangezogen.

Quincke (Berlin. klin. Wochenschr. 1870, Nr. 21) macht für akzidentelle Geräusche in der Pulmonalarterie außer den gewöhnlichen noch folgende Ursachen verantwortlich:

Einmal ein Mißverhältnis in der Weite zwischen Lungenarterie und ihrem Conus arteriosus,

und zweitens eine Abplattung der Pulmonalarterie durch die abnormerweise ihr anliegende Brustwand.

Wenn Geräusche sehr häufig fehlen, wo doch sonst alle Bedingungen hierfür vorhanden wären, wie bei Mitralstenose, so führt er dieses darauf zurück, daß in solchen Fällen die Abplattung der Art. pulm. nicht bedeutend genug ist, um zu merklichen Stromwirbeln Veranlassung zu geben.

Lüthje (Zur physikalischen Diagnostik am Herzen, speziell über systolische Geräusche an den Valvulae mitrales und pulmonales. Ärztl. Verein z. Frankfurt, 17. XII. 1906. Münch. med. Wochenschr. 1907, Nr. 10, S. 495. Mediz. Klinik 1906, Nr. 16) ist geneigt, systolische Geräusche bei Kindern, die man am lautesten über der Pulmonalis hört, durch eine möglicherweise aufgetretene Lageveränderung zwischen Sternum und Arteria pulmonalis zu erklären und eine richtige Pulmonalstenose anzunehmen, ebenso macht er auf solche Pulmonalgeräusche bei Mitralinsuffizienz aufmerksam, während Albrecht (Der Herzmuskel, seine Bedeutung für Physiologie, Pathologie und Klinik. Berlin 1903. S. 525) dagegen derartige Geräusche in einen Zusammenhang mit einer Dilatation am Conus pulmonalis bringen will.

Welche Erklärung können wir, unter der Voraussetzung, daß keine Bildungsanomalien und ähnliche Störungen vorliegen, für das in der Pulmonalgegend im Verlaufe der Beobachtung immer deutlicher werdende diastolische Geräusch geben?

Bei der ausgesprochenen Schlußunfähigkeit der Semilunarklappen der Aorta, wie sie in unserem Falle VII (55j. M.) vorlag, muß nach allem eine abnorme Querleitung des diastolischen

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Geräusches nach links angenommen werden, wofür wir auch noch andere Eigenbeobachtungen haben. In dieser Deutung bestärkt mich eine Beobachtung an der Klinik von Insuffizienz der Semilunaren der Aorta, eine junge Frau betreffend, die in den Jahren 1892—95 wiederholt ambulatorisch und klinisch behandelt wurde.

A. E., im Jahre 1894 25jährige Friseursfrau.

War in den Kinderjahren wiederholt schwer und durch lange Zeit krank. Im 12. Lebensjahr schwere Halsentzündung. Mit 18 Jahren verheiratet, in rascher Folge 5 Geburten. Schon seit der ersten gewisse Herzbeschwerden, Atemnot. Grazile, anämische Frau, Wangen und Ohren livid zyanotisch verfärbt. Herzbuckel, stark wogende Pulsation in der Herzgegend, Spitzenstoß nach aus- und abwärts gedrängt, hebend. Ungemein starkes diastolisches Schwirren im 2. und 3. linken Interkostalraum zu palpieren und zwar mehr gegen die Parasternallinie zu.

Herzdämpfung nach rechts bis zur Median-, nach links bis 2 Querfinger außerhalb der Papillarlinie verbreitert, im Längsdurchmesser verlängert. Über der Mitte des Sternums und namentlich gegen die linke Seite im 2. und 3. I. R. entsprechend der Parasternal- bis gegen die Papillarlinie zu ein lautes, schabendes, lang gezogenes diastolisches Geräusch. Alle ausgesprochenen Zeichen der Aorteninsuffizienz vorhanden (Pulsus celer, Kapillarpuls, sichtbare Pulsation der mittleren und selbst kleinen Arterien, Tönen der Gefäße, Gefäßgeräusche an der Cruralis usw.). Keinerlei Anhaltspunkte für ein Aneurysma der Aorta oder Pulmonalis, ebensowenig für Pulmonalinsuffizienz.

Klinische Diagnose: Insufficientia semilunarum aortae. Insufficientia mitralis relativa.

Bezüglich des abnorm nach links gelagerten diastolischen Geräusches wurde die Vermutung ausgesprochen, daß es sich um eine abnorme Querleitung desselben vielleicht infolge Arteriosklerose beider Gefäße der Aorta und Pulmonalis oder Verzerrung durch Pseudomembranen, vielleicht auch beides, handle.

Bei den übrigen Beobachtungen ist jedoch mangels aller Symptome, die gerade diesem Fehler in so charakteristischer Weise eigen sind, das Bestehen desselben ausgeschlossen.

Das diastolische Geräusch könnte seine Entstehung einer wirklichen oder einer relativen Insuffizienz der Pulmonalarterienklappen verdanken.

Abgesehen davon, daß erstere ganz enorm selten ist, spricht wohl auch gegen diese der Umstand, daß in der weitaus überwiegenden Mehrzahl der Fälle eine sehr ausgesprochene Akzentuierung des 2. Pulmonaltones bestand.

Die Möglichkeit, daß hierbei stets zwei Klappen noch schließend die Betonung, die dritte geschrumpft und insuffizient das Geräusch verursachte, ist wohl ganz in das Reich der Unwahrscheinlichkeit zu verbannen.

Für die relative Pulmonalklappeninsuffizienz bei Mitralstenose und Pulmonalarteriensklerose trat in erster Linie Bryant 1904 (l. c.) ein, dem anscheinend die sehr ausführliche Arbeit Pawińskis schon aus

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dem Jahre 1894 (allerdings nur für die Mitralstenose allein) entgangen war.

In der Literaturkasuistik wurden, um nicht bei jedem einzelnen Falle das gleiche wiederholen zu müssen, die Auskultationsergebnisse Bryants nicht berücksichtigt. Derselbe gibt in jedem seiner Fälle mehr oder weniger ausgesprochene, kürzere oder längere diastolische Geräusche mehr gegen die Basis, gegen die Pulmonalis zu, an.

Der historischen Treue sind wir schuldig, bevor wir Bryants Schlüsse darlegen, das von Pawiński Vorgebrachte in allergedrängtester Kürze zu erwähnen.

Pawiński (Über relative Insuffizienz der Lungenarterienklappen. Deutsch. Arch. f. klin. Mediz. 1894, Bd. 52, S. 519) führt ein diastolisches am linken Sternalrand in der Höhe des 3., zuweilen 4. I. R. (schwächer in der Parasternallinie und im 2. I. R.), das er bei kompensierten Mitralstenosen mehrmals hörte, auf eine relative Insuffizienz der Pulmonalarterienklappen zurück. Er hat dasselbe gewöhnlich bei jungen, gut genährten Individuen weiblichen Geschlechts gehört, dasselbe ist jedoch keineswegs konstant.

Trotz dieser relativen Insuffizienz trete keine Zyanose auf.

Die Gründe, warum er das Geräusch auf eine relative Pulmonalinsuffizienz und nicht u. a. als fortgeleitetes Mitralgeräusch ansieht, werden von ihm eingehend dargelegt.

Wie hier gleich vorausgeschickt werden soll, erwähnt Bryant mit keinem Worte die Möglichkeit einer klinischen Diagnose der Pulmonalarteriensklerose und zieht auch die klinischen Befunde der von ihm obduzierten Fälle diesbezüglich in gar keine weitere Erörterung. Für ihn dreht sich die ganze Frage um den Nachweis der relativen Insuffizienz der Pulmonalarterienklappen. In nähere Details einzugehen, würde uns zu weit führen, es mögen hier nur die Schlußfolgerungen Platz finden.

Als Resultat einer starken Mitralstenose wächst der Blutdruck beträchtlich in den Lungenarterien. Infolge dieses gesteigerten Blutdrucks wieder werden die kleinsten Äste der Pulmonalarterie dilatiert, hypertrophisch und atheromatös. Ferner bewirkt der gesteigerte Blutdruck in der Pulmonalarterie eine Dilatation der Arterie selbst und auch ihres Ostiums.

Infolge der Erweiterung der Lungenarterie und ihres Ostiums wiederum findet ein Rückströmen von Blut in den rechten Ventrikel wegen funktioneller Insuffizienz der Klappen statt. Das hauptsächlichste klinische Symptom dieser funktionellen Pulmonalinsuffizienz besteht in einem leichten, diastolischen Geräusch über der linken Seite des oberen Brustabschnittes; der Punkt, an dem dieses Geräusch am deutlichsten und am häufigsten hörbar ist, befindet sich im 3. linken Interkostalraum in der Mitte zwischen dem linken Sternalrand und der linken Mammillarlinie.

Obgleich klinisch diese Pulmonalinsuffizienz deutlich wahrnehmbar ist, so kann doch bisweilen die ev. Sektion an der Leiche weder eine Insuffizienz der Klappen,

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noch eine Dilatation der Pulmonalarterie aufdecken. Dieses Fehlen eines tatsächlichen Befundes ist aber kein Beweis dafür, daß nicht bei Lebzeiten wirklich eine Schlußunfähigkeit der Lungenarterie bestanden hat. Die Erklärung dieses letzten Vorganges ist folgende: Die Elastizität der Pulmonalarterie ist nicht ständig durch den wechselnden Blutdruck in der Pulmonalis beeinträchtigt worden und sobald der Blutdruck sank, hat sich das Gefäß wieder auf seine normale Größe zusammengezogen.

Zu Bryants Ansicht ist folgendes zu bemerken:

Wie aus seinen Auskultationsschemata ersichtlich, waren die diastolischen Geräusche zumeist in ziemlich großem Umfang wahrnehmbar und reichte das Gebiet häufig in der linken Sternallinie weit nach abwärts. Nur in einigen Fällen beschränkte sich das diastolische Geräusch auf eine ganz kleine Zone in der Pulmonalgegend.

Die Annahme Bryants einer relativen Pulmonalklappeninsuffizienz besteht in gleicher Weise wie die von Pawiński schon früher entwickelte bei einer Reihe von Mitralstenosen zu Recht, allerdings wird hier das Gebiet wirklicher relativer Insuffizienzen immer mehr einzuengen sein, wenigstens nach dem, was verschiedene Kliniker und Experimentalpathologen über selbe denken.

Mágnus-Alsleben (Versuche über relative Klappeninsuffizienzen. Arch. f. exper. Pathol. u. Pharmak. 1907, Bd. 57, S. 48) gibt zwar die Möglichkeit der experimentellen Erzeugung relativer Klappeninsuffizienzen an den Vorhöfen zu, doch ist eine solche nur unter ganz extremen Bedingungen zu erzielen. Auf die Kinik übertragen, möchte er relative Insuffizienzen für recht selten halten und zur Erklärung eines so häufigen Vorkommens, wie die akzidentellen Geräusche darstellen, nur in beschränktem Maße heranziehen.

Wir geben auch ohne weiteres zu, daß in manchen Fällen von Pulmonalarteriensklerose bei Mitralstenose das diastolische Geräusch über der Pulmonalis möglicherweise auf eine derartige relative Insuffizienz ihres Klappenapparates bezogen werden kann; eine Verallgemeinerung jedoch läßt dieses auskultatorische Phänomen nicht zu. Es sind nämlich, wie ja die einfach mechanischen Verhältnisse wohl ohne weiteres plausibel machen, alle jene Fälle ausgeschlossen, bei denen gleich vom Klappenringe an eine Verdickung, Verdichtung und Starre des Gefäßes Platz gegriffen. Daß unter solchen Umständen auch eine wesentliche und sogar sehr hochgradige Steigerung des intravaskulären Pulmonaldruckes eine derartige Ausdehnung, die zu einer relativen Schlußunfähigkeit hätte führen können, ein Ding der Unmöglichkeit ist, braucht wohl keine besondere Begründung.

Bei Durchsicht von Bryants gesammelten Fällen zeigt es sich denn auch, daß selbe fast durchweg die Lokalisation an den großen

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und kleinen Verzweigungen zeigten, eine hochgradige Alteration des Stammes war vielleicht nur ein einziges Mal zu supponieren.

Wie ich gleich hier unterstreichen möchte, erlangen dennoch Bryants Fälle für verschiedene im folgenden darzulegende Momente große Wichtigkeit und sind ganz besonders die auskultatorischen Befunde als recht gut verwertbare diagnostische Mitbehelfe für unsere Affektion zu betrachten, obwohl der Autor nach dieser Richtung nichts verlauten läßt.

Die bei den allermeisten Fällen der Literatur, auch die Bryants eingeschlossen, und bei unseren Eigenbeobachtungen zu konstatierende ausgesprochene, oft ganz ungewöhnlich starke Akzentuierung des 2. Pulmonaltones darf hierbei füglich doch auch in allererster Linie auf semiotische Verwertung, speziell gegen obige Verallgemeinerung Anspruch erheben.

Pawińskis Eigenbeobachtungen von relativer Pulmonalinsuffizienz bei Mitralstenose bilden in ihrem Verhalten, wie aus unseren weiteren Auseinandersetzungen hervorgeht, eine Bestätigung für unsere auskultatorischen diagnostischen Momente, wenn auch nur im exklusiven Sinne. Es konnten bei denselben keinerlei weitere von uns festgestellten Symptome der Sklerose gefunden werden, im Gegenteil zeichnete sie u. a. das Fehlen von Zyanose aus. Auch bei den Sektionen fand sich keine Spur von Atherosclerosis pulmonalis.

Auf ein für die Diagnose der Insuffizienz der Pulmonalklappen verwertbares Zeichen, exspiratorische Verstärkung des in seinem Beginne als Schwirren fühlbaren diastolischen Geräusches, machten Bernhard (Deutsch. Arch. f. klin. Med. Bd. 18) und Gerhardt (Verh. des Kongr. f. inn. Med. 1902, XI.) aufmerksam.

Auf dieses Gerhardtsche Symptom wurde von Bryant, wie er selbst angibt, nicht geprüft.

In unseren Fällen konnte irgendeine respiratorische Beeinflussung des Geräusches mit Sicherheit nicht nachgewiesen werden.

Die bekanntlich enorm seltenen „akzidentellen diastolischen Herzgeräusche“ wurden von Sahli bei Chlorose als während dieser Phase verstärkte Anteile sehr starken Nonnensausens konstatiert. (Über derartige Geräusche findet sich auch Genaueres bei Tripier und Devic in Bouchards Pathol. génér. t. IV.)

Krehl (Pathol. Physiol. 5. Aufl. 1907. S. 104) stimmt Sahli bei, daß ein Teil der so ungemein seltenen diastolischen akzidentellen Geräusche fortgeleitete Venengeräusche sind. „In anderen Fällen ist das ausgeschlossen, und dann ist gegenwärtig nicht einmal eine Vermutung über den Ursprung möglich.“

Übrigens findet sich in den verschiedensten ausgezeichneten Lehr- und Handbüchern (u. a. Gerhardt, Eulenburg, Kolle und Weintraud) keine Erwähnung dieser und der nächstfolgenden Verhältnisse.

Wie hier gleich vorweggenommen werden soll, ist das hier in Rede

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stehende fortgeleitete diastolische Mitralstenosengeräusch jedoch unschwer von derartigem blasenden Sausen zu unterscheiden, ganz besonders wegen seines Rhythmus und akustisch ganz anderen Charakters, ebenso dürfte die Differenzierung der übrigens sehr seltenen kardiopulmonären Geräusche mit dem eigenartigen Knacken[1]) keine allzu großen Schwierigkeiten machen. Im übrigen gibt ja im ersten Falle die Anämie und Chlorose, im letzteren Pulmonalaffektionen (starke Adhäsionen, Kavernen, Schwielenbildungen usw.) den richtigen Fingerzeig.

Verdoppelung des 2. Herztones an der Basis infolge asynchronen Schlusses der Aorten- und Pulmonalklappen ist nicht selten bei hoher Lungenarterienspannung infolge Mitralstenose zu hören und zeigt das Punctum maximum zumeist am linken Sternalrand und im 2. Interkostalraum.

Im Mittelpunkt der Besprechung über die auskultatorischen Zeichen steht die Möglichkeit der abnormen Lokalisation oder der Fortleitung des diastolischen Geräusches von der zweifellos bestehenden Mitralstenose aus gegen die Basis, speziell die Pulmonalarterie zu.

Die Kliniker sind wohl darüber einig, daß nur in den allerseltensten Fällen eine Fortleitung des diastolischen Geräusches von der Mitralklappe gegen die Basis zu stattfindet. Tripier und Devic (Sémiologie du cœur et des vaisseaux. Bouchard, Traité de Pathol. génér. t. IV), die in gründlichster Weise diese Verhältnisse erörtern, konnten niemals eine Fortpflanzung des Geräusches bis zur Basis feststellen.

Von Steell (The auscultatory signs of mitral stenosis: a statistical enquiry. Med. Chron. 1895, Sept.) rührt eine Übersicht über die physikalischen Befunde bei 60 Eigenbeobachtungen von Mitralstenose her.

Von den diastolischen Geräuschen waren 30% auf die Spitze beschränkt, 36,7% waren zugleich an der Spitze und oberhalb derselben zu hören und außerdem waren bloß in 13,3% der Fälle ein diastolisches Geräusch nur oberhalb der Spitze zu konstatieren. „Die Deutung dieser letzteren war nach ihm überhaupt sehr unsicher, wie denn die Kenntnisse über die diastolischen Geräusche seiner Meinung nach noch mangelhaft sind."

Bei v. Jürgensen (Erkrankungen der Kreislaufsorgane, Herzklappenfehler. Nothnagels Spez. Path. 1903, XV, S. 81) fand ich folgenden Passus:

„Bisweilen hört man das diastolische Geräusch (bei Mitralstenose) auch in dem 2. linken Interkostalraum, in der Gegend der Pulmonalarterie oder etwas nach außen davon, sogar, wenn etwa an der Herzspitze noch ein systolisches Geräusch besteht, deutlicher als an ihr. Es ist solches Verhalten auffallend, da die Richtung des Blutstromes, welcher das Geräusch bewirkt, vom Vorhof gegen den Ventrikel gerichtet ist (Liebermeister)."

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1) Dieses Geräusch ist ungemein charakteristisch. Ich möchte es mit dem Knacken vergleichen, das manche Leute durch rasches, starkes Abbiegen ihrer Fingergelenke hervorbringen können.

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Von dem Autor werden jedoch keine Erklärungsversuche für dieses exzeptionelle Verhalten, auch keine näheren Umstände angegeben, unter denen es sich findet.

Sahli (Lehrbuch. 5. Aufl. 1908) läßt zwar die präsystolischen Geräusche bei Mitralstenose an der Herzspitze am lautesten sein (wohl auch diastolische), von den rein diastolischen Mitralgeräuschen dagegen gibt er an (S. 357), daß sie häufig, trotz der nach der Herzspitze gehenden Richtung des Blutstromes mehr gegen den Vorhof zu am besten gehört werden, wohl deshalb, weil sie im Anfang der Diastole entstehen, wo der linke Vorhof weit, der linke Ventrikel dagegen noch eng ist. Nach Sahli ist es möglich, daß man mitunter bei ein und demselben Falle an der Herzbasis mehr den rein diastolischen, gegen die Herzspitze dagegen mehr den präsystolischen Anteil hört.

Bei einer einfachen unkomplizierten Mitralstenose würde jedoch eine derartige ganz abnorme Lokalisation des Geräusches allen physikalischen Gesetzen zuwider sein. Abgesehen von sonstigen ungünstigen Verhältnissen muß die einfache Erwägung, daß sich hierbei das Geräusch ganz entgegengesetzt der Blutstromrichtung fortpflanzen müßte, zur Widerlegung dieser Annahme genügen.

Im Gegenteil wissen wir ja, daß man gerade beim Aufsuchen des diastolischen (in der Regel präsystolischen) Geräusches bei der Mitralstenose infolge der durch den hypertrophischen, sehr stark vergrößerten rechten Ventrikel nach hinten und links verdrängten Lage des linken, zumeist sehr weit nach links gehen muß. Dieses Moment und die geringe Fortpflanzung des Geräusches nach aufwärts wird doch in allen Lehr- und Handbüchern als Charakteristikum für das diastolische Geräusch der Mitralstenose angeführt.

Um zu obigen Notizen von Steell und Sahli Stellung zu nehmen, wäre es ja in Übereinstimmung mit unseren Behauptungen vielleicht gar nicht ausgeschlossen, daß diese seltenen Fälle gerade solchen mit Pulmonalsklerose vergesellschafteten entsprachen.

Es verlohnt sich wohl nach unseren Darlegungen der Mühe, diesen Verhältnissen in Zukunft näher nachzugehen.

Jedenfalls erachte ich es als erwähnenswert, daß eben bei mehreren unserer Fälle von Mitralstenose mit Atherosklerose der Pulmonalis eigentümlicherweise das nach allgemeinem Urteil so ungemein seltene rein „diastolische“ Geräusch bestand, sogar in einem solchen Falle, wo Insuffizienz vorausging, deren Erscheinungen aber nach und nach durch die der Stenose so verdrängt wurden, daß schließlich diese allein zur Geltung kamen.

Wenn Sahli die Vermutung ausspricht, daß den verschiedenen Unterarten der diastolischen Geräusche (die Kompliziertheit der Einteilungen seitens verschiedener Forscher sei nur nebenbei bemerkt) möglicherweise verschiedene Grade der Stenose entsprechen, so möchte ich dem hinzufügen, daß unseres Erachtens als ein weiteres

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Moment hierfür das Verhalten der Pulmonalarterie in Betracht zu ziehen wäre.

Nach unseren Beobachtungen scheint ein rein diastolisches Geräusch mit auffällig basaler Lokalisation bei Mitralstenose sehr den Verdacht auf gleichzeitige Atherosclerosis pulmonalis zu rechtfertigen, speziell in Verbindung mit ungewöhnlich starker Zyanose, für die alle sonstigen Momente zur Erklärung nicht hinreichen.

Der Vollständigkeit halber sollen auch einige recht seltene Vorkommnisse, die jedoch zu unseren Darlegungen in Beziehung stehen, Platz finden:

In einer Mitteilung Duroziers (Du souffle veineux simulant l'insuffisance aortique — Lithiase biliaire et maladies du cœur. L'union méd. 1885, no. 126) fand ich das Auftreten eines diastolischen Geräusches über der Basis am Sternum bei einer 40jährigen Frau mit den Erscheinungen einer Mitralstenose.

Dieses Geräusch entwickelte sich erst im Verlaufe des Spitalsaufenthaltes. Es bestand Hypertrophie und Dilatation des rechten Ventrikels, Lebervenenpuls. Bei der Nekropsie das linke venöse Ostium kanalförmig verengt, nur für 1 Bleistift durchgängig. Aortenostium und Klappen intakt. Durozier vermutet eine allgemeine Neigung zu Schrumpfungsprozessen und glaubt, daß das diastolische Geräusch über dem Sternum in der Vena cava entstanden sei.

Ein auskultatorischer Befund Voisins (1874) ist von Interesse. Bei dem 45jährigen Kranken bestand bei auffallender Zyanose ein systolisches Geräusch am Angulus Ludovici, am Rand des Sternums das Geräusch sehr deutlich, manchmal verdoppelt. 2. Ton an der Basis klingend. Bei der Obduktion fand sich beträchtliche Herzhypertrophie, Trikuspidalinsuffizienz, jedoch keine Störung im linken Ventrikel. Ausgedehnte Atheromatose der Pulmonalis bis in die kleinen Endverzweigungen zu verfolgen. — Wir dürfen der Vermutung Raum geben, daß hier die auskultatorischen Verhältnisse bei Trikuspidalinsuffizienz durch die Pulmonalaffektion in ähnlicher Weise beeinflußt wurden.

Falls sich sohin trotzdem eine abnorme Fortleitung eines diastolischen Geräusches bei Mitralstenose (bei reinen unkomplizierten Fällen konnte ich, per parenthesim bemerkt, keine einzige derartige Beobachtung machen) konstatieren läßt, so müssen folgerichtig ganz eigenartige exzeptionelle Verhältnisse vorliegen.

Und solche sind eben in der arteriosklerotischen Verdickung und Verhärtung der Pulmonalis gegeben.

Durch die Verdichtung, Verdickung und Starrheit des Pulmonalarterienrohres wird ein Widerlager geschaffen, das zur Fortpflanzung und Verstärkung vibrierender Herzgeräusche sicherlich eine physikalische Grundlage abgeben kann.

Durch die schweren arteriosklerotischen Prozesse an diesem Gefäßrohr, das von Haus aus doch viel dünner, schlaffer, weicher gegenüber der Aorta ist, muß die Änderung der physikalischen Beschaffenheit um so schwerwiegender sein. Die frühere elastische, weiche, nachgiebige Suspension hat sich sonach wenigstens teilweise

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in eine unnachgiebige, derbe, starre Fixation verwandelt, zu deren Verstärkung noch ev. periarterielle Verdickungen, Verwachsungen, Membranbildungen mit sekundär möglichen Verzerrungen beitragen können.

Es sei hier ein Vergleich gestattet, dessen Mängel mir wohlbewußt sind, der aber trotzdem die Sache vielleicht etwas verständlicher machen wird. Wenn jemand eine dünne, elastisch federnde Metall- oder Hartgummiplatte an einem Ende mit der Hand frei hält, am anderen durch Anschlagen zur Schwingung bringt, so wird sich ein dumpfes, wenig intenses, schwirrendes Geräusch bemerkbar machen.

Bringt man jedoch diese elastische Platte, oder besser ein solches Rohr in eine starre Zwinge, so wird sich das nun viel lautere, schärfere Geräusch ganz besonders gegen den Fixationspunkt hin fortpflanzen und das horchende Ohr gerade an dieser Stelle den stärksten Eindruck bekommen, namentlich dann, wenn die Fixationsstelle zugleich, wie es für unser Verhältnis zutrifft, einen resonanzverstärkenden Hohlraum darstellt. Vorstehendes bezieht sich auf die Schwingungen der Wand selbst. Hinsichtlich der Verhältnisse der durchströmenden Flüssigkeit läßt sich nach dem Weberschen Vorgang für unseren speziellen Fall kaum eine richtige Anordnung, die diesem ganz entsprechen würde, treffen.

Bei hochgradiger Mitralstenose und Atherosklerose der Pulmonalarterie findet sich, wie die Autopsien beweisen, geradezu eine Umwälzung der physikalischen Verhältnisse und Relationen der einzelnen Herzabschnitte und der großen basalen Gefäße.

Der rechte Ventrikel mächtig erweitert und verdickt (mechanische Korrelation) prävaliert über den unbedeutenden, verkleinerten und geschrumpften linken Ventrikel[1]), so daß ersterer in Ausdehnung, Fassungsvermögen, Wanddicke diesen um mehrfaches übertreffen kann. (Das Verhalten des linken Vorhofes hängt in speziellen Fällen, z. T. vom Grade der Stenose ab [dabei starke Dilatation], andrerseits von der Beschaffenheit der Lungenvenenmündungen [bei Stenose dieser konsekutive Verkleinerung des schlecht gefüllten Vorhofes]).

In ähnlicher Weise hat sich der Zustand zuungunsten der Aorta mit zumeist beträchtlicher Ausdehnung und Wandverdickung des Wurzelgebietes der Pulmonalarterie verändert, wobei noch die Starre und Rigidität dieser letzteren hinzukommt.

Die mechanischen Aufhänge- und Schwerpunktsverhältnisse des

1) Das linke Herz wird häufig, ganz besonders auch bei den primären Atherosklerosen wie en miniature, den Dimensionen nach einem kindlichen Herzen gleich, angegeben.

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Organs müssen sonach eine ganz wesentliche Alteration erlitten haben. Daß diese Verschiebung der Massen auf den Schwerpunkt, die Torsion der Gefäße (Aorta und Pulmonalis bilden gewissermaßen ein Stück eines nach links gewundenen Spiralganges), das Drehungsmoment, die Herzbewegungen und Kontraktionen der einzelnen Abschnitte einen Einfluß ausüben müssen, bedarf keiner näheren Begründung, ebenso daß hierdurch mannigfache Bedingungen für Modifikationen der Fortleitung von Herzgeräuschen gegeben erscheinen.

Wenn auch nur in indirekter Beziehung stehend, verdient hervorgehoben zu werden, daß in der von Ortner (Wiener klin. Wochenschrift 1897, Nr. 33, S. 753) inaugurierten Literatur über Rekurrenslähmung bei Mitralstenose verschiedenerseits eingehende Erörterungen über die Größenverhältnisse, Lage und Dislokationen der hier in Betracht kommenden basalen Abschnitte gebracht werden.

So erwähnt u. a. Kraus das auffällig nach vorn und oben Geschobensein der Pulmonalis, ebenso Frischauer, wie durch den mächtig erweiterten linken Vorhof die Pulmonalarterie emporgehoben und nach vorn geschoben werden könne.

Daß alle die geschilderten Momente und die förmliche Umwälzung der physikalischen Verhältnisse eine Änderung der auskultatorischen Ergebnisse und eine Fortpflanzung des diastolischen (auch präsystolischen) Mitralgeräusches gegen das Pulmonalostium zu bedingen können, dürfte wohl nach allem recht wahrscheinlich geworden sein.

Eine wesentliche Stütze erfährt diese unsere Annahme durch das charakteristische Verhalten des Geräusches, das abgesehen vom rein diastolischen, auch im präsystolischen, im Rhythmus (wachtelschlagartig) und in seinem sonstigen Verhalten die mitrale Provenienz erkennen läßt.

Bei der Ortsveränderung des diastolischen Geräusches gegen die Basis und zwar dem 2. l. I. R. zu muß dem ganz allmählichen sukzessiven Eintreten und dem langsamen Aufwärtsrücken des Punctum maximum Rechnung getragen werden. Hierin möchte ich einen wichtigen Fingerzeig für den Zusammenhang mit den auch nur allmählich sich ausbildenden pathologisch-anatomischen Veränderungen an dem Gefäßrohr erblicken, die schließlich zu immer stärkerer Derbheit, Starrheit, Dicke und Unnachgiebigkeit der Pulmonalarterienwandung führen, wodurch eine ganz langsame, allmähliche Entwicklung dieser Resonanzverstärkung statthat.

Plötzliche oder zum mindesten recht rasche Änderungen der Lautheit, des Ortes, selbst Charakters von Herzgeräuschen können durch Setzung von Herzthromben entstehen, mehr subakut oder subchronisch durch Ausbildung von Herzschwielen, in beiden Fällen durch Änderung der mechanischen Zusammenziehungs-

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und Ausdehnungsfähigkeit der Muskelfasern ganzer Abschnitte des Herzens, durch Änderung ihrer Elastizität und Dichte, ihrer Eigenschwingungen infolge Bildung von, wenn es zu sagen erlaubt ist, Knotenpunkten.

Meines Erachtens dürften diese Verhältnisse auch diagnostische Würdigung beanspruchen, ganz besonders hinsichtlich vorliegenden Vitiums, da bekanntlich die Mitralstenose, wie auch Minkowski hervorhebt, physikalisch-diagnostisch von der arteriosklerotischen Myokarditis oft schwer zu unterscheiden ist.

Es erlangt sonach auch hier die zeitliche Ausbildung und Raschheit des Ablaufes bestimmter Zeichen einen gewissen semiologischen Wert.

Nach allem, wie ich mir die Verhältnisse zurecht zu legen suchte, dürfte, meiner Ansicht nach, eine Deutung der bekannten und oft zitierten Fälle Rombergs und Austs von anscheinend primärer Sklerose der Lungenarterie, deren Erklärung bisher aussteht, doch möglich sein. Merkwürdigerweise wurde auf die speziell im Falle Rombergs ganz auffällige Enge der Lungenvenen nicht Bedacht genommen[1]). Der diesbezügliche Befund lautet: Die Venae pulmonales sind an ihrer Einmündung in den linken Vorhof eng, für einen dünnen Bleistift eben durchgängig.

Nachdem von der Beschaffenheit ihrer Wandung nichts angegeben erscheint, kann allerdings kein Urteil abgegeben werden, ob nicht in ihnen, wie ja wiederholt pathologisch-anatomisch und histologisch nachgewiesen wurde, ähnliche Verdickungs- und Verhärtungsprozesse mit Verkleinerung des Lumens vorlagen, wie in den Arterien.

Das wahrscheinlichere (wenn auch nicht absolut sichere) ist es jedoch, daß der Zustand auffallender Enge der Pulmonalvenen angeboren war; dafür spricht wohl das eklatante Zurückgebliebensein des ganzen linken Herzens. Vom linken Ventrikel ist bei der Eröffnung sonst nichts sichtbar. Ausschließlich dem rechten Ventrikel gehört die stark abgerundete Herzspitze an. Von vorne gesehen erscheint der linke Ventrikel nur als ein Anhängsel des rechten, während auf der Hinterfläche beide Ventrikel annähernd gleiche Dimensionen zeigen (Herzmaße [s. o.]).

Das linke Herzohr ist kaum halb so groß als das rechte, es erscheint sogar kleiner als normal. Der enorme Unterschied zwischen rechter und linker Herzhälfte fällt am aufgeschnittenen Herzen noch mehr in die Augen. Der linke Vorhof ist eher etwas kleiner als normal. Die Dicke seiner Wand beträgt 2—3 mm, nur die Hälfte von der des rechten. Der linke Ventrikel ist ziemlich eng und besitzt nur wenig ausgebildete Trabekel. Aortenklappen zart, Aorta auffallend eng.

Aus der ganzen Beschreibung können wir auf Verhältnisse zwischen

1) Rombergs Annahme nach ein sekundärer Befund, s. S. 87.

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beiden Herzhälften mit direkter Umkehr der Norm schließen, ein Befund, wie er z. B. höchstgradigen Mitralstenosen eigen ist, wobei außerdem noch infolge der Pulmonalvenenverengung die Restrinktion des linken Vorhofes dazu kommt.

Der Zustand braucht ja nicht in dem landläufigen Sinne angeboren zu sein[1]), wie die wirklichen Herzklappenfehler und Entwicklungsstörungen; es kann sich wohl z. T. auch um eine lokalschlechtere Entwicklung der Lungenvenen und ein Zurückbleiben während des weiteren Wachstums gehandelt haben, eine ähnliche Hypoplasie, wie sie an der Aorta (hier vielleicht mehr sekundär) bestand. Der schließliche Effekt bei einer derartigen wesentlichen Restrinktion der Lungenvenen ist wohl ganz analog dem Bestehen einer Mitralstenose.

Vor 1½ Jahr bestand Muskelrheumatismus, vor 1¼ Jahr setzte sein jetziges Leiden ein; ersterem dürfte wohl hierbei nur die Rolle eines auslösenden Momentes für lange schon Vorbereitetes zukommen.

Infolge der auffälligen Pulmonalvenenverengerung Stauung durch das gesamte Lungengefäßgebiet auf die Pulmonalis und das rechte Herz, Drucksteigerung, mit abnormer Inanspruchnahme des Gefäßes und des rechten Ventrikels (reaktive Korrelation) reaktive Endarteriitis und Sklerose.

Die Verengerung der Mehrzahl der kleineren Pulmonalverzweigungen scheint allerdings nicht recht im Einklang zu stehen. Dieselbe fand jedoch nach Romberg nur auf Kosten der Wandverdickung statt. Bei noch bestehender Elastizität hätte hier immer noch eine vorherige Ausdehnung statthaben können.

Es scheint mir hierbei auch eine Art Circulus vitiosus bestanden zu haben. Der enorm erweiterte und stark hypertrophische rechte Ventrikel hat jedenfalls die Tätigkeit des enorm kleinen, fast geschrumpften linken noch mehr behindert, hierfür spricht ja auch, daß selbst noch im Leichenbefund das Septum ventriculorum von rechts her in den Ventrikel hinein vorgewölbt erscheint, um wie viel mehr muß diese Ausbauchung intra vitam zur Geltung gekommen und der Füllung des linken Ventrikels hinderlich gewesen sein. Die schlechte Ansaugung des Lungenvenenblutes tat ihr übriges. Wir dürfen deshalb unter diesen Verhältnissen sozusagen eine Stenosierung des ganzen linken Herzens durch den mächtig ausgedehnten enorm hypertrophischen rechten Ventrikel annehmen, wodurch der Kreis geschlossen erscheint.

Ganz ähnliches gilt vom Falle Austs.

Das Herz war enorm vergrößert. Die in situ sichtbare Vorderfläche desselben ist fast ausschließlich von der rechten Ventrikelwand

1) Vgl. übrigens die kongenitalen Störungen bei fötaler Endokarditis (auch im linken Herzen), welche sozusagen eine exzessive Steigerung der vorliegenden Verhältnisse darstellen. Vierordt (l. c. S. 149 und 150).

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gebildet, während von der linken Ventrikelwand nur ein schmaler Saum, etwa 3—4 cm oberhalb der Spitze beginnend, sichtbar ist.

Der stark dilatierte rechte Vorhof ist schwappend gefüllt mit Blut, seine Wand stark hypertrophiert. Der rechte Ventrikel ebenso, die Wand enorm verdickt (2 cm), die Papillarmuskeln bis auf das Zwei- bis Dreifache vergrößert. Herzfleisch derb, einzelne Papillarmuskeln mit myokarditischen Schwielen durchsetzt. Pulmonalarterie stark erweitert, innerer Umfang am Klappenansatz 9,5 cm, oberhalb der Klappen deutliche Tendenz zu aneurysmat. Erweiterung (größter Umfang 10,2).

Die Lungenvenen sind an ihrer Einmündungsstelle in den linken Vorhof für den kleinen Finger kaum durchgängig.

Die ganze linke Herzhälfte entspricht etwa der Größe eines Kinderherzens; der linke Vorhof kommt in seinem Rauminhalt etwa dem rechten Herzohr gleich. Die Muskulatur ist schwach entwickelt.

Das Endokard zeigt eine grobe, strahlig angeordnete Strichelung. Mitralklappe zart, intakt. Der kleine, enge linke Ventrikel wird von dem rechten Ventrikel halbmondförmig umlagert, seine Wand ist dünn (1 cm). Papillarmuskeln wenig entwickelt. Die Aorta ist eng, für den kleinen Finger kaum durchgängig. Innerer Umfang dicht oberhalb der Semilunarklappen 6,6, im Brustteil 4 cm, im Bauchteil 3,5 cm. Klappen zart, intakt, die Intima weist einige wenige atheromatöse Einlagerungen auf.

Halten wir diese anatomischen Befunde im Gedächtnis, so werden, wie sich aus folgendem ergibt, für unsere obige Annahme die hierbei erhobenen eigenartigen auskultatorischen Erscheinungen eine kräftige Stütze bilden.

Bei Rombergs (Deutsch. Arch. f. klin. Med. 1891, 48. Bd., S. 197) Kranken (24jährigen Mann) wurde nachstehendes konstatiert:

Bei der Auskultation hört man in der Gegend der starken Pulsation (4. I. R.), am lautesten an der Stelle des scheinbaren Spitzenstoßes ein systolisches, weiches, den ersten Ton verdeckendes Geräusch und am Ende der Systole ein kurzes, sehr scharfes Geräusch. Während das erste nur im 3. und 4. I. R. hörbar ist, wird das 2. nach der Auskultationsstelle der Pulmonalis hin sehr deutlich fortgeleitet. Man hört hier vor ihm den 1. und nach ihm den akzentuierten 2. Pulmonalton. Über der Aorta sind die Töne sehr viel leiser als über der Pulmonalis.

Austs (München. mediz. Wochenschr. 1892, 27. Sept., S. 689) Notizen besagen: 25jähriger Mann, blaß mit deutlicher Zyanose. Der 1. Ton an der Spitze ist unrein und geht in der Richtung nach dem Proc. xiphoid. und dem mittleren Teil des Sternums allmählich in ein deutliches kurzes Geräusch über, wird nach dem oberen Teil des Sternums wieder schwächer und undeutlicher.

Der 2. Ton an der Spitze ist leise und geht unmittelbar in ein die ganze Diastole ausfüllendes leises Geräusch über, welches in der Richtung nach dem oberen Teil des Sternums deutlicher wird und im 3. linken Interkostalraum in prägnantester Weise einen rauschenden Charakter, wie bei Insuffizienz der großen Gefäße, zeigt. Im 2. rechten I. R. und dem angrenzenden Sternalteile sind die Herztöne leise und dumpf, ein deutliches Geräusch ist daselbst nicht zu konstatieren.

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Im weiteren Verlaufe wechselt der Befund, indem das systolische Geräusch zeitweise schwächer wird, selbst gelegentlich ganz verschwindet, das diastolische dagegen in Charakter und Intensität unverändert bleibt.

Weiteres scheinen für unsere Behauptungen folgende Befunde zu sprechen:

Kidd (Clin. Soc. of London 1904). 21jähriges Mädchen. Auskultatorisch ein lauter, kurzer, knackender Ton über der ganzen Herzgegend hörbar, ähnlich dem 1. Ton bei Mitralstenose. Das Maximum fand sich über dem Pulmonalostium, gelegentlich konnte man hier auch ein kurzes diastolisches Geräusch nach dem 2. Ton hören. Der erste Ton war überall rein, weich, von keinem Geräusch begleitet.

Romberg und Aust möchten das systolische Geräusch an der Pulmonalis z. T. auf eine Wirbelbildung im Stamme der Lungenarterien beziehen.

„Über die Entstehung des am Ende der Systole hörbaren, scharfen Geräusches kann Romberg sich jedoch kein Urteil bilden." Überhaupt bietet nach ihm die Pathologie seines Falles manche Schwierigkeiten, die man wohl zu deuten versuchen, aber nicht mit Sicherheit erklären kann.

Aust glaubt die etwas ungewöhnliche Lokalisation des zweifellos durch relative Insuffizienz der Arteria pulmonalis hervorgerufenen diastolischen Geräusches durch die Hypertrophie des rechten Ventrikels und Vorhofes und die dadurch bedingte Überlagerung und Verdrängung der linken Herzhälfte genügend erklären zu können.

Nach obigen ausführlich dargelegten anatomisch-physikalischen Herzverhältnissen können wir, ohne der Sache Zwang anzutun, eine förmliche Stenosierung des linken Herzens und mithin des linken Atrioventrikularostiums durch Druck von außen annehmen.

Es dürfte nun nicht allzugewagt sein, anzunehmen, daß an dem so schwer zu deutenden diastolischen Geräusch ein von dieser sozusagen äußeren Mitralstenose fortgeleitetes partizipierte, wobei außerdem eine eben durch dieselben Umstände resultierende relative Pulmonalinsuffizienz im Sinne Bryants mit ihrem Geräusch Anteil haben konnte.

Wieviel dann auf das eine oder andere im einzelnen Fall zu setzen ist, entzieht sich intra vitam vorderhand jeglicher Beurteilung.

Wir wissen aus zahlreichen Beispielen, daß einerseits schwere angeborene Herzfehler ohne jegliche Geräusche am Herzen verlaufen können, andererseits, daß gerade von den erworbenen Vitiis die Mitralstenose[1]) dasselbe Verhalten zeigen kann.

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1) U. a. konnte Hilton Fagge 40 Fälle mit genauem Obduktionsbefund sammeln, bei denen keine Spur von Geräuschen zu entdecken war.

Der enormen Seltenheit und des klinisch-diagnostischen Interesses halber soll auch der entgegengesetzten Möglichkeit: funktionelle Mitralstenose, Pseudomitralstenose (wohl noch sehr fraglichen Charakters) gedacht werden.

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In der Praxis können wir daher in einem konkreten Falle mit allen sonstigen bisher entwickelten klinischen Zeichen vor die Entscheidung gestellt werden, denselben auch ohne Bestehen auskultatorischer Phänomene einzureihen. Daß hier die Diagnose auf große Schwierigkeiten stößt, ist fraglos.

Eine äußerst sorgfältige Anamnese (wie das Verhalten bei der Geburt und frühesten Kindheit war, ob gewisse Infektionskrankheiten, speziell Polyarthritis, Variola, Scharlach bestanden), die eigenartigen Zyanosekrisen und typischen Anfälle (Hypercyanosis intermittens angiosclerotica dolorosa [adyspnoica] s. u.) können im jeweiligen Falle einen Fingerzeig geben.

Im großen und ganzen würde Infantilismus mit trophischen Störungen und Verminderung des Intellektes[1]), ebenso ausgesprochene Trommelschlegelfingerbildung eher für ein kongenitales Vitium sprechen; weiterhin sind Anfälle von Kopfschmerz, Schwindel, ganz besonders aber Ohnmachtsanwandlungen und vor allem konvulsivische und epileptiforme Zustände diesem in erster Linie eigen.

In Zusammenfassung der auskultatorischen Phänomene, vor allem bei Beurteilung des interessanten diastolischen Geräusches in der Pulmonalostiumgegend, gelangen wir zu dem Ergebnis, daß ebensowenig die Verallgemeinerung für stetes Vorhandensein einer relativen Pulmonalklappeninsuffizienz, wie die, daß ihm unter allen Umständen eine Fortleitung des diastolischen resp. präsystolischen Mitralstenosengeräusches zugrunde liegt, zutrifft. Die auf anatomischen Veränderungen aufgebaute physikalische Möglichkeit ist für beides ausschlaggebend.

Eine relative Semilunarenschlußunfähigkeit wird dann zu supponieren sein, wenn es sich um eine Atherosklerose der großen Äste,

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Dmitrenko, Le rétrecissement mitral relatif. Rev. de méd. 1907, t. XXVII, p. 286.

Perlis, Kasuist. Beitrag zur Kenntnis der anorganischen präsystol. Geräusche an der Herzspitze. Inaug.-Dissert. Berlin 1907.

Ceconi (Mitralspasmus, Riforma med. 1908, no. 30) gibt die Krankengeschichte und den Obduktbefund eines Falles, in dem klinisch die Diagnose Mitralstenose zweifellos war.

Bei der Autopsie bloß Hypertrophie und Dilatation des r. Ventrikels bei normalem Verhalten sämtlicher Klappen, weshalb eine funktionelle Mitralstenose oder Pseudomitralstenose anzunehmen sei, über deren Ursachen die Ansichten noch geteilt sind.

1) Um die Ähnlichkeit beider Zustände noch größer zu gestalten, kommt es nämlich in seltenen Fällen auch bei der Mitralstenose, wie u. A. Brissaud, Mecklen, Ferranini erwähnen, zu einem Zurückbleiben der körperlichen und geistigen Entwicklung. Inwieweit jedoch hier bis zu einem gewissen Grad kongenitale Momente mitwirken, möge dahingestellt bleiben.

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Verzweigungen usw. handelt mit hauptsächlichem Freibleiben oder nur sehr geringer Entwicklung im Stamm und vor allem bei Nachgiebigkeit dieses und des erweiterten Wurzelgebietes und Klappenringes.

Andererseits setzt die Fortleitung des Mitralgeräusches zur Basis ein ganz besonderes Prävalieren des Prozesses in den Stammbezirken voraus.

Schließlich ist es nach der Lage der Dinge gar nicht ausgeschlossen, sondern sogar sehr wahrscheinlich, daß gegebenenfalls beide Momente konkommittieren, wobei es nach der Natur des jeweiligen Kasus darauf ankommt, welches von ihnen vorherrscht.

Bei unseren Darlegungen setzten wir immer ein gewisses Unbeeinflußtsein der Kaliberverhältnisse des Gefäßes voraus.

Wir dürfen das Kapitel über die physikalische Untersuchung nicht schließen, ohne in allergedrängtester Kürze zum wenigsten der beiden Extreme: hochgradigste Verengerung und der Aneurysmabildung zu gedenken.

Es würde auch zu weit führen, alle hier möglichen Verhältnisse und Kombinationen zu besprechen.

Je mehr sich die Stenosierung dem Pulmonalklappenringe nähert, um so ähnlicher werden die dadurch gesetzten Erscheinungen denen der Klappenstenose sein.

Je entfernter selbe zur Ausbildung kommt, desto mehr treten die Erscheinungen in ihr Recht, auf welche eine Reihe von Autoren (namentlich auch bei von außen gesetzten Hindernissen) hingewiesen haben:

Litten, Über Verengerungen im Stromgebiete der Lungenarterie, über deren Folgen und die Möglichkeit, dieselben während des Lebens zu diagnostizieren. Berl. klin. Wochenschr. 1882, S. 425.

Aufrecht, Systolische und diastolische Geräusche, entstanden durch Verengerung des Strombettes des linken Pulmonalastes. Arch. f. klin. Mediz. 1876, Bd. 18.

Mader, Beiträge zur Auskultation des Herzens und der großen Gefäße. Wien. mediz. Wochenschr. 1903, Nr. 1.

Weinberger, Über periphere Verengerung der Pulmonalarterien und die klinischen Zeichen derselben. Wiener klin. Wochenschr. 1903 Nr. 42 und Nachtrag Nr. 44.

Weiß, Zur Diagnose der langsamen Verstopfung der Lungenarterie. Ztschr. f. klin. Medizin 1907, LXII, S. 481.

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Das andere Extrem stellt die Erweiterung bis zur wirklichen Aneurysmabildung dar, wobei bezüglich der klinischen Diagnose letzterer hingewiesen sei u. a. auf: Skoda (1864), Gilewski (Wiener mediz. Wochenschr. 1868), Buchwald (Deutsche mediz. Wochenschr. 1878), Storch (I.-Diss. Breslau 1899), Lissauer (Virchows Arch. 1905, CLXXX) und vor allem auf Henschen (Das Aneurysma arteriae pulmonalis. Volkmanns Sammlung klin. Vortr. 1906. Inn. Mediz. Nr. 126/127).

Aufs äußerste kompliziert können die Verhältnisse durch die verschiedenartigsten Kombinationen beider werden.

Bei sehr eingehender und durch große Zeiträume hindurch möglicher Untersuchung dürfte sich die klinische Diagnose auch hinsichtlich der Kaliberverhältnisse weiter ausbauen lassen, wenigstens in bezug auf extremes Verhalten nach beiden Richtungen hin, in ganz besonders günstigen Fällen vielleicht sogar auf verschiedene Kombinationen solcher Verhältnisse.

Unter den **klinischen Erscheinungen** verdienen Lungenblutungen, die oft abundant sein und sich über geraume Zeit hinziehen können, unser vollstes Augenmerk.

Bekanntlich spielt die „hémoptysie cardiaque" in den Mitteilungen französischer Autoren in der Herzpathologie eine große Rolle.

Als Ursache kommen vor allem in Betracht Embolie der Arteria pulmonalis, venöse Stauung, insbesondere in Verbindung mit Degeneration der Gefäßwände, wobei der erhöhte Druck zu Dilatation, kapillärer Aneurysmen- und Varizenbildung, Dehiszenzen der alterierten Gefäßschichten und zu Berstungen und Blutungen führt.

Als zugrunde liegende Herzaffektionen dominieren weitaus Mitralfehler und hier wiederum am häufigsten Stenosen.

Unter 72 Fällen von „hémoptysie cardiaque" Dorgeins (Thèse de Bordeaux 1894/95) wurde 27 mal Mitralstenose, 16 mal Mitralinsuffizienz, 10 mal Kombination beider gefunden. Ausnahmsweise erklärt er Pulmonalsklerose als Ursache von Hämoptöe (Fälle von Lancereaux und Cruveilhier).

In dieser Fassung ist der Satz sicherlich nicht zutreffend. Die Seltenheit bei der Lungenarteriensklerose ist nur so aufzufassen, daß diese gegenüber den übrigen Störungen in der Frequenz in den Hintergrund tritt.

An und für sich ist aber gerade die Pulmonalsklerose durch öftere Hämoptöe ausgezeichnet.

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Um einige Beobachtungen aus der Literatur herauszugreifen, seien u. a. erwähnt solche von Klinger (1855), Bristowe (1860), Martineau (1861), Deroye (1870), Rosapelly (1872), Sauné (1872), Yeo (1873), Voisin (1874), Kirchner (1883), Rattone (1885), Wilbouschewitch (1891), Bryant (1901), McPhedran u. Mackenzie (1903), Bosc (1904), Schwartz (1907).

Einige Male waren mehr oder weniger auch klinisch ausgesprochene hämorrhagische Infarzierungen die Ursache (z. B. bei Deroye, Sauné, Rosapelly). — Im Falle Schwartz[1]) standen die Lungenblutungen weitaus im Vordergrunde aller klinischen Erscheinungen. Für den Kliniker gewinnen das höchste Interesse jene Fälle, bei denen ein primäres Herzleiden und die Zeichen eines Lungeninfarktes nicht nachweisbar sind. Daß hier die Annahme einer Tuberkulose der Lunge mit Hämoptöe sehr naheliegt, ist ohne weiteres begreiflich.

s. Bosc, Endocardite pulmonaire aiguë avec rétréc. ayant simulé la tuberc. pulm. Bull. Soc. de pédiat. Paris 1904, VI, p. 299.

Auch im Falle Schwartz wurde der Kranke bei der Aufnahme der Tuberkulosenstation zugewiesen.

Im Gegensatz zu Schwartz, der in der Literatur keine einwandsfreien Fälle von Lungenblutung infolge Ruptur der Pulmonalis finden konnte und angibt, daß unter den von ihm erwähnten Umständen von abnormer Zerreißlichkeit der Gefäßwand keine Rede sein kann, möchte ich auf einige Literaturangaben hinweisen, die diese seltene Möglichkeit dartun und auch Eigenbeobachtungen hierfür erbringen.

Für eine leichtere Zerreißlichkeit spricht schon die Angabe Tiedemanns, daß man an mehreren Stellen membranöse Schichten ablösen konnte, wobei sich speziell leicht die innere Haut des Gefäßes stückweise ablösen ließ.

Nach Bamberger (Lehrbuch. 1857) hat der Prozeß häufig Gefäßrupturen und infolgedessen hämorrhagische Infarkte zur Folge.

Endarteriitis und Atherom kann Rattone (l. c.) zufolge auch primär zu Lungenhämorrhagien führen.

Bei v. Jürgensen (Herzklappenfehler. Nothnagels Spez. Path. 1903, XV, S. 83—86) fand ich eine Notiz, die die abnorme Lädierbarkeit und Zerreißlichkeit des Gefäßes unter solchen Umständen bekundet.

Beobachtung II: Hochgradige Muskelschwäche des Herzens, welche die Anwesenheit einer Mitralinsuffizienz und der Stenose des Ostium venosum sinistrum nicht erkennen ließ. Geringe Stenose des Ostium venos. dext.

Hydrops fehlt trotz schwerer Erscheinungen von Herzschwäche vollkommen.

Tod durch Verschluß der linken Carotis cerebralis. 58j. Frau. $3^1/_2$ Jahre klinische Beobachtung.

Bei der Sektion fand sich zudem die Pulmonalarterie in ihrer Wand verdickt, ebenso ihre Verzweigungen in der Lunge, deren Intima an vielen Stellen verfettet, brüchig erscheint und in großen Fetzen leicht abziehbar ist.

1) Schwartz, Über einen Fall von abundanter Lungenblutung bei Mitralstenose und hochgradiger Sklerose der Arteria pulmonalis. Münchener med. Wochenschr. 1907, Nr. 13.

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Wenn auch hier nicht in die näheren pathologisch-anatomischen und histologischen Verhältnisse eingegangen werden kann, so erlangt doch der Rupturbefund an der Pulmonalarterie in unserem Falle IV wegen der ungewöhnlichen Seltenheit solchen Vorkommnisses erhöhtes Interesse. Es zeigte sich bei dieser 45jährigen Frau bei der Nekropsie Stenose und Insuffizienz der Mitralis, adhäsive Perikarditis, Pleuritis, hochgradige Sklerose und Erweiterung der Pulmonalarterien, besonders der rechten, und eine ganz beträchtliche, in die Breite und Tiefe weitreichende Ruptur dieser letzteren in unvollständiger Abheilung.

Unser Fall VIII kann als Beispiel dafür dienen, daß es trotz allerhöchstgradiger hämorrhagischer Infarzierung der Lunge klinisch durchaus nicht zu Hämoptöe zu kommen braucht.

Andererseits möchte ich auch vom klinisch-diagnostischen Standpunkt aus hervorheben, daß des öfteren recht langdauernde Lungenhämorrhagien ohne jeglichen Infarktcharakter (weder in den klinischen Symptomen, noch im objektiven physikalischen Befund) in Erscheinung traten, welche bis zu einem gewissen Grade sogar einen bedingten diagnostischen Wert beanspruchen können.

Aus meiner Praxis ist mir ein Fall erinnerlich, der eine Frau anfangs der 40er Jahre betraf, welche an eigentümlichem periodischen Bluthusten litt und deshalb bei den Angehörigen auch als phthisisch galt. Vollkommen negativer physikalischer Befund, trotz sorgfältigster Sputumuntersuchungen niemals Bazillen. Mäßige Hypertrophie des rechten Ventrikels, leichte Zyanose, Atmen vollkommen frei. Andeutung neurotischer Symptome. Von ihr und den Angehörigen wird die bestimmteste Angabe gemacht, daß sich die Lungenblutungen häufig zur Zeit der Periode einstellen, diese jedoch dann immer ausbliebe.

Tuberkulose war auszuschließen, ebenso Hämophilie. Ich faßte den Fall so auf, daß ich eine besonders leichte Vulnerabilität der Lungengefäße annahm (möglicherweise durch endarteriitisch-degenerative Prozesse bedingt) und ihn nach allem in das hypothetische und wohl sehr fragliche Gebiet der vikariierenden Menstrualblutungen einreihte.

Wenn nach obigem in außerordentlich seltenen Fällen eine abnorme Zerreißlichkeit der Gefäßwände vorkommen kann, so stellt doch dieser Modus ein verschwindend kleines Kontingent der Fälle von Lungenblutungen. Die Hauptursache für diese liegt in den Kapillaren der Stauungslunge.

Mit einer gewissen Reserve möchte ich nach allem das Verhalten bezüglich der Lungenblutungen derart resümieren, daß bei ausgesprochener Mitralstenose oder Erkrankungsfällen, die teils an diese, teils an kongenitale Vitia cordis erinnern, länger dauernde und öfters sich wiederholende Lungenblutungen ohne Infarktcharakter den Verdacht auf Endarteriitis (Sklerose) der Pulmonalis erwecken können.

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Wenn auch von keiner ausschlaggebenden Bedeutung, so doch immerhin bemerkenswert und differential-diagnostisch nicht ganz wertlos erscheint mir der Mangel von kolbigen Auftreibungen der Endphalangen, Trommelschlegelfingern[1]).

Wie Rühle (1877) hervorhebt, gehören die stärksten Grade der Trommelschlegelfinger (kolbige Auftreibung der dritten Phalanx, Krümmung des Nagels) besonders jenen angeborenen Herzfehlern an, welche mit erheblicher Zyanose verbunden sind.

Speziell im Hinblick auf letztere Tatsache erlangt das Fehlen dieser Bildung bei der Atherosclerosis pulmonalis trotz höchstentwickelter Zyanose eine gewisse differential-diagnostische Dignität.

Laache (1899) und Kidd (1904) heben bei ihren Beobachtungen ausdrücklich den Mangel dieses Symptomes hervor.

Es wird wohl des öfteren der auffälligen, oft tiefdunkelblauen bis blauschwarzen Verfärbung der Endphalangen, auch der Fingernägel gedacht, aber ausdrückliche Hervorhebung obigen klinischen Befundes traf ich nicht an. Nachdem die Trommelschlegelfingerbildung eine so ungemein auffällige und stets gewürdigte klinische Erscheinung darstellt, hätten die Beschreiber sicherlich ihrer gedacht. Auch in unseren Fällen bestand zwar mehrfach allerstärkste dunkelblaue Färbung der Endphalangen, speziell der Nägel, zu einer kolbigen Auftreibung derselben kam es jedoch nie.

Allerdings gibt es eine nicht unbedeutende Literatur über Fehlen von Zyanose und Trommelschlegelfinger bei angeborenen Vitia cordis, selbst bei sehr erheblichen Entwicklungsstörungen (worüber Vierordt, Nothnagels Spez. Path. eingehender berichtet). Auch wir verfügen über eine hier einschlägige Beobachtung:

Bei einem 25jährigen Manne E. G. konnte ich folgende klinische Diagnose stellen (auszugsweise): Vitium cordis congenit. Stenosis ostii pulmonalis. Hypertrophia ventriculi dextri. Defectus septi ventriculorum. Anaemia, Cyanosis. Insufficienta cordis.

Die Obduktion bestätigt die klinische Diagnose[2]). Der auf das Herz bezügliche Befund lautete:

Defekt im vorderen (membranösen) Septum der Herzventrikel mit teilweiser ringförmiger Verengerung des Konus der Pulmonalis.

Insuffizienz der Klappen der Pulmonalarterie bei ausgebreiteter verrukoser Endokarditis dieser, sowie der Aortenklappen und des oberhalb der Verengerung sehr erweiterten Konus der Pulmonalis.

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1) In letzterer Zeit fand das Thema eine übersichtliche Darstellung von E. Ebstein (Zur klinischen Geschichte und Bedeutung der Trommelschlegelfinger. Deutsches Arch. f. klin. Med. 1907, Bd. 89, S. 67), in welcher er schließlich dem Ausspruch Wests: „Clubbing is one of those phenomena with which we are all so familiar that we appear to know more about it than we really do" vollkommen beipflichtet.

2) s. Pommer, Bericht über 2 Fälle von Pulmonalstenose. Wiener klin. Wochenschr. 1904, Nr. 27, S. 762.

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Offenes Foramen ovale. Enge der Aorta im Isthmusgebiete, exzentrische Herzhypertrophie.

Der Fall ist dadurch ausgezeichnet, daß er zu den vorwiegend mit Anämie vergesellschafteten gehört (die Zyanose trat lange Zeit in den Hindergrund), und daß keine Spur einer Trommelschlegelfingerbildung statthatte (s. Vierordt, l. c., S. 89).

Unter den klinischen Symptomen der Pulmonalarteriensklerose nimmt die zumeist ganz besonders hochgradige **Zyanose** in ihrem eigenartigen Verhalten als Frühsymptom eine dominierende Stellung ein.

Bis zu einem gewissen Grade ist wohl ein bläuliches Kolorit bedingt durch die primären Ursachen oder indirekte Prozesse: Mitralfehler, vor allem Stenosen (violettbläuliche Facies mitralis), Perikardverwachsungen, Pleuraadhäsionen u. dgl.

Die Blausucht findet sich aber auch in hohem Maße entwickelt bei der reinen, genuinen Sklerose des Gefäßes.

Als diagnostisch zu verwertendes Moment kommt ihr jedoch nicht so sehr die zumeist ganz ungewöhnliche Intensität als der Umstand zu, daß sie schon zu einer Zeit, wo die anderen Erscheinungen wenig ausgebildet sind, im Vordergrund des klinischen Bildes steht und mit dem sonstigen Befund (am Herzen, Lunge, Allgemeinsymptome) und dem verhältnismäßig guten Kompensationszustand nicht recht in Einklang zu bringen ist. Es drückt sich dies in der oft lange Zeit persistierenden, höchst auffallenden Differenz aus zwischen der enormen Blausucht und dem Fehlen von anderweitigen Stauungen, Ödemen, Dyspnöe oder nur sehr schwachen und zeitlich späten Ausbildung letzterer.

Es verlohnt sich, diesen Verhältnissen etwas ausführlicher an der Hand der Literatur und unserer Eigenbeobachtungen nachzugehen.

Allgemein gehaltene Andeutungen lassen sich schon aus zerstreuten Bemerkungen der älteren Literatur herauslesen.

Kreysing (Die Krankheiten des Herzens. Berlin 1816. T. II, Abt. 2, S. 824) schließt bei der Unterscheidung der Blausucht aus angeborenen Fehlern des Herzens von der aus Fehlern der Lunge herrührenden (im Hinblick auf einen Fall von Green, Philosoph. transact., no. 454, IV., welcher in der Leiche einer 80jährigen Frau das eirunde Loch sehr weit offen fand, dabei selbe jedoch im Leben nicht blausüchtig gewesen), „daß zu diesem Bildungsfehler noch andere Bedingungen hinzukommen müssen, wenn Blausucht entstehen solle; und in der Tat scheinen diese nach den vorhandenen Fällen teils in einer krankhaften Veränderung der Lungenarterie, teils in anderen organischen Metamorphosen des Herzens zu bestehen."

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Durch ganz bedeutende zyanotische Verfärbung zeichneten sich u. a. aus die Fälle von Broca, Klinger (1855), de Buman (1858), Lancereaux (1861), Klob (1865), Tommasi Crudeli (1868), Rosapelly (1872), Voisin (1874), Sainton (1875), Sauné (1877), Bryant (1901), Rolleston (1902).

In den krankengeschichtlichen Notizen bei Kidd (1904) wird allerdings anfänglich nur eine leichte Zyanose erwähnt; daß der Fall jedoch für einen angeborenen Herzfehler gehalten wurde, deutet darauf hin, daß dieses Symptom im weiteren Verlaufe doch erheblicher hervorgetreten sein mußte.

Hohe Grade erreichte sie bei der Kranken Boinets und Poesys (1906). Dasselbe traf für beide Fälle Mönckebergs (1907) zu.

Auch der fettsüchtige Potator Kitamuras (1908) zeigte starke Zyanose, wenigstens dem Sektionsbefunde nach.

Romberg (Deutsch. Arch. f. klin. Mediz. 1891, Bd. 48, S. 197) führt unter den klinischen Eigentümlichkeiten bei den Allgemeinerscheinungen das völlige Fehlen von Ödemen bei der außerordentlich hochgradigen Zyanose als besonders merkwürdig an.

Diese hochgradige Zyanose läßt sich nach ihm nicht mit Sicherheit erklären.

Nachdem wir bei verschiedenen Gelegenheiten auf seinen Erklärungsversuch zurückkommen müssen, möge derselbe im folgenden dargelegt werden:

Die Mehrzahl der feineren Verzweigungen der Pulmonalis war verengt, wie die Verkleinerung des Lungenvenenkalibers beweist[1]). Die abnormen, dadurch gesetzten Widerstände wurden aber dennoch fast vollständig überwunden. „Das stärker arbeitende und kräftiger sich kontrahierende Herz treibt" — um mit den Worten Cohnheims zu reden — „durch die verkleinerte Blutbahn ebensoviel Blut und überwindet . . . sich entgegenstellende Widerstände so vollständig, daß . . . die normale Menge Blut in der Zeiteinheit die Lunge passiert." Es ist klar, daß dann die Stromgeschwindigkeit sich entsprechend der Verengerung der Lungengefäße steigern wird. Die Zeit, während welcher das Blut mit den Alveolarepithelien in Berührung ist, wird eine gegen die Norm verkürzte sein. Die Oxydation des Hämoglobins, die Abgabe der Kohlensäure wird eine mangelhaftere und das Blut wird mehr oder minder venös in die Lungenvenen gelangen." Für diese seine Hypothese, daß die Strombeschleunigung ausreicht, um die ungenügende Lüftung des Blutes zu erklären, führt Romberg auch die Ansicht Bohrs ins Feld, der zufolge die Lüftung des Blutes nicht ausschließlich auf der Diffusion der Gase beruhe, sondern durch „eine Art von Sekretionsprozeß, analog den Ausscheidungsprozessen in den Drüsen" vor sich gehe. Indem nun Romberg diese Verhältnisse, wie sie z. B. an den Speicheldrüsen bestehen, auf die Lunge überträgt, sagt er: Nehmen wir die normalen Stromverhältnisse als die besten für einen hinreichenden Gaswechsel an, so folgt daraus für unsern Fall eine schwere Schädigung desselben; denn das sezernierende Lungenparenchym erhielt zwar annähernd die

1) Vgl. oben S. 436.

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normale Menge Blut, aber es war in jedem Augenblick mit einer wesentlich kleineren Blutmenge als unter normalen Verhältnissen in Berührung.

Bei diesem Erklärungsversuch für die hochgradige Zyanose glaubt Romberg die von Cohnheim angezogene Verdickung der Kapillarwandungen umgehen zu können.

Nach Aust (Münchener medizin. Wochenschr. 1892, S. 689) ist die Ätiologie seines Falles von primärer Sklerose der Pulmonalis in völliges Dunkel gehüllt. Als schädigende Momente kämen in Betracht: das langandauernde angestrengte Einatmen heißer Dämpfe[1]) und die Obliteration der einen Pleurahöhle.

„Das am stärksten hervortretende Symptom der Erkrankung bildete die allmählich bis zu enormer Hochgradigkeit zunehmende Zyanose und Dyspnöe bei völligem Mangel von Ödemen, ein Verhalten, das bei dem relativ jugendlichen Alter des Patienten zunächst die Annahme eines kongenitalen Herzdefektes mit abnormer Mischung von venösem mit arteriellem Blute wohl rechtfertigte."

Neusser (Über Zyanose. Wiener klin. Wochenschr. 1893, Nr. 32, S. 591[2]) berichtet in aller Kürze über den Prozeß wie folgt: „Bei Sklerose der Lungenarterie, auch ohne aneurysmatische Erweiterung derselben, bestand in den von Klob und Romberg veröffentlichten Fällen intra vitam hochgradige Zyanose mit Hypertrophie des rechten Ventrikels, systolischen Geräuschen an der Herzspitze, akzentuiertem 2. Pulmonalton und kleinen, weichen Radialpulsen. Das Merkwürdige dieser Fälle liegt in der außerordentlich hochgradigen Zyanose bei völligem Fehlen von Ödem bis zum Tode."

Laache (Norsk. Magaz. f. Laegevid. 1899, Nr. 1) betont ausdrücklich, daß die Kranke ganz das Bild des Morbus caeruleus bot.

Er geht die in der Literatur mitgeteilten Fälle von Arteriosklerose der Pulmonalis durch und findet bei den meisten keine auffallende Zyanose erwähnt. Nur der von Romberg gleiche dem mitgeteilten klinisch ganz auffallend.

Nach Laache fehlt für den Grad der Zyanose noch eine ausreichende Erklärung.

Durch Stauung allein ist sie nicht bedingt, zumal ja in seinem Falle gar keine Stenose, sondern nur Erweiterung vorlag. Auch er weist auf die Möglichkeit hin, daß die Theorie Bohrs richtig sei, nach der die Respiration nicht eine Diffusion, sondern eine Sekretion darstelle, so daß es sich in diesen Fällen um eine Behinderung der Ausscheidung der CO_2 handle.

Die hochgradige Zyanose in seinen beiden Fällen ist Mönckeberg (Deutsche mediz. Wochenschr. 1907, Nr. 31, S. 1243) geneigt, mit Romberg auf die mangelhafte Sauerstoffaufnahme des Blutes in den Lungen infolge der erhöhten Stromgeschwindigkeit zurückzuführen.

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1) Bezüglich des obigen schädlichen Momentes möchte ich daran erinnern, daß nach François-Franck (Trav. du labor. de Marey IV. 1880. 379) beim Einblasen stark reizender Gase in die Lunge eine Blutdrucksenkung resultiert. Dabei ist noch die schädigende Wirkung hoher Temperaturen mit zu berücksichtigen. Ungewöhnlich starke und sehr wechselnde thermische Einflüsse beschuldigt Öhler (Deutsches Arch. f. klin. Med. 1908, Bd. 92) als ätiologisches Moment für das Entstehen von Dyskinesia intermittens brachiorum (s. u.).

2) Vollkommen gleichlautend spricht sich Neusser in neuester Zeit aus (Ausgewählte Kapitel der klinischen Symptomat. und Diagnostik. 3. Heft. Dyspnöe und Zyanose. Braumüller 1907. S. 157).

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Schwartz vergleicht das Krankheitsbild, welches bei Pulmonalsklerose zustande kommen kann, mit dem Morbus caeruleus.

Wenn auch Kitamura (l. c.) bei seinem Kranken, einem 33j. Kellner, der täglich 30 Glas Bier und reichliche Mengen von Wein und Schnaps trank, keine näheren klinischen Symptome angab (der Mann wurde mit den Zeichen hochgradiger Herzschwäche in das Krankenhaus eingeliefert, wo er kurz darauf starb), so verdienen doch zwei Sektionsbefunde besonders hervorgehoben zu werden, da sie einen Rückschluß auf die im Leben bestandenen Verhältnisse gestatten: die starke Zyanose des Gesichtes und das Fehlen jeglicher Ödeme.

Falls Bohrs (Über die Gasspannungen im lebenden arteriellen Blute. Zentralbl. f. Physiologie 1887, 1. Okt., Nr. 14, S. 293) Ansichten, die er auch wiederholt in späteren Publikationen erläuterte und auf die sich Romberg und Laache berufen, zu Recht bestehen, würde dadurch allerdings eine Reihe von hier vorhandenen Symptomen, namentlich die beträchtliche Zyanose bei geringer Dyspnöe befriedigend erklärt werden können.

Barcroft (Zur Lehre vom Blutgaswechsel in den verschiedenen Organen. Ergebnisse der Physiol. 1908, VII, S. 720) wendet, als die ernsteste Kritik von Bohrs und Henriques Resultaten, ein, daß sie den Stoffwechsel der Lunge selbst ignorieren. Die Lunge ist nicht nur eine sekretorische Drüse, sondern auch eine tätige solche. Die Energie, dank welcher sie den Sauerstoff von einem Ort niedrigerer Spannung zu einem Ort mit höherer Spannung befördert, muß auf Kosten des Gasstoffwechsels des Lungengewebes geschehen.

Als ungünstiger Faktor bei der supponierten Strombeschleunigung stellt sich die Rauhigkeit der Gefäßintima der Pulmonalis und deren Elastizitätsverringerung dar, was eine um so mehr erhöhte Inanspruchnahme des rechten Ventrikels involviert.

Als weiterer Umstand wäre in Betracht zu ziehen, daß die befallenen Individuen recht oft in jugendlichem und mittlerem Alter und beiläufig zur Hälfte weiblichen Geschlechtes waren[1]).

Dies glaubte ich deswegen hervorheben zu sollen, weil ja bekanntlich der Dicke der Haut und der Blutschichte ein wesentlicher Anteil an der bläulichen Färbung zugeschrieben wird.

Diese Annahme findet sich zuerst in ausführlicher Weise begründet von Peacock, Cases of malformation of the heart. London, R. Barrett, 1864.

Derselbe, On malformations of the human heart etc. Churchill, 1858, II. Ed. 1866.

In diesen Arbeiten wird das Thema in extenso besprochen. Das Ergebnis fand auch Aufnahme bei Fuller (Krankheiten des Herzens

1) Unseren Untersuchungen zufolge gestaltete sich das Verhältnis
M : W = 27 : 25.

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und der großen Gefäße, übersetzt von Schultzen. Berlin 1864. XI. Kap., S. 185).

Eingehende Würdigung erfuhr der Gegenstand durch Vierordt (Über Zyanose. Verh. des Kongr. f. inn. Mediz. XVIII, 1900, S. 307). Nach ihm wird die blaue Farbe der Haut u. a. bestimmt durch die Dicke der Blutschichte und der überlagernden Hautschichte, welch beide Faktoren wieder in verschiedenem Maße zur Gesamtwirkung beitragen können.

S. auch Vierordt, Angeb. Herzkrankh., S. 29.

Wiederholt findet sich in der Literatur und bei unseren Fällen Ausdehnung der kleinsten Hautgefäßchen und Überfüllung derselben verzeichnet.

Wir müssen uns hier mit dem bloßen Hinweis auf verschiedene andere Momente, die einen Einfluß für das Auftreten und den Grad der Zyanose erlangen können, begnügen:

Vermehrung der roten Blutkörperchen[1]) in der Volumseinheit, stärkere Konzentrierung und Eindickung des Blutes.

Literatur u. a. bei Grawitz (Klin. Pathologie des Blutes, S. 609).

In zwei Fällen unserer Eigenbeobachtungen von zyanotischen Mitralstenotikern mit Pulmonalsklerose bestanden ebenfalls derartige Verhältnisse (s. Krankengeschichten).

Es bestehen zweifellos vielerlei Analogien mit der Blausucht bei angeborenen Herzfehlern. Vgl. Neusser (l. c., Kap. Dyspnöe).

Huber (Charité-Annalen 1905, XXIX, S. 18) führt die Ursache der Blausucht bei angeborenen Herzfehlern auf eine Reihe von Ursachen zurück: Vermischung des arteriellen und venösen Blutes und Vermehrung des Hämoglobingehaltes und der roten Blutkörperchen bei ungefähr normalem, absolutem Sauerstoffgehalt. Dazu kommt noch die Stauung des dickflüssigen Blutes, die die kleinen Gefäße erweitert und so die dunkle Farbe mehr hervortreten läßt.

Nach Buhl (Beitrag zur pathologischen Anatomie der Herzkrankheiten. Zeitschr. f. Biologie 1880, Bd. XVI, S. 215) gebühre bei kongenitalen Herzfehlern, wenigstens für das Ostium pulmonale und das übrige rechte Herz, der mehr oder minder spät auftretenden, aber immer mehr zunehmenden, nicht entzündlichen Endarteriitis obliterans oder auch frischen endokarditischen Prozesses ein wesentlicher Anteil bei der Spätzyanose.

Bei den Mitralfehlern, daher auch bei der Stenose, ist die Zyanose im Grunde genommen eine Respirationszyanose.

Die eigenartige, periodische Steigerung[2]) und die direkten Anfälle

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1) Vereinzelt steht die Annahme Kitamuras da (s. o.). Derselbe plädiert für eine echte Plethora, eine absolute Vermehrung der Blutmenge, bei reichlichem Biergenuß als Ursache für die Pulmonalsklerose mit Herzhypertrophie, wobei sich die Erschwerung der Zirkulation besonders im Lungenkreislauf geltend gemacht habe.

2) Natürlich sind hiebei Schwächezustände des rechten Ventrikels nicht ge-

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machen es durch die Art und Weise des Eintretens und der Begleiterscheinungen bei vorliegender Affektion sehr wahrscheinlich, daß dabei reflektorische vasomotorische Einflüsse vorhanden sein müssen. Hierfür sprechen das Bestehen von Schweißausbrüchen, Frösteln, Parästhesien, lokale Ausdehnung feinster Hautgefäßchen. Zweimal kam es sogar zur Bildung von pemphigusartigen Zuständen.

Es hat förmlich den Anschein, daß diese periodischen Steigerungen der Zyanose bis zu einem gewissen Grad als regulatorische Hilfsaktion zur zweckmäßigen Blutverteilung zum Teile eine kompensierende Bedeutung für die Entlastung des Lungenkreislaufs zuzuerkennen sei [1]).

Das krisenhafte Auftreten auf gewisse äußere Veranlassungen, psychische Reize hin und das Gebundensein an die Schmerzattacken legt den Gedanken an einen reflektorischen Charakter sehr nahe.

Um Wiederholungen zu vermeiden, muß darauf verwiesen werden, daß manches von dem bisherigen Besprochenen und das Nächstfolgende auch für die Dyspragia (s. u.) Geltung hat.

Wenn auch außerordentlich spärlich, liegen doch immerhin Angaben vor, welche die Möglichkeit des Auftretens von Zyanose durch Vasomotoreneinfluß unter gewissen Umständen dartun.

Tordéus (De la cyanose. Journ. de médec. chir. et pharm. Bruxelles 1889, LXXXVIII, p. 38. Derselbe, Un cas de cyanose névropathique. Ibid. 1890, XC, p. 545) bringt eine Reihe von vorübergehenden, neuropathischen Zyanosefällen, u. a. bei einem 7monatlichen Kind während des Zahnausbruches.

Marseille gedenkt einer Beobachtung Biards, nach der eine Frau während menstrueller Störungen Zyanose bekam.

Bibliographie bei Grancher (Cyanose. Dict. encycl. des scienc. méd. 1883. I. S. XXIV. p. 501).

Auch an das Auftreten lokaler Zyanose (u. a. Passow) sei erinnert.

Rosenbach (Ein Fall von halbseitiger, im Anschluß an starke Körperbewegungen auftretender Zyanose des Gesichts. Ztrbl. f. Nervenheilk. 1886, S. 231) sah bei einem 9jährigen Knaben nach Körperbewegungen die ganze rechte Gesichtshälfte, inkl. Ohren exquisit blau gefärbt erscheinen (Temperaturerhöhung). Die linke war auffallend blaß und kühl, bei körperlicher Ruhe bekamen beide Hälften wieder

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meint, solche würden sich wohl im klinischen Gehaben kenntlich machen. Ebensowenig bezieht sich das obige auf enorme Steigerungen bis zu allerintensivsten Graden durch dazutretende Pulmonalthrombosen.

1) Das Gebiet der Regulierungsvorgänge durch Vasomotorenwirkung infolge von Reflexvorgängen ist noch dunkel. Nach François-Franck (Arch. de Physiol. 1896, p. 193) können außer pressorischen Reflexen auf den großen Kreislauf auch solche auf den kleinen ausgelöst werden. Die dadurch bedingte Abnahme des Blutzuflusses zum linken Herzen soll regulatorisch den gesteigerten Blutdruck im großen Kreislauf herabsetzen. Nach Krehl ist es sehr wohl möglich, daß die außerordentliche Ausbildung der Hautgefäße am Menschen denselben einen ganz anderen Einfluß auf den allgemeinen Blutdruck zuwendet, als das am Tiere der Fall ist.

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gleiches Aussehen. Bei fortgesetzter Bewegung ging die blaue Färbung in eine blaurote über (die Seite wird glühend heiß und endlich bricht Schweiß aus). Keine Sympathikus- oder Herzanomalien nachweisbar. Nach Rosenbach ist die veränderte Blutzirkulation das Primäre, die Schweißsekretion das Sekundäre. Er vermutet eher das Ausbleiben des Schweißes auf der anderen Seite als das Pathologische. Die Ursache erblickt er in einer lokalen Relaxation der Gefäßwände auf einer individuellen Disposition, nicht auf Lähmung oder Reizung von Vasomotoren beruhend.

Ein allgemeingültiges Verhalten bezüglich der **Dyspnöe** festzustellen geht nicht an, da sich je dieselbe nach den ursprünglichen Affektionen, dem jeweiligen Zustand des Herzens, den möglichen sonstigen Komplikationen richtet.

Am auffälligsten war der wiederholt erhobene Befund ganz beträchtlicher Differenz zwischen den geringen Graden derselben, auch anderer Stauungserscheinungen und der ungemein intensiven Zyanose.

Weiterhin, daß die Schweratmigkeit gar nicht selten lange Zeit fehlte, sich nur bei Körperanstrengungen einstellte und, wie aus manchen krankengeschichtlichen Notizen ersichtlich, mitunter Vorliebe für krisenhaftes, anfallsweises Auftreten zeigte.

In Übereinstimmung mit dem gewöhnlichen Befund bei den ursprünglichen Störungen zeigte sich bei unseren Fällen überhaupt nur eine leichte Neigung zu Verlängerung der Inspiration[1]).

Die Paroxysmen, wie sie während der Dyspragia intermittens pulmonalis statthatten, stellten sozusagen nur eine exzessive Steigerung der Inspirationsphasen, die enorm vertieft und verlängert wurden, dar.

An dyspnoischen Mitralstenotikern zeigte Kraus, daß sich kein vorwiegend in- oder exspiratorischer Charakter oder Zeichen erschwerten Einströmens der Luft in die Lungen bemerklich machten. Es handelte sich nur um eine einfache Beschleunigung und Vertiefung der Respiration (Hyperpnöe).

Bei kardialem Asthma macht sich eine Neigung zur Verflachung der Kurven bemerkbar; bei dieser verflachten Atmung ist der inspiratorische Schenkel durch seinen Abfall, den geringen Winkel, den er mit der Abszisse bildet, und seine geringe Höhendimension charakterisiert. Die Respirationspause ist kaum angedeutet. Die Frequenz wechselt sehr.

Die geschilderten Anfälle bei Atherosclerosis pulmonalis unterscheiden sich demnach ebenso vom gewöhnlichen Asthma cardiale als dem bei Koronarsklerose auftretenden (s. Hofbauer, Semiologie und Differentialdiagnostik der verschiedenen Arten von Kurzatmigkeit auf Grund der Atemkurve. G. Fischer, Jena 1904).

So wie bei allen übrigen Erscheinungen und Symptomenkomplexen

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1) Eine solche findet sich unter den Herzkrankheiten speziell beim Asthma cardiale, der Perikarditis und bestimmten Klappenfehlern unter gewissen Umständen.

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kommt es ganz besonders auch bei der Form der Dyspnöe darauf an, wie lange und oft man einen derartigen Kranken beobachten konnte und vor allem, in welchem Stadium man ihn kennen lernte. In den Endstadien verwischen sich die markanten Erscheinungen und es steht im Mittelpunkt aller die schwerste Herzinsuffizienz mit allen ihren Konsequenzen.

An dieser Stelle möchte ich auch in Kürze darauf hinweisen, daß die differenten Ansichten über das Verhältnis des Prozesses zum Lungenemphysem[1]) sich vielleicht in der Weise zurechtlegen ließen, daß es zwar als unterstützendes Moment in manchen Fällen, ähnlich wie Pleura- und Perikardaffektionen (vorzüglich ausgedehnte Adhäsionen), wirken kann, daß aber allen diesen Alterationen sicherlich kein entscheidender Einfluß zusteht, wenn man bedenkt, wie enorm häufig Lungenemphysem gerade auch mit den genannten Störungen vergesellschaftet ist, ohne daß man auch nur leise Ansätze zu solchen Gefäßveränderungen nachweisen könnte.

Früher stellte man sich die Beeinträchtigung der Atmung infolge Druckerhöhung in den Lungengefäßen so vor, daß die stark gefüllten, unter hohem Druck stehenden Lungenkapillaren sich in das Lumen der Alveolarräume vorbuchten.

Die von Basch experimentell konstatierte „Lungenstarrheit" bei Stromhindernissen in den Lungengefäßen stimmt mit manchen klinischen und pathologisch-anatomischen Erfahrungen nicht überein.

An den einfachen Stauungslungen des Menschen zeigt sich weder Schwellung noch Starrheit[2]).

In jüngster Zeit hat H. E. Hering nachgewiesen, daß beim ruhig liegenden, spontan atmenden Tier (Kaninchen) nach relativer Insuffizienz des linken Herzens (Kompression des freigelegten Arcus aortae ohne Thoraxeröffnung) erhebliche Lungenhyperämie ohne objektiv wahrnehmbare Dyspnöe vorhanden sein kann.

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1) Sauné macht auf das häufige Zusammentreffen mit Lungenemphysem aufmerksam (7mal unter 12 Fällen), dem Laache beistimmen möchte, während Runeberg (II. Nordischer Kongreß für Innere Medizin. Christiania, Aug. 1898) im Gegenteil bemerkt, daß bei Lungensklerose und Emphysem trotz des hohen Druckes Sklerose der Lungenarterien selten ist.

2) Gegen die Versuche Baschs und seiner Schüler macht Gerhardt (Arch. f. exp. Path. 1901, Bd. 25) den Einwand, daß sie zumeist mit außerordentlich großen Druckschwankungen arbeiten, wie sie in der menschlichen Pathologie kaum vorkommen. Daß die Blutstauung zu einer Erweiterung der Alveolen und Vergrößerung des Lungenluftraums führe, gibt er zu, dieselbe sei jedoch so gering, daß sie eine für die Lungenlüftung kaum in Betracht kommende Größe darstelle. Die dadurch erzeugte Starrheit ist dabei zu unbedeutend, um eine merkliche Erschwerung des Luftzutrittes zu verursachen.

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Die Gefäßstauung in der Lunge scheint mithin auf die Atmung nicht so sehr eine unmittelbare als eine mittelbare Einwirkung zu haben. Infolge der schlechteren Ernährung der spezifischen Gewebselemente (an der Bronchialschleimhaut und den Alveolarepithelien) sinkt die Widerstandsfähigkeit gegenüber mannigfachen Reizen und Schädlichkeiten, speziell gegen Infektionserreger (sekundäre Bronchitis usw.).

Unter Zugrundelegung des Tigerstedtschen Schemas ist bei der natürlichen Atmung bei der Inspiration die Ansaugung des Blutes nach dem rechten Herzen zunehmend, die Diastole erleichtert, die Weite der Lungengefäße zunehmend, die Strömung des Blutes in den Lungengefäßen erleichtert (Blutzufuhr nach dem linken Herzen, erst Abnahme, dann Zunahme). Die Gefäße des großen Kreislaufs werden weiter.

Die aktive Inspirationsstellung durch Anstemmen der Arme und langsames tiefes Einatmen, wie es im Anfalle bestand, hat demnach einen kompensatorischen Charakter gegen den supponierten Spasmus der Lungengefäße[1]). Infolge dieser Stellung sollte dem entgegengewirkt werden, was in erster Linie dadurch erzielt wird, daß sich durch die Inspiration die Lungengefäße erweitern und sich stärker mit Blut füllen, die Strömungsgeschwindigkeit in ihnen zunimmt.

Der Blutdruck in der Arteria pulmonalis ist abhängig von der durch die rechte Herzkammer ausgeworfenen Blutmenge und von dem Widerstand in der Gefäßbahn.

Die zur Verfügung stehende Blutmenge wird bedingt durch die aus dem Venensystem dem Herzen zuströmende, die ihrerseits auf den Vorgängen im Aortensystem beruht, zum Teil aber auch auf den bei den verschiedenen Respirationsphasen wechselnden Zuflußmengen zu der Brusthöhle.

Wie verhält es sich nun aber mit dem Druck in der Pulmonalarterie während der Inspiration?

In der Norm findet während dieser Phase eine Ansaugung in den intrathorakalen Venen statt.

Man würde nun erwarten, daß durch die vermehrte Blutzufuhr während der Inspiration der Druck in der Lungenarterie steigen müsse. Tatsächlich sinkt aber unter normalen Verhältnissen, wie zuerst Talma am Hunde, Tigerstedt am Kaninchen nachwies, der Druck während dieser Phase im rechten Ventrikel, um während der Exspiration zu steigen. Die Erklärung liegt darin, daß der erniedrigte intrathorakale Druck auch auf den Inhalt des relativ schwachwandigen rechten Ventrikels wirkt und so ein Herabgehen des in ihm herrschenden Druckes bewirkt.

Aber auch dieses für die Norm geltende minimale Sinken des Blutdruckes kommt für unsere pathologischen Befunde nicht in Betracht. Da ja doch der rechte Ventrikel ungemein hypertrophisch, in seiner Wand (ähnlich dem linken) mächtig verdickt ist, wodurch obiges Moment wegfällt.

Es kommt demnach, allem obigen zufolge, die Inspiration dem Lungenkreislauf zugute; Rubow (Untersuchungen über die Atmung bei Herzkrankheiten. Ein Beitrag zum Studium der Pathologie des kleinen Kreislaufes. Deutsches Arch. f. klin. Mediz. 1908, Bd. 92, S. 255) drückt ebenfalls die Vermutung aus, daß eine tiefe Respiration mit verlängertem Inspirationstadium die für die Lungenzirkulation günstigste Respirationsart sein dürfte.

1) s. Dyspragia intermittens pulmonalis (Angina hypercyanotica).

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Wiederholt wurde festgestellt, daß bei vielen Leuten während der Inspiration eine Beschleunigung der Pulsfrequenz eintritt.

Sicherlich ist die Lungenblähung und das Emphysem in manchen Fällen eine direkte Folge der Pulmonalatherose, wobei ich bei längerer Beobachtungszeit an den klinischen Kranken den Eindruck gewann, daß nach der Art und Weise des Atmens, dem Aufstemmen, die sich ausbildende Lungenblähung als sekundärer Zustand, ja mitunter als eine Art Kompensationseinrichtung gelten kann.

In dieser Hinsicht hat die Theorie Bohrs[1]), dem sich Rubow (Über die Atmung bei Herzkrankheiten. Klinische Untersuchungen aus dem Kgl. Frederiks Hospital, Nordisk Forlag, Kopenhagen 1907) anschließt, sehr viel Bestechendes, nach der die Dyspnöe lediglich auf der Anstrengung beruht, die Lungen stark ausgedehnt zu erhalten, wodurch die Lungenkapillaren gestreckt und deshalb leichter passabel werden, mithin auch durch Verringerung des Widerstandes im Lungenkreislauf die Arbeit für das Herz erleichtert werde.

In der Tat dürfte dies bei kaum einer anderen Störung mehr zutreffen und seine Berechtigung haben wie bei der Atherosclerosis pulmonalis, wenigstens unseren Erfahrungen zufolge.

In den vorgeschritteneren Stadien muß zur Erzielung dieser kompensatorischen Blähung um so größere Anstrengung und Kraft aufgebracht werden, als doch der Wegfall oder zum mindestens der hochgradige Verlust der Gefäßelastizität und die eintretende Starre der kleineren und kleinsten Gefäße in die Wagschale fällt.

Es ist wohl zweifellos, daß die Affektion jahrelang latent bleiben kann, was auch schon Sauné (1877) hervorhebt. Nach ihm läßt die Wiederkehr von Dyspnöeanfällen, welche sich namentlich erst in einer vorgerückten Periode der Krankheit zeigen, kein besonderes Zeichen bei der Auskultation erkennen.

Dies erkläre sich aus der Möglichkeit des Einströmens der Luft und daß die teilweise Asphyxie nur dem Mangel an Blut zuzuschreiben ist.

Romberg (l. c. S. 206) führt die Dyspnöe auf den Sauerstoffmangel des Blutes zurück. „Dieselbe war im Vergleich zu der enormen Zyanose recht unbedeutend.“[2])

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1) Bohr (Die funktionellen Änderungen in der Mittellage und Vitalkapazität der Lungen. Ztschr. f. klin. Med. Jan. 1907, Bd. 88) betrachtet das akute Lungenemphysem nicht als eine Schädigung der Lunge, sondern als einen kompensatorischen, zweckmäßigen Vorgang, das chronische nicht als primären Verlust der Lungenelastizität, sondern als zweckmäßige Anpassung an die Verödung der Lungenoberfläche.

2) Ihre Geringfügigkeit möchte er vielleicht — von der absoluten Ruhe des

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Das Ausbleiben des Dyspnöe selbst bei gesteigerter Muskelarbeit bei hochgradiger Zyanose, wie es bei „angeborenen Herzfehlern" (was hier für die prinzipielle Frage nicht releviert) möglich ist, sucht Neusser (Dyspnöe und Zyanose. 1907. S. 89) dadurch zu erklären, daß für die Sauerstoffversorgung der Gewebe, bzw. den Sauerstoffverbrauch andere regulatorische Kräfte aufkommen: Änderung der Strömungsgeschwindigkeit des Blutes durch kompensatorische Herzhypertrophie, Vermehrung des respirationsfähigen Hämoglobins, Polyzythämie, vermehrte Konzentration des Blutes durch Austritt von Plasma. Außerdem dürfte nach ihm aber auch durch Angewöhnung das Sauerstoffbedürfnis des Organismus herabgesetzt werden und das Atmungszentrum seine Erregbarkeit gegen die erhöhte Venosität des Blutes einbüßen.

Für die verhältnismäßige Geringfügigkeit der Atembeschwerden spricht wohl auch der häufig in den Krankengeschichten der Literatur und unserer Eigenbeobachtungen wiederkehrende Befund, daß die Kranken noch jahrelang arbeitsfähig waren, allerdings zumeist nur für leichtere Verrichtungen; außerdem verdient daran erinnert zu werden, daß bei verschiedenen Patienten, z. B. zwei unserer Kasuistik, durch die längste Zeit periodische Störungen medikamentös wieder leicht zu beheben waren.

Die von Neusser herangezogenen Momente finden nach jeder Hinsicht ihre Bestätigung, speziell in unserer Kasuistik. In ganz besonderem Maße imponierte ja die ungewöhnlich starke Hypertrophie des rechten Ventrikels als Ausdruck der Organkorrelation; in den darauf untersuchten Fällen ergaben sich ziemlich hohe Hämoglobinwerte, einige Male war eine ausgesprochene Polyzythämie nachweisbar.

Als wichtige Erklärungsgründe erscheinen mir die langsame Anwöhnung an das verminderte Sauerstoffbedürfnis, wofür wir ja so zahlreiche Analogien besitzen, ferner die Untererregbarkeit des Atmungszentrums.

Sicherlich darf auch nach unseren Erfahrungen dem langen Ausbleiben stärkerer sekundärer Bronchitis eine nicht zu unterschätzende Rolle zugeschrieben werden.

Dieser günstige Faktor muß vor allem der guten Kompensation durch die mächtige hypertrophische rechte Herzkammer zugeschrieben werden. Es hat aber nebstbei förmlich den Anschein, als wenn unter

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Kranken abgesehen — auf die außerordentlich langsame Zunahme des Sauerstoffmangels bis zu dem Grade, in welchem er den Kranken kennen lernte, zurückführen.

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besonderen Umständen die Pulmonalsklerose bis zu einem gewissen Grade als kompensierende Komponente dyspnoischer Störungen hierbei auftreten könne.

Alles in allen genommen scheinen doch die Umstände, welche zu dieser auffälligen Differenz zwischen schwerer Zyanose und verhältnismäßig geringen Atemstörungen führen, einer Erklärung zugänglich zu sein.

Diezugleich mit Steigerung der Zyanose anfallsweise auftretenden, mit Unruhe, Angst und Beklemmung einhergehenden **Herz-Lungenschmerzen** (von der Herzbasis in die Tiefe der Lunge strahlend) erinnern uns in ihrem ganzen Charakter und der Art und Weise des Auftretens unwillkürlich an mehrere andere fast analoge, ebenfalls auf arteriosklerotischer Basis beruhenden Krankheitserscheinungen und fordern zu einem Vergleich auf mit dem sog. „intermittierenden Hinken", der Claudication intermittente Charcots, der Dysbasia angiosclerotica Erbs bei Arteriosklerose der Extremitätenarterien[1]), oder der mit Anfällen von Schmerzen in der Nabelgegend, starkem Spannungsgefühl, Meteorismus und Obstipation einhergehenden „Dyspragia intermittens angiosclerotica intestinalis" Ortners[2]) bei Intestinalarteriensklerose, welche beide Symptomenkomplexe wiederum in Analogie mit der Angina pectoris gebracht werden.

Nach allem, was ich sah, dürften sich diese krisenartigen Zustände bei Arteriosclerosis pulmonalis ebenfalls in das Gebiet dieser intermittierenden Dyspragien einreihen lassen, wobei man sich stets vor Augen halten muß, daß die Analyse und Deutung der hier auftretenden Erscheinungen wegen der noch vielfach ungeklärten und strittigen physiologischen und pathologischen Vorgänge ungleich schwieriger als in allen übrigen Regionen sich gestaltet.

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1) Auch für die oberen Extremitäten wurde ein ähnlicher Symptomenkomplex festgestellt. Steuder, Ein Fall von Dyskinesia intermittens angiosclerotica brachii. St. Petersburger med. Wochenschr. 1907, Nr. 4. Oehler, Über einen bemerkenswerten Fall von Dyskinesia intermittens brachiorum. Deutsches Arch. f. klin. Med. 1908, Bd. 92, S. 154.

2) Ortner, Zur Klinik der Angiosklerose der Darmarterien (Dyspragia intermittens angiosclerotica intestinalis) nebst einem Beitrage zur Klinik des intermittierenden Hinkens und des Stokes-Adamschen Symptomenkomplexes. Volkmanns Samml. klin. Vortr. (Inn. Med.) 1903, Nr. 102.

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Vielleicht ließen sich diese fast krisenartigen Anfälle bezeichnen als „Dyspragia intermittens angiosclerotica pulmonalis".

Eben bei Vergleich der hier möglichen klinischen Symptomenkomplexe: Angina pectoris, Asthma cardiale und den Huchardschen Aortenbeschwerden wurde ich veranlaßt, diesen krisenhaften Zufällen eine gesonderte Stellung anzuweisen und ihre Deutung zu versuchen.

In unseren Fällen war auch klinisch zwischen den Zuständen von (Herz-Lungengefäß-)Schmerz und Herzangst zu unterscheiden. Wie sich letztere ganz besonders bei Aortenwurzelsklerose und den nächsten Gefäßgebieten finden kann, so auch hier bei der Atherosclerosis pulmonalis, wobei auch da gewissermaßen der krampfhafte Schmerz auf die allernächsten Gebiete übergreifen kann.

Ein Irradiieren des Schmerzes, wie bei der Stenokardie, war entschieden weniger ausgeprägt, wenigstens nicht in so weite Gebiete.

An einem sehr reichen klinischen Beobachtungsmateriale hatte ich Gelegenheit Stenokardieanfälle aller möglichen Stadien, Grade und Abstufungen genau zu verfolgen, ebenso Asthma cardiale-Anfälle.

Diese beiden Erscheinungskomplexe sind so oft und eingehend besprochen worden, daß hier kein Wort zu verlieren ist.

Mehrfach werden bei Aortensklerosen schmerzhafte Sensationen, ein gewisses Unbehagen am oberen Sternum, ein Druck längs des Sternums, ein Brennen daselbst angegeben, die Huchard einer Aortitis zuschreiben möchte[1]).

Die in Rede stehenden Attacken zeigen entschieden ein von den genannten Zuständen differentes Verhalten und dokumentieren sich in anfallsweise auftretendem, mit nervösen Störungen, Angst, Unruhe, Schwäche, Oppression einhergehenden Druck- und Schmerzgefühl in der Herzwurzelgegend am Manubrium sterni, vorzüglich gegen den linken 2. und 3. Interkostalraum zu (ein stärkeres seitliches Ausstrahlen fehlt, die Schmerzen werden immer in die Oberbrustgegend und in die Tiefe der Lunge zu lokalisiert), verbunden mit hochgradiger Zyanose, zu der das verhältnismäßig frei Atmen (fehlende oder nur ganz unbedeutende Dyspnöe) in auffälligem Kontrast steht. Das der Stenokardie eigene Vernichtungsgefühl fehlte.

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1) Schon Thoma hat auf Schmerzen bei Arteriosklerose aufmerksam gemacht. Stengel (Nervous manifestations of arteriosclerosis. Amer. journ. of the med. scienc. 1908. Febr.) bringt die Arteriosklerose mit verschiedenen peripheren, nervösen Symptomen in Zusammenhang; so führt er ebenfalls plötzlich auftretende Schmerzen in der Brust fast ähnlich wie bei Stenokardie, zumeist aber mehr diffus auf Atheromatose der Aorta thoracica zurück.

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Diese kardiopulmonäre Angina tritt erst bei vollentwickelter Krankheit, auf dem Höhepunkt und späterhin in Erscheinung, was auch mit den anatomischen Veränderungen in Einklang zu bringen wäre. Die Gefäßintima wird als empfindungslos angegeben, es ist deshalb auch naheliegend, daß diese Beschwerden, Schmerzen und sonstigen Symptome erst den späteren Stadien eigen sind, wenn bereits vorgeschrittenere und in die Tiefe greifende Veränderungen vorliegen.

Die Hauptregion des lokalen spontanen Schmerzes war das Gebiet im linken Abschnitt des Manubrium sterni und in der 2. und 3. Rippenknorpelgegend. Mehrfach bestand auch hier besonders bei tiefer Perkussion in vorgebeugter Haltung Empfindlichkeit bis Druckschmerz. Sehr variabel in seiner Stärke und Ausdehnung war der reflektierte Schmerz bei Kneifen der Haut, Nadelstichen u. dgl., mitunter zeigte er eine etwas weitere Area, eine stärkere Ausbreitung wurde jedoch auch hier vermißt.

Ich möchte hier mit Krehl (l. c. p. 111) zu bedenken geben, ob es sich überhaupt bei Herzangst und Herzschmerzen im allgemeinen nicht vielleicht um ganz besondere Störungen handelt, die im Zentralnervensystem entstehen und vom Herzen ausgelöst werden. Dies würde sogar den Vorstellungen der ältesten Beobachter am meisten entsprechen.

Diese (sit venia verbo) Lungenarterien-Schmerzanfälle könnten entweder auf Spasmen der kleinsten Lungengefäße oder (wodurch die Analogie mit der Angina pectoris noch mehr gesteigert würde) auf solche der Vasa vasorum, in diesem Falle der Bronchialarterien zurückzuführen sein, wobei die Möglichkeit besteht, einerseits, daß die Schmerzen durch den Krampf in diesen selbst entstehen oder daß dieselben infolge akuter Ischämie der Pulmonarwandungen eintreten.

Wir müssen Romberg (Über Arteriosklerose. 21. Kongr. f. inn. Med. 1904) ganz beipflichten, wenn er auf die Paradoxheit hinweist, daß die sklerotischen Arterien, welche den gewöhnlichen Reizen nicht mehr in normaler Weise zu entsprechen vermögen, auf krankhafte nervöse Einflüsse hin mit krampfartiger Verengerung oder lähmungsartiger Erweiterung reagieren.

Die sklerotische Gefäßveränderung allein ist nicht imstande, eine befriedigende Erklärung für die Zustände der Angina pectoris, intermittierenden Hinkens usw. zu geben. Als funktionelles Moment wird die Abhängigkeit von vasomotorischen Nerveneinflüssen angenommen (Erb, Kaufmann und Pauli, Goldflam, Ortner, Breuer).

Nach Muskelanstrengungen und ähnlichem wird die Gefäßwand stärker gespannt, die Gefäßnerven reagieren mit einem reflektorischen

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Gefäßkrampf[1]), wodurch ischämische Muskel- und außerdem Gefäßschmerzen entstehen, auf welche besonders Weber und Nothnagel das Hauptgewicht legen.

Unter allen Gefäßregionen, in denen sich auf sklerotischer Basis beruhende Dyspragien ausbilden können, gestalten sich die Verhältnisse bei der Lungenschlagader, dem venöses Blut führenden funktionellen Gefäß, das in seinen Wandungen selbstverständlich durch arterielles Blut der Vasa vasorum ernährt wird, am eigenartigsten. Gerade letzteres Moment läßt die Vermutung aufkommen, daß vielleicht unter gewissen Umständen Alterationen im Chemismus eine Rolle bei dem in Rede stehenden Prozeß wenigstens indirekt spielen können.

Die ältesten Fälle der hier einschlägigen Kasuistik sind einer klinische Analyse kaum zugänglich, gleichwohl finden sich hier immer wieder Schmerzen in der Herzgegend, die verschieden geschildert werden, angegeben.

Im Falle Stöller glichen selbe vollständig der Angina pectoris; nachdem jedoch kein Befund bezüglich der Koronargefäße mitgeteilt wird, ist keine Entscheidung zu treffen[2]).

Huguiers Kranke, bei der sich eine vollkommene Verknöcherung der Pulmonalis fand, hatte an heftigen Schmerzen in der Herzgegend zu leiden.

Nach der Beschreibung muß Adlers Fall als primärer gelten. Die Schmerzen in der Brust wurden durch die finale Thrombose gesteigert.

Martineaus Kranke, bei der nur Oppression angegeben erscheint, zeigte Stamm und beide Äste des Gefäßes frei, nur in den feinsten Ästen Veränderungen. Oppression auf der Brust, lange vor Ödemen hatte der kranke Knabe von Conway Evans.

Heftige Schmerzanfälle in der Brust bestanden bei den Kranken Crookes (Mitralstenose, Aorta frei von jeglichem Atherom).

Rombergs Kranker hatte bei Beginn der Erscheinungen an Druck in der Magengegend, gegen Ende der Krankheit an starken, den Atem versetzenden Schmerzen in der linken Seite des Thorax, unter dem Rippenbogen, zu leiden, für die kein anatomisches Substrat gegeben erschien. Ein objektiver Grund für dieselben war nicht nachweisbar.

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1) Oppenheim (Intermittierendes Hinken und neuropathische Diathese. Deutsche Zeitschr. f. Nervenheilk. Bd. 17) ist der Hauptvertreter der Annahme einer auf nervöser Grundlage entstehenden spastischen Kontraktion in den Arterien.

2) Bei Durchsicht der krankengeschichtlichen Aufzeichnungen Stöllers hatte ich entschieden den Eindruck, daß es sich um Stenokardieanfälle handelte. (Typische in die Arme ausstrahlende Schmerzen vom Sternum ausgehend, dabei Blässe, Vernichtungsgefühl usw.)

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Die Respirationsfrequenz stieg auf das Doppelte. Der Gesichtsausdruck wurde ängstlich.

Ausdrückliche Anfälle von Präkordialangst notiert Aust bei seinem Kranken. „Dyspnöe und Zyanose mit starken Exazerbationen" ist ein weiterer Befund.

Laaches allerdings neuropathische Frau klagte häufig über sehr starke Beklemmungen.

Das 21jährige kranke Mädchen Kidds litt von Kindheit an an „Herzkrämpfen".

Bei Boinet und Poesy findet sich angegeben: Schweratmigkeit mit Exazerbationen, Beklemmungen, nervöse Anfälle mit Schwäche, Aufgeregtheit und Krämpfen.

Bevor im Falle Schwartz die Lungenblutungen auftraten, stellten sich plötzlich heftige Anfälle von Atemnot mit krampfartigen Schmerzen in der Brust ein.

Weit entfernt behaupten zu wollen, daß sich diese Zustände mit der von uns aufgestellten Dyspragia intermittens pulmonalis decken, begnügen wir uns auf das paroxysmusartige dieser Beschwerden, speziell von Schmerzen und Oppression in der Herzgegend in diesen krankengeschichtlichen Notizen hinzuweisen.

Wir konnten uns wiederholt überzeugen, daß die von uns oben geschilderten Schmerzanfälle vorzüglich durch körperliche Anstrengungen und psychische Affekte provoziert wurden. Als erstere muß man natürlich hier schon geringfügige Hantierungen, rasche Bewegungen, Drehen, Bücken u. dgl. bezeichnen.

Übrigens sei auch an dieser Stelle erinnert, daß gar nicht wenige Kranke trotz ihrer Beschwerden oft noch jahrelang wenigstens leichterer Arbeit nachgehen konnten.

Für die Beurteilung dieser Schmerzzustände releviert in erster Linie der Umstand, daß wir in der allerüberwiegendsten Mehrzahl Mitralstenotiker vor uns haben. Nun ist es eine lange bekannte Tatsache, daß weitaus am häufigsten Aortenfehlerkranke (wegen Übergreifen der Affektion auf die Koronarien und die Aorta selbst) tatsächlich an Schmerzen leiden, während Patienten mit Mitralfehlern von solchen vielmehr verschont bleiben.

Nothnagel (Schmerzhafte Empfindungen bei Herzerkrankungen. Zeitschr. f. klin. Med. 1891, Bd. 19, S. 209) notierte unter 483 Herzklappenfehlern bei 22 Fällen von Mitralstenose nur in 18% Herzschmerzen, bei 82% fehlten selbe.

Die Tatsache, daß gerade bei Aortaklappenfehlern die schmerzhaften Sensationen so sehr viel häufiger sind als bei anderen Klappenfehlern drängt zur Annahme, daß weniger der Herzmuskel als vielmehr das Gefäß hiebei beteiligt ist.

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Weitere Mitteilungen brachte derselbe Autor über Gefäßschmerz (Wiener klin. Wochenschr. 1893, Nr. 46 und 47). In der Diskussion (S. 854) weist Schrötter darauf hin, daß nicht allein Angiospasmus mit Schmerz vergesellschaftet sei, bei der angioparalytischen Form der Migräne fände sich kein geringerer Schmerz. Die Gefäßschmerzen fanden wiederholt ihre Interpretatoren z. B. Laache (Zur Lehre von den Schmerzen, sog. vaskulären Ursprunges), Ortner (l. c.) u. a.

Hare (The diagnosis and relief of cardial pain. Philadelphia polyclin. 1898, no. 50) gibt eine ausführliche Darstellung der Schmerzen bei Herzleiden.

Nach ihm ist ebenfalls der Schmerz nur äußerst selten bei Mitralstenose, wobei er hervorhebt, daß diese Störung überhaupt nur selten und nur schwer von anderen vielleicht gleichzeitigen Affektionen zu scheiden ist und weil der Schmerz sich meist entwickelt im vorgerückten Stadium, wenn die Herzaktion unregelmäßig wird und sich verschiedene andere Erscheinungen bemerkbar machen. Er vermutet, daß diese mitunter bei Stenose der Mitralis auftretenden, der Stenokardie ähnlichen Schmerzen von einer besonders starken Dilatation des Vorhofes herrühren.

Wenn nun einerseits bei Aortenfehlern mit Sklerose und Atheromatose des Gefäßes so ungemein häufig, bei Mitralstenose so selten Schmerzen, speziell Paroxysmen auftreten, so müssen wir unwillkürlich nach dem Grund forschen, der für das Auftreten bei letzteren verantwortlich zu machen ist. Derselbe ist meiner Überzeugung nach in erster Linie in der Atherosclerosis pulmonalis gelegen, wofür ich folgende Momente ins Treffen führe:

1. die Analogie mit den Aortenverhältnissen.

2. Falls Hare mit der Dilatation des Vorhofes recht hätte, müßten ja alle halbwegs stärkeren Stenosefälle Schmerzen zeigen, die bei den stärksten Vorhofsdilatationen am heftigsten auftreten würden.

Das ist aber, wie ich mich in einer größeren Reihe von Eigenfällen, denen der Literatur, speziell auch den mit Rekurrenslähmung vergesellschafteten, überzeugen konnte, durchaus nicht der Fall.

3. Liegt in den Angaben Hares über Auftreten erst in den vorgerückteren Stadien, dem stenokardieähnlichen Charakter und wegen der Beschuldigung des Vorhofes jedenfalls auch von ihm konstatierten basalen Lokalisation eine Bestätigung unserer Ansicht.

4. Wäre ein sehr wichtiger Beweis das Vorkommen derartiger basaler Schmerzen mit ihrem charakteristischen Gepräge in Fällen, die, wie wir oben dargelegt, ihre Entstehung Pulmonalvenenverengungen verdanken, bei denen naturgemäß die Vorhofsausdehnung vermißt wird.

5. Dürfte, was sehr in die Wagschale fällt, der obige Prozentsatz

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schmerzhafter Mitralstenosen mit den mit Atherosklerose der Lungengefäße behafteten im großen und ganzen übereinstimmen.

In Zukunft müßte demnach ganz besonders dem Zusammentreffen von Schmerzen, obigen auskultatorischen Erscheinungen und der Blausucht Rechnung getragen werden.

In Zusammenfassung des Symptomenkomplexes der Dyspragia intermittens angiosclerotica pulmonalis (oder vom rein symptomatologischen Standpunkt, der Hypercyanosis intermittens angiosclerotica pulmonalis dolorosa adyspnoica) können wir kurz rekapitulierend sagen: .

Es handelt sich um ein durch verschiedene Momente (rasche Körperbewegungen, psychische Emotionen, Dyspesie mit Obstipation und Meteorismus und ähnliches) ausgelöstes oder begünstigtes krisenhaftes Auftreten von Schmerzen in der Gegend der Herzbasis (gegen die Tiefe der Lunge zu ausstrahlend), mit Aufregung, Unruhe, beklemmendem Angstgefühl einhergehend, mit denen fast stets eine beträchliche Steigerung der Zyanose vergesellschaftet ist, ohne daß es zu besonderer Atemnot zu kommen braucht, wobei im Gegenteil zur intensiven Blausucht längere Zeit die relativ leichte Atmung auffällig kontrastiert.

Die Mannigfaltigkeit der für die ätiologischen Verhältnisse zu beschuldigenden Ursachen und die sonstige Variabilität der klinischen Erscheinungen, beides Folgen der so zahlreich möglichen Kombinationen sonstiger Störungen, erschweren naturgemäß eine allgemeingültige zutreffende Erklärung.

Wenn ich dennoch eine solche wage, so geschieht es mit dem Bewußtsein, in dieser durch den Zusammenfluß so verschiedenartiger Möglichkeiten schwierigen Materie, die von Haus aus leider noch allzuviel des Ungeklärten enthält, Theorien aufzustellen, die durch weitere klinische Beobachtungen und Experimente gestützt werden müssen.

In diesen vorzüglich für den Praktiker und die klinische Beobachtung bestimmten Umrissen des abzuhandelnden Gegenstandes muß ich mich darauf beschränken, hinzuweisen, daß die Forscher, so sehr ihre Ansichten in vielen Punkten divergieren, in der außerordentlichen Schwierigkeit gerade dieser Experimente übereinstimmen.

Und eben bei diesen sind, wie aus unseren klinischen Besprechungen unzweideutig zu folgern, eine große Reihe einfacher und weiterhin immer komplizierterer experimenteller Prüfungen notwendig, weil eben hier allzuviel zusammentrifft: Funktion zweier Organe, deren Wechselbeziehungen, Ernährung, Nervenbeeinflussung und Störungen mannigfaltigster Art.

Die Möglichkeit gleichzeitigen Bestehens verschiedener Prozesse und die Mannigfaltigkeit solcher Kombinationen verwischt den Typus der einzelnen Symptome und erschwert deren Analyse.

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So bestanden bei Fall I, 67jähriger Mann, Alkoholist mit Leberzirrhose, Arteriosklerose und Myodegeneratio cordis nach seinen und seiner Angehörigen Aussagen angeblich niemals typische Stenokardieanfälle; allerdings war es schwer, von dem Kranken etwas herauszubringen.

Während des Spitalaufenthaltes werden ein einziges Mal „rheumatische" Schmerzen im linken Arm angegeben.

Es bestanden drückende Schmerzen am Brustbein, Oppressionsgefühl. Die klinischen Symptome waren derart, daß eine rechte Einreihung unmöglich war; bei der Nekropsie: Hochgradige Sklerose der Brustaorta und Pulmonalis, ganz besonders starke Verkalkungen der Koronarien und Einengungen ihrer Mündungen. Myodegeneratio cordis.

An der Mitralklappe Verdickungen, außerdem Verkürzung der Sehnenfäden.

Vom klinischen Standpunkte aus müssen wir ein ungewöhnlich langes Latentbleiben nach beiden Richtungen hin annehmen mit spätem Eintreten wenig ausgesprochener Symptome von Angina pectoris und nicht charakteristischer für die Pulmonalarterienaffektion.

Die zweifellos bestehende, auf alkoholischer Basis beruhende psychische Minderwertigkeit erschwerte, wie erwähnt, die Analyse der Befunde und die nähere Detaillierung der Verhältnisse.

Im Falle IV, 39jährige Frau, mit eigentlich 5jähriger Beobachtungszeit, hatte die Frau immer wieder schwere Anfälle von Polyarthritis.

Es bestanden starke Zyanose mit Dyspnöe, schwere Stauungskatarrhe. Bei Zusammenhalt aller Befunde konnte der Verdacht auf vorliegende Komplikation ausgesprochen werden.

Der 34jährige Mann (Fall V), (mit wiederholtem Gelenksrheumatismus, Endo- und Perikarditis, Typhus) zeigte bei seiner Mitralstenose eine Reihe obiger charakteristischer Momente. Wegen der für diese Affektion wenigstens kurzen Beobachtungszeit wurde eine Diagnose quoad Pulmonalarterie in suspenso gelassen; ebenso bei dem nächsten Pat., 29jährigen Mann (Fall VI), mit Polyarthritis, in dessen Familie Herzleiden zu Hause waren.

Es bestand Aortensklerose und Insuffizienz der Semilunaren mit enorm starker, im Herzbefund selbst nicht motivierter Zyanose, Hypertrophie des rechten Ventrikels und Fortpflanzung des diastolischen Geräusches nach links. Ganz das gleiche gilt vom nächsten, mit ihm völlig kongruenten Fall VII, bei welchem nur eine einmonatliche Beobachtungszeit bis zum letalen Ausgang zu verzeichnen war.

Durch das gleichzeitige Bestehen einer Reihe der verschiedensten Komplikationen bei Fall VIII, eine 43jährige Frau betreffend (Anamnese:

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Blattern, Pneumonie) und zwar Mitralstenose, Myodegeneratio cordis, Anämie, Zyanose, Nephritis, Urämie, Bronchopneumonie (Synechien beider Lungen) wurde der Typus vollkommen verwischt, abgesehen von dem weit vorgeschrittenen Zustand und dem nur 12tägigen Spitalaufenthalt.

Im IX. Falle konnte ich auf Grund der früheren Erfahrungen und der schon früher wiederholten Untersuchungen und langen Beobachtungsmöglichkeit mit höchster Wahrscheinlichkeit, bei Fall X mit Sicherheit die Diagnose auf Atherosclerosis pulmonalis bei Mitralstenose stellen.

Bei der Kleinheit, schlechten Füllung des linken Ventrikels und der für die Behinderung der Blutströmung in diesem maßgebenden Verhältnisse ist es begreiflich, daß wir bei manchen Beobachtungen die Kombination von „Anämie mit Zyanose" antreffen. Auf Anfälle von Schwäche des linken Ventrikels sind dann die hierbei notierten Zufälle von Kopfschmerzen, Schwindel, Schwarzsehen, Übelkeiten u. dgl. zurückzuführen (u. a. Fall VIII und X).

Bei Berücksichtigung aller Möglichkeiten wird es uns nicht wundernehmen, daß öfters schon frühe schwere Zeichen von Herzschwäche, Stauungszuständen, Stauungskatarrhen usw. auftreten können.

Gerade wie Angina pectoris mit Dyskinesia intermittens vergesellschaftet sein kann, wie u. a. aus der Mitteilung Curschmanns (Über vasomotorische Krampfzustände bei echter Angina pectoris. Deutsche mediz. Wochenschr. 1906, Nr. 38) hervorgeht, so liegt die Kombination von Stenokardie mit Dyspragia intermittens pulmonalis nahe, im weiteren die mit wirklichem Asthma cardiale.

Bei der Angina pectoris überwiegen, wie bekannt,, die Männer (nach Gauthier z. B. 78,8%), als Mittelzahlen finden sich durchschnittlich 80% angegeben. Bei der Atherosclerosis pulmonalis partizipieren beide Geschlechter fast in gleicher Zahl (M : W = 27 : 25).

Gewissermaßen prämonitorische Zeichen gehen jedoch schon geraume Zeit voraus. So zeigten z. B. zwei von unseren Kranken durch viele Monate hindurch rasch eintretende Steigerung der Zyanose mit allgemeinem Unbehagen, Frösteln und kaltem Schweiß.

Vielleicht ließen sich diese initialen Zyanosekrisen mit der primären Sklerose der Vasa vasorum in Zusammenhang bringen und zwar dadurch ausgelösten Zirkulationsstörungen.

In Zukunft müßte der zeitlichen Entwicklung und Ausbildung der klinischen Erscheinungen noch näher Rechnung getragen werden.

Zu wiederholten Malen war der Einfluß psychischer Affekte und.

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äußerer Reize für das Auslösen oder Stärkerwerden auch solcher Zustände unverkennbar.

Es darf uns daher nicht wundernehmen, daß mitunter eine Arhythmie des Pulses während derselben eine Zunahme erfuhr.

Durch längere Zeit stellten sich die vollentwickelten Anfälle, in Analogie mit der Angina pectoris, erst bei rascheren Bewegungen, Bücken, Umdrehen, raschem Gehen, Aufheben von schweren Gegenständen, Einwirkung von Kältereizen (z. B. beim Verlassen des warmen Zimmers im Winter) ein.

Späterhin überfielen sie die Kranken beim bloßen Aufrichten im Bette, sogar bei ruhiger Lage[1]), und es mischten sich immer mehr schwere dyspnoische Zustände, manchmal direktes Asthma cardiale bei.

Je geringfügiger die auslösende Ursache, desto ungünstiger gestaltet sich im allgemeinen die Prognose.

Die klinischen Erscheinungen, die hierbei beobachtet werden können, stehen im Einklang mit dem von Heidenhain hervorgehobenen Verhalten der Blutverteilung auf sensible Reize; es erweitern sich nach ihm in der Regel die Hautgefäße auf reflektorische Reizung, während sich die inneren Gefäße verengern, wodurch ein gewisser Antagonismus zwischen oberflächlichen und tieferen Gefäßen statthat. Im allgemeinen scheinen bei drucksteigernden Reflexen die Gefäße der inneren Organe meist verengt zu werden, während zu gleicher Zeit die der Haut und Skelettmuskeln mehr oder weniger erweitert werden. Bei drucksenkenden Reflexen ist das Umgekehrte der Fall. Der Antagonismus zwischen Haut und inneren Organen ist jedoch kein absoluter.

Das bei der Zyanose Erwähnte findet seine natürliche Ergänzung in den Befunden, die den innigen Zusammenhang vasomotorischer Reizerscheinungen in den kleinen Hautgefäßen bei ähnlichen Symptomenkomplexen im Anfalle dartun:

Hagelstam, Über intermitt. Hinken als Symptom von Arteriosklerose. Deutsche Ztschr. f. Nervenheilk. 1901, XX, S. 65. Higier, ibid. 1901, Bd. 19.

Brissaud, Claudication intermittente douloureuse. Rev. neurol. 1899, no. 13.

Goldflam, Neurol. Zentralbl. 1901, S. 197.

Die weite Entfernung stünde mit der physiologischen Erfahrung in keinem Widerspruch. Es können Körperteile, welche vom Verbreitungsgebiet der zurzeit gereizten zentripetalen Nerven weit entfernt liegen, bei sensibler Reizung eine reflektorische Verengerung oder Erweiterung ihrer Gefäße zeigen.

U. a. erwähnen Dastre und Morat (Rech. exp. sur le syst. vaso-mot.) als Folge einer Ischiadikusreizung Gefäßerweiterung in den Teilen des Kopfes, welche vom Halssympathikus gefäßerweiternde Nerven erhalten.

Für die in Rede stehenden Verhältnisse ist es nun von ganz besonderem Interesse, daß die gleichen Forscher eben diese Nerven auch durch Reizung des N. laryngeus superior und des Lungenvagus in Tätigkeit versetzen konnten.

Auf dieses Verhalten beziehen sie dann auch die Gesichtsröte, welche Lungenkrankheiten zu begleiten pflegt[2]).

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1) Jedem Praktiker gilt mit Recht das Eintreten von Stenokardieanfällen schon bei vollkommener Ruhe, namentlich nachts, als böses Omen.

2) Ich erinnere hiebei (abgesehen von der hektischen Röte der Lungentuber-

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Beim bekannten Zusammenhang zwischen Nase und Lungenaffektionen (Asthma bronchiale) ist es bemerkenswert, daß Franck (Arch. de physiol. 1889, p. 550) bei Reizung der Schleimhaut eine Gefäßerweiterung im ganzen Kopf bemerkte, die auf der entsprechenden Seite am stärksten war.

Die Steigerung der Zyanose während der Anfälle möchte ich als Reflex infolge der Schmerzanfälle ansehen. Nachdem von Haus aus schon Zyanose und Neigung zu Erweiterung der Hautgefäße besteht, ist es erklärlich, daß ein Reiz viel leichter in schon präformierten Bahnen und ausgefahrenen Geleisen sich bewegt.

Im Gegensatz zu diesem, wenn es zu sagen erlaubt ist, Dolor caeruleus, ist der Angina pectoris, bei der an und für sich infolge der Aortenstörung Anämie vorherrscht, ein Dolor pallidus eigen. Im ersteren Falle scheint demnach eher Hautvasomotorenlähmung (Relaxation der Gefäße), im zweiten Falle eher Spasmus zu bestehen[1]). Auch andere Momente scheinen für einen gewissen Antagonismus der Verhältnisse bei Affektionen dieser beiden Gefäße zu sprechen (s. u.).

Die mechanischen Verhältnisse des Lungenkreislaufes ermöglichen in der Norm ein Durchströmen mit der geringsten Verschwendung von Kraft.

Wie verhält sich der Widerstand in den peripheren Gefäßgebieten der Pulmonalis zum Blutdruck in derselben.

Daß ersterer im allgemeinen ein nur sehr geringer ist und daß nur sehr beträchtliche Veränderungen der Gefäßweite einen merklichen Einfluß auszuüben vermögen, wird auf Grund der Lichtheimschen Versuche angenommen, der am kuraresierten Hunde $^3/_4$ des gesamten Pulmonalgefäßgebietes ausschalten konnte, ohne daß dadurch der Druck im großen Kreislauf im mindesten herabgesetzt wurde[2]).

Tigerstedt führt die stärkere Durchströmung des restlichen Gefäßgebietes auf Grund von Injektionsversuchen darauf zurück, daß schon unter normalen Verhältnissen die Blutverteilung in den Lungenabschnitten eine außerordentlich verschiedene ist. Daß der Widerstand in den Lungengefäßen ein sehr geringer ist, ergibt sich auch aus der ungemein großen Strömungsgeschwindigkeit im Pulmonalgebiet (Stewart, Tigerstedt, 2—4 Sekunden).

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kulose) an das alte Peter-Franksche Symptom der gleichseitigen Gesichtsrötung bei Pneumonie.

1) S. Nothnagels Angina pectoris vasomotoria. D. Arch. f. klin. Mediz. III.

2) Landgraf (Klinisches und Experimentelles zur Lehre von der Embolie der Lungenarterie. Zeitschr. f. klin. Med. Bd. 20) erhielt vollkommen entgegengesetzte Resultate. Er unterband am spontan atmenden Kaninchen ohne Eröffnung der Pleurahöhlen die linken Arteria pulmonalis. Die sehr schwierige Operation gelang nur in 4 Fällen, wobei jedoch auf die Kompression der linken Lungenarterie prompt ein Sinken des Aortendruckes folgte. Dabei füllte sich der Stamm der Lungenarterie, dann sah man die rechte Kammer sich erweitern und zugleich das linke Herzohr blasser werden. (Aortenkurve sank auf die Hälfte und stieg mit dem Aufhören der Kompression wieder an.) Landgraf meint, daß die Lichtheimschen Ergebnisse nur für kuraresierte und künstlich respirierte Tiere Geltung hätten. Bei einer Wiederholung dieser Versuche durch Tigerstedt konnten die Angaben Landgrafs nicht bestätigt werden.

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Tigerstedt (Über den Kreislauf nach Bindung der linken Lungenarterie. Skand. Arch. f. Physiol. 1907, XIX, 4 und 5, p. 231) stellte an kuraresierten Kaninchen fest, daß durch temporäre Abklemmung der linken Lungenarterie die Sekundenvolumina der linken Herzkammer zwar abnehmen, daß die Abnahme aber im großen und ganzen als sehr geringfügig zu erachten ist. Es genügt demnach, nach Ausschaltung der einen Lunge der noch übriggebliebene Teil des kleinen Kreislaufs, um das linke Herz mit etwa derselben Blutmenge wie vorher zu speisen. Auch der Blutdruck im großen Kreislauf erfährt hierbei nur unwesentliche Änderungen.

Der Lichtheimsche Versuch ist nur so zu erklären, daß hiebei der rechte Ventrikel sein Blut durch den offenen Teil der Gefäßbahn mit noch erhöhter Geschwindigkeit treibt.

In den normalen Partien der Lunge werden die Gefäße erweitert, bald jedoch vermag ihre Dilatation allein das Hindernis nicht mehr auszugleichen und nun steigt auch der Druck in der Pulmonalarterie stärker an.

Bei diesem dem Körperkreislauf entgegengesetzten Verhalten hängt es ganz von der Stärke und Ausbreitung des Hindernisses ab, wie weit es durch Gefäßdilatation, wie weit es durch verstärkte Herzaktion ausgeglichen wird, was in jedem Fall das Verhalten des Ventrikels beeinflußt, dessen Arbeit sich vergrößert und bei längerem Bestehen Hypertrophie seiner Wandung herbeiführt.

Die mechanischen Verhältnisse beider großen Gefäßstämme an der Herzwurzel verdienen ebenfalls eine Berücksichtigung.

Bei Sklerosierung und Erweiterung der Aorta vermag die ohnehin weiche, nachgiebige Pulmonalis leicht auszuweichen; anders umgekehrt —, etabliert sich ein derartiger Prozeß mit Erweiterung an der Pulmonalis, so setzt die starre, stark gespannte Aorta einen Widerstand entgegen. Eine Prädilektionsstelle der Pulmonalsklerose ist nun nach Reiche anscheinend die Stelle, die der Aorta ascendens unverschieblich anliegt. Nun verlaufen im Bindegewebe zwischen den beiden Gefäßen Herznervengeflechte (Nervus depressor).

Eine Ausdehnung der Pulmonalis, zumal wenn sie mit Verhärtung der Wand und gewissen periarteriitischen Vorgängen vergesellschaftet ist, kann nun zu einem Druck, Verzerrung u. dgl. der Nerven und konsekutiven Reiz- oder Lähmungserscheinungen führen.

Nach Köster, Tschermak, Hirsch und Stadler ist der Nervus depressor nicht ein Reflexnerv des Herzens, sondern der Aorta. Diese hat in ihrem Anfangsteil die Bedeutung eines „Windkessels“ (das systolisch ausgeworfene Blut aufzuspeichern und zu verteilen). Als wesentlichste Funktion des Depressors scheint, da er durch Steigerung des Füllungsdruckes im Aortenbogen erregt wird, die Verhütung der Überdehnung dieses Windkessels zu bestehen. Eine Beeinflussung dieser Funktion infolge derartiger geschilderter pathologischer Pulmonalarterienprozesse scheint demnach der Örtlichkeit nach nicht ausgeschlossen zu sein und es dürften möglicherweise auch Anomalien der Blutverteilung im großen Kreislauf z. T. hierauf beruhen.

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Wenn auch an anderer Stelle noch eingehender über das Tierexperiment zu berichten sein wird, so mögen doch hier schon einige Tatsachen gestreift werden, die bei tierexperimentellen Untersuchungen gefunden, uns zur Erklärung oder zum näheren Verständnis mancher hier niedergelegten klinischen Befunde dienen können.

Der Einfluß gewisser infektiöser Schädlichkeiten für das Entstehen der Arteriosklerose wurde auch experimentell geprüft.

Von Klotz (Experimental production of arteriosclerosis. Brit. med. journ. 1906, II.) wurden nach mehrfachen intravenösen Injektionen von Typhus- und Streptokokkenkulturen Veränderungen in der Aorta und Arteria pulmonalis gefunden. Die Intima wies Verdickungen auf, die durch Bindegewebsproliferation nach vorheriger Verfettung des subendothelialen Gewebes zustande gekommen waren; die Elastica interna war in mehrere Lamellen gespalten und die Wucherung von Bindegewebe erstreckte sich bis in die innersten Schichten der Media; Verkalkungen, Ausbuchtungen waren nie vorhanden.

Die Seltenheit des Prozesses in der Lungenarterie lassen im ganzen wohl auch die Tierexperimente erkennen, wobei hier natürlich in nähere pathologisch-anatomische und histologische Details und Streitfragen nicht eingegangen werden kann.

Grober (Massenverhältnisse des Herzens bei künstlicher Arterienstarre. Verhandl. des XXIV. Kongr. f. inn. Med. 1907, S. 449) konstatierte bei Adrenalininjektionen, daß die Nekrosen am stärksten an der Aortenwand oberhalb des Zwerchfells, nur selten an der Bauchaorta vorhanden waren, nie dagegen in den Splanchnikus-, den Extremitäten- und den Lungenarterien.

Über die positiven Befunde sind die Akten noch nicht geschlossen.

Cox (Experim. Beitr. zur patholog. Anatomie der Lungenentzündung. Zieglers Beitr. V. 1889) erhielt bei Tieren, die durch Injektion von Krotonöl in die Bronchien pneumonisch gemacht waren, eine Entzündung der Adventitia, Media und selbst der Intima der Lungenarterien, die nach wenigen Tagen zu einem sehr beträchtlichen Dickenzuwachs der Intima führte.

Schon Friedländer (Experim. Untersuchungen über chron. Pneumonie usw. Virchows Arch. 1878, Bd. 68) erzielte dieses bei Durchschneidung der Nervi recurrentes.

Dagegen macht Jores (Wesen und Entwicklung der Arteriosklerose. Wiesbaden, Bergmann, 1903) geltend, daß die Pulmonalisintima des Kaninchen schon ohne Erkrankung oft eine sehr erhebliche Dicke annehmen könne.

D'Amato (Weitere Untersuchungen über die von den Nebennierenextrakten bewirkten Veränderungen der Blutgefäße und anderer Organe. Berl. klin. Wochenschrift 1906) sah bei seinen Versuchen einmal ein kleines Aneurysma in den Lungenarterien (zuweilen degenerative Veränderungen in der Media und Adventitia der Hohlvenen).

Braun (Über Adrenalinarteriosklerose. Sitzungsber. der Kais. Akad. d. Wissensch. Wien. Math.-naturw. Klasse, 1907, Bd. III, 116) machte sehr schwache aber häufige Adrenalininjektionen. Die stärkste Intimaverdickung wird dabei an einer Lungenarterie beschrieben.

Nach Heubner (Experim. Arteriosklerose. Ergebn. der inneren Medizin 1908, I, S. 280) scheint die von Friedländer deutlich beschriebene Umwandlung eines anfangs auftretenden Granulationsgewebes in derbes Bindegewebe innerhalb der Intima deutlich darzutun, daß er einen reaktiven Prozeß als Folge des künstlich gesetzten Entzündungsreizes vor sich gehabt hat.

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Wegen des Joresschen Befundes (s. o.) von Verdickungen auch normaler Pulmonalarterienintima bei Kaninchen möchte Heubner dem Braunschen Ergebnis (s. o.) nur sehr beschränkte Bedeutung zuerkennen.

Eine umfassende Darstellung der Frage nach den Vasomotoren der Arteria pulmonalis ist wohl hier nicht gut möglich.

Cohnheim und Lichtheim nehmen an, daß die Arterien im Lungenkreislauf keinen durch Vasomotoren unterhaltenen Tonus besitzen.

Der Einfluß der Gefäßnerven ist nach Knoll für den kleinen Kreislauf allerdings viel geringer als im großen.

Es bedarf im ersteren ganz beträchtlich stärkerer Einwirkungen, um einen Effekt hervorzurufen.

Durch Reizung des Sympathikus erfolgt ein Steigen des Druckes in der Pulmonalis und ein Sinken desselben in der Aorta.

Auf Grund experimenteller Studien gelangt François-Franck (Recherches sur l'action du système nerveux sur la circulation pulmonaire à l'état normal et pathologique. Bull. de l'acad. de méd. 1896, no. 6) zu dem Resultat, daß in der Tat den Lungenarterien die Fähigkeit zukommt, sich auf verschiedene sensible Reize energisch zu kontrahieren.

Eine reflektorische Einwirkung auf die Vasomotoren der Lungengefäße wird bei der sog. Potainschen Krankheit[1]) supponiert.

Bei schmerzhaften Magen- und Leberaffektionen kann eine wesentliche Vergrößerung des rechten Herzens nachgewiesen werden, einhergehend mit den Symptomen von Blutstauung in demselben. Letztere werden auf einen Krampf der Pulmonalarterie bezogen, der von den Magen- und Lebernerven reflektorisch ausgelöst wird.

Pagano (Arch. ital. di Biol. 1900, vol. XXXIII) konnte von der Pulmonalis aus keinerlei Reflex auf die Schlagfrequenz des Herzens auslösen.

Broie und Russel (Journ. of Physiol. 1900, XXVI, 98) erhielten nach Reizung der pulmonalen Vagusäste eine Blutdrucksenkung (vielleicht infolge Verengerung der Lungengefäße nach Ansicht von François-Franck).

Die ganz verschiedenen Wirkungen einer Reihe von Giften auf die Gefäße des großen und kleinen Kreislaufs (vergl. Tigerstedt, Der kleine Kreislauf. Ergebn. der Physiol. 1903, II) dokumentieren die Eigenartigkeit der Lungengefäße im Verhältnis zu denen des großen Kreislaufs, sowie deren Selbständigkeit und Unabhängigkeit.

Velich (Über die Einwirkung des Nebennierenextraktes auf den Blutkreislauf. Wien. mediz. Wochenschr. 1898, S. 1257) kam zu dem Versuchsergebnis, daß Nebennierenextrakt in größeren Dosen nicht

1) Potain, Rapports de la dilatation cardiaque avec les affections du foie. Journ. de méd. et de chir. 1898, April 4.

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allein den Druck im großen Kreislauf, sondern auch den in der Arteria pulmonalis steigert. In kleinerer Menge bewirkte er keine Drucksteigerung im kleinen Kreislauf, obgleich der Aortendruck zu gleicher Zeit von 80 auf 260 mm Hg anstieg.

Nolf (Action des injections intraveineuses de propepton sur la pression dans l'artère et la veine pulmonaires. Mém. cour. et autres Mém. publ. par l'Acad. des Scienc. de Belgique 1903, 63, p. 1) gibt an, daß bei Injektion von Grüblers oder Wittes Pepton neben der längstbekannten Druckabnahme im großen Kreislauf eine in der Regel bedeutende und dauernde Drucksteigerung in der Lungenarterie, sowie ein entsprechender Druckabfall im linken Vorhof eintritt. Die nähere Analyse der Erscheinungen ergab, daß hierbei die Lungenvasomotoren tätig waren; es liegt hier eine deutliche Gefäßkontraktion in den Lungen vor.

Die Traube-Heringschen Wellen der Blutdruckkurve sollen der Ausdruck der direkten Beeinflussung des vasomotorischen Zentrums durch das Atemzentrum sein, und nach Hering durch eine rhythmische Irradiation der Erregungen des Atemzentrums auf das vasomotorische Zentrum entstehen.

Auch die Pulmonalarterie läßt diese Wellen erkennen, allerdings mit geringerer absoluter Amplitude.

Nach Durchschneidung des Annulus Vieussenii beiderseits (von welchem nach François Franck die sympathischen Vasomotoren der Lunge ausgehen) verschwinden die Wellen in der Arteria pulmonalis.

F. B. Hofmann (Allgemeine Physiologie des Herzens. Nagels Handb. der Physiol. Bd. I. 1905) legt in ausführlicher Weise mit Berücksichtigung der gesamten Literatur die Experimente dar, die den Beweis für die Existenz von Gefäßnerven für die Lunge zu erbringen imstande seien (S. 298), wobei er zu dem Schlusse gelangt, daß alles zusammengenommen heute die Existenz von Vasomotoren für die Lungengefäße ziemlich wahrscheinlich ist, wobei jedoch über deren nur geringe Wirkung alle Autoren übereinstimmen.

Strubell (Über vasomorische Einflüsse im kleinen Kreislaufe. Verhandl. des XX. Kongr. f. inn. Mediz. 1902, S. 404, und Über die Vasomotoren der Lungengefäße. Arch. f. Physiol., Jahrg. 1906, Supplem.-Bd. S. 328) tritt auf Grund seiner Experimente bei Verwendung kombinierter Messung des Arteriendruckes und linken Vorhofsdruckes für die Existenz von Vasomotoren in den Lungengefäßen (Pneumovasomotoren) ein und vermutet, daß die Bronchokonstriktion (Lungenblähung) und die Vasokonstriktion (Lungenvolumen-Verkleinerung und Sinken des Vorhofsdruckes) nacheinander auftreten.

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Anhangsweise sei hinzugefügt, daß Krogh (Verhandl. der biolog. Gesellsch. in Kopenhagen, 28. Febr. 1907) den Nachweis eines vasomotorischen Systems in der Lunge der Schildkröten erbrachte.

Vielerlei Fragen sind uns jedoch bezüglich der Lungenvasomotoren noch dunkel: ob sie einen Tonus besitzen, unter welchen Bedingungen eine normale Erregung stattfindet, wie die Regulierung der Blutzufuhr, zu der sie zweifellos in Beziehung stehen, erfolgt.

In neuester Zeit stellte sich bei der experimentellen Erforschung ein gewisser Antagonismus oder zum mindesten ein differentes Verhalten zwischen Pulmonalarterie und Koronararterien heraus (vgl. oben).

Langendorff (Über die Innervation der Koronargefäße. Ztrlbl. f. Physiol. 1907, Bd. 21, S. 551) kam zu dem unerwarteten Resultate, daß Suprarenin und Adrenalin erschlaffend auf die Muskulatur dieser Gefäße wirken, während dieselben Stoffe die Muskulatur der Lungenarterie zur Kontraktion veranlassen. Damit steht im Einklang, daß Sympathikusreizung beim isolierten Herzen eine Erweiterung der Koronargefäße bedingt.

Dieses Verhalten fordert zu weiteren Untersuchungen auf, da hieraus mancherlei Einblicke in Verhältnisse, die auch die Klinik lebhaft interessieren, zu erhoffen sind.

Die präzise Fragestellung, die unserer Mitteilung zugrunde liegt, lautete:

Unter welchen Umständen ist eine klinische Diagnose der Pulmonalarteriensklerose möglich?

Auf Grund eines bei der relativ großen Seltenheit des Befundes reichlich zu nennenden Beobachtungsmateriales kam ich zu folgenden Schlußsätzen:

Eine klinische Diagnose der Atherosclerosis pulmonalis ist (falls die Kranken in einem entsprechenden Stadium und durch sehr lange Zeit beobachtet werden können):

A. unter Voraussetzung einer primären Mitralstenose möglich und bei Anwesenheit nachstehender Symptome und Erscheinungskomplexe mit allergrößter Wahrscheinlichkeit zu stellen:

I. bei physikalischer Untersuchung:

1. Dämpfungszone am oberen linken Sternalrand und den benachbarten Gebieten mit Druck- und Perkussionsempfindlichkeit (besonders in bestimmter Lage).

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2. auch für diese Affektion ungewöhnlich starke Verbreiterung der Herzdämpfung nach rechts.
3. Verhalten der oberen Anteile des mittleren Bogens bei Röntgendurchleuchtung.
4. allmähliches Aufwärtswandern des diastolischen Schwirrens und des diastolischen (präsystolischen) Geräusches gegen das Pulmonalostium zu.

II. klinischen Symptomen:

1. auffallende Zyanose als Frühsymptom und lange Zeit hindurch bestehende ausgesprochene Differenz zwischen dieser und der fehlenden oder geringen Dyspnöe, sonstigen Stauungserscheinungen, Ödemen.
2. Auftreten der Anfälle der Dyspragia intermittens angiosclerotica pulmonalis (Angina hypercyanotica).
3. trotz hochgradiger Zyanose Fehlen der Trommelschlegelfingerbildung.
4. Wiederholte abundante Lungenblutungen ohne ausgesprochenen Infarktcharakter.

B. Unter genauer Berücksichtigung vorliegender Momente ist auch das klinische Erkennen der so überaus seltenen primären Pulmonalsklerose in das Bereich der Möglichkeit gerückt.

Wir werden bei Vorhandensein obiger Erscheinungen und Fehlen der Zeichen eines wirklichen Vitium cordis (Mitralstenose) an einen derartigen atherosklerotischen Prozeß der Lungenschlagader denken, ganz besonders wenn es sich um eigenartige zweifelhafte Herzaffektionen handelt, die einem angeborenen Vitium cordis ähnlich erscheinen.

Hierbei verdienen namentlich basale diastolische Geräusche am Pulmonalostium ohne Zeichen von Insuffizienz der Klappen, auffällige Hypertrophie der rechten Herzkammer, das Prävalieren der Zyanose über sonstige Stauungserscheinungen (Dyspnöe, Ödeme), Attacken von basalen Schmerzen mit dem Charakter der Dyspragia intermittens pulmonalis, Fehlen von Trommelschlegelfingerbildung besondere Berücksichtigung.

Einen weiteren Hinweis bildet das verhältnismäßig noch jugendliche Alter und vorausgegangene schwere Infektionskrankheiten (Polyarthritis, Perikardaffektionen, „Variola").

Es soll hier nochmals daran erinnert sein, daß Mitralstenosen und gewisse angeborene Herzfehler, die einen ganz ähnlichen klinischen Befund bieten, beide mitunter sehr hohen Grades, infolge Fehlens auskultatorischer Erscheinungen einer sicheren Diagnose ganz ungewöhnliche Schwierigkeiten bereiten können. Die dargelegten Momente

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dürften auch einen Mitbehelf bieten zur Differentialdiagnose und Entzifferung derartiger rätselhafter Herzkrankheiten.

C. Falls die Pulmonalissklerose bei der viel seltener mit ihr kombinierten Sklerose und Insufficientia semilunarum aortae auftritt, geben einige klinische Eigenheiten ebenfalls einen Fingerzeig zur diagnostischen Verwertung.

Solche sind statt der zu erwartenden blassen Facies aortica ausgesprochene Zyanose, die ebenso wie eine bestehende Hypertrophie des rechten Herzens durch andere Ursachen nicht erklärt werden kann, namentlich wenn die Zyanose anfallsweisen Charakter zeigt. Ferner eine abnorme Querleitung des diastolischen Geräusches nach links.

Eine Analyse der Dyspragia pulmonalis gestaltet sich wegen der Möglichkeit gleichzeitiger Stenokardie schwieriger.

Allgemein und schematisch für den gesamten Prozeß ausgedrückt, liegen den subjektiven und allgemein klinischen Symptomen mehr die Affektion der kleinen Gefäße (Endarteriitis obliterans) zugrunde, während für das Zustandekommen der objektiven Befunde, speziell der physikalischen Untersuchung, das Befallensein des Stammes mehr releviert.

Vorstehende Notizen bezwecken vor allem die Aufmerksamkeit der Kliniker auf die bisher nur als zufälliger Sektionsbefund rangierende Atherosclerosis pulmonalis hinzulenken und sie zu weiteren Beobachtungen und Untersuchungen über die von uns behauptete Möglichkeit der klinischen Diagnose des Leidens zu veranlassen.

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(Gynäkologie Nr. 185.)

Die wechselseitigen Beziehungen der innersekretorischen Organe, insbesondere zum Ovarium.

Zugleich ein Beitrag zur Lehre von der Menstruation.

Von

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In den letzten Jahren ist so viel Material in bezug auf die innere Sekretion verschiedener Organe aufgestapelt worden, wobei wiederholt auch auf das Ovarium Rücksicht genommen wurde, daß es sich der Mühe lohnt, alle diesbezüglich bekannten Tatsachen von einem einheitlichen Gesichtspunkte aus zusammenzufassen, um so mehr, als sich aus dieser Betrachtung einige neue Anschauungen zu ergeben scheinen. Dies soll im folgenden versucht werden.

I. Theorie der Menstruation.

Meine diesbezüglichen Studien haben mich vor allem zu der Erkenntnis geführt, daß man mit keiner der bisher bekannten Theorien der Menstruation sein Auskommen findet. Wollen wir aber das Verhältnis der anderen sekretorischen Organe zum Ovarium verstehen, so müssen wir vor allem über die Funktion der Genitaldrüsen Klarheit gewinnen.

Nachdem eine Reihe von Theorien verlassen wurde, kam man auf Grund der Born-Fränkelschen Theorie zu der Anschauung, daß ein durch die periodische Funktion des Ovariums in ihm produzierter, an die Blutbahn abgegebener Stoff es ist, der den Anstoß zu den menstruellen und Schwangerschaftsveränderungen gibt. Man ist sich nicht ganz klar darüber, ob es das Corpus luteum oder die inter-

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stitiellen Zellen sind, welche hier in Betracht kommen. Die Funktion des Ovariums soll eine Kongestion der Sexualorgane und ferner eine Toxämie, aber auch zugleich eine Entgiftung des Organismus hervorrufen. Diese beiden einander widersprechenden Funktionen will man sich oder vielmehr könnte man sich nur so erklären, daß das Ovarium giftige Stoffe an den Organismus abgibt, während die Entgiftung dadurch stattfindet, daß diese Stoffe durch die Menstruation ausgeschieden werden. Nach dieser Anschauung wäre also das Ovarium den den Organismus vergiftenden innersekretorischen Organen zuzuzählen. Ich werde später zeigen, daß alle den Organismus vergiftenden Substanzen Blutdrucksteigerung hervorrufen. Wir wissen aber, daß das Eierstockssekret Blutdrucksenkung bewirkt. Schon dieser Umstand spricht einigermaßen gegen die herrschende Anschauung. Der beste Prüfstein für die Funktion eines innersekretorischen Organes sind jene Erscheinungen, welche nach der Exstirpation des Organes auftreten. Nach der ausführlichsten diesbezüglichen Arbeit von Mandl und Bürger[1]) erlischt mit dem Erlöschen der Funktion des Ovariums die Menstruation und die Menstruationswelle. Schon hier stoßen wir auf einen Widerspruch in der Literatur. Ohlshausen hat nach Kastration wegen Myom noch 3mal typische Menstruation auftreten gesehen. Neuerer Zeit hat Gellhorn[2]) einen Fall veröffentlicht, wo nach Exstirpation der Ovarien noch immer Menstruation auftrat, bis man einen Peritonealstrang durchtrennte. In beiden Fällen könnte man eventuell an das Zurückbleiben von Ovarialresten oder eines überzähligen Eierstocks denken. van de Velde hat durch Darreichung von Ovarialtabletten auch im Klimakterium Menstruation erzeugen können, sofern man unter Menstruation Blutung versteht. Das gleiche ist Gellhorn gelungen. Man könnte derlei Erfahrungen ohne Zweifel auf die unbedingte Abhängigkeit der Menstruation von Ovarialsubstanzen beziehen. Und doch stimmt dies nicht ganz. F. Deales[3]) zeigte ebenso wie Ficarelli und Holterbach, daß man Brunsterscheinungen beim Tiere, die man sicherlich biologisch, insbesondere bei Hunden, an denen ersterer experimentierte, der Menstruation gleichstellen kann, durch Yohimbin zu erzielen vermag. Daraus müssen wir folgern, daß die Menstruation (Blutung) ein dem Uterus eigentümlicher Vorgang ist, der durch jede Substanz, welche ebenso wie das Ovarium zu Blutüberfüllung im Uterus führt, hervorgerufen werden kann. Viel-

1) Die biologische Bedeutung der Eierstöcke nach Entfernung der Gebärmutter. Deuticke 1904.

2) Menstruation ohne Ovarien. Zentralbl. f. Gyn. 1907, Bd. 40.

3) Berliner klin. Wochenschr. 1907, Bd. 42.

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leicht gehört auch hierher die ursprünglich Pflüger-Straßmannsche Anschauung, daß Druckerhöhung im Ovarium Hyperämie der Uterusschleimhaut erzeugt. Umgekehrt könnte man eine Erscheinung, welche bisher eine Erklärung nicht gefunden hat, nämlich das Ausbleiben der Menstruation i. e. Blutung bei Fisteln auf das Fehlen eines druckerhöhenden Momentes, nämlich das Fehlen der Blasenfüllung zurückführen.

Ich habe im vorhergehenden nur auf das äußere Moment der Menstruation, nämlich die Blutung, Rücksicht genommen, und schon hier zeigte es sich, daß eine unbedingte Abhängigkeit der Blutung vom Ovarium nicht besteht. Noch mehr zeigt sich dies bei Betrachtung der Sekretionserscheinungen. Ich will hier gar nicht darauf eingehen, daß bei der Annahme, die Tätigkeit des Ovariums erzeuge die Vergiftung und löse bei maximaler Tätigkeit oder Anhäufung der Stoffe die Entgiftung aus, der Zustand des Ovariums zur Zeit der Menstruation stets derselbe sein müsse, und doch sind bis jetzt sekretorische Veränderungen in den interstitiellen Zellen, eine allmähliche periodische Zunahme und Abnahme des Sekrets nicht nachgewiesen. Auf noch größere Unregelmäßigkeiten stoßen wir bei der Annahme, daß die Menstruation mit der Follikelreifung in ursächlichem Zusammenhang steht. Ich verweise hier auf die Arbeit von Ancel und Villemin[1]) und von Leopold und A. Ravano[2]). Ja, es gibt Versuche, die zeigen, daß die Menstruation auch von anderen Drüsen abhängig ist, so von der Schilddrüse. Französischen Autoren, so Faveau de Courmelles, und später M. Fränkel ist es gelungen, durch Bestrahlen der Schilddrüse die Menstruation i. e. die äußeren Zeichen derselben, die Blutung, ganz zu sistieren oder zu schwächen.

Wenn wir vollends zu den Ausfallserscheinungen übergehen, so läßt sich meiner Ansicht nach die Theorie, daß der Uterus gleichsam nur der Ausführungsgang des Eierstockes ist, nicht recht festhalten. Es ist wohl richtig, daß man sich die Erscheinungen des Klimakteriums durch den Ausfall der Eierstockstätigkeit ohne weiteres erklären kann, ebenso wie die strumipriven Symptome durch den Ausfall der Schilddrüsentätigkeit. Aber schon hier ist es sehr auffallend, daß der Ausfall der Eierstockstätigkeit ähnliche Symptome macht, wie nach der bisherigen Theorie in der prämenstruellen Zeit und in den ersten Tagen der Menstruation, die maximale Tätigkeit des Ovariums beziehungsweise die Anhäufung der Sekretionsprodukte des Ovariums. Wird der Uterus entfernt, und werden die Ovarien belassen, so er-

1) Société de Biologie. Juillet 1907.
2) Arch. f. Gyn. Bd. 83, H. 3.

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liecht zwar zunächst die Menstruation als sichtbarer Ausdruck der Ovarialtätigkeit, die Welle aber bleibt erhalten. Ich frage nun, wie in diesen Fällen eigentlich Entgiftung zustande kommt. Es ist ja richtig, daß nicht allzuselten die Schilddrüse hypertrophiert. Man könnte also annehmen, daß hier die Entgiftung durch die Schilddrüse stattfindet. Dies alles setzt aber voraus, daß tatsächlich das Ovarium eine blutdrucksteigernde Drüse ist, was sicherlich nicht zutrifft. Nun treten in einer Reihe von Fällen Molimina menstrualia auf. Darunter versteht man nervöse Erscheinungen, die mit Blutdrucksteigerung einhergehen, und gerade zu der Zeit, welche jener entspricht, in der die Menstruation eintreten sollte, sich äußern. Daß die Erscheinungen mit dem Hochstande des Blutdruckes zusammenfallen, davon konnte ich mich wiederholt überzeugen. Ich verfüge überdies über zwei Blutdruckkurven von Frauen, denen der Uterus entfernt wurde. Die Kurve zeigt, ähnlich wie die von Mandl und Bürger veröffentlichte, eine der normalen Menstruationswelle ganz analoge Gestalt mit Molimina menstrualia auf dem Höhepunkt des Blutdruckes. Diese Erscheinung mußte also nach der herrschenden Anschauung auf das Ovarium bezogen werden, indem die Anhäufung der sekretorischen Produkte schließlich zu den Beschwerden führt, während die Entgiftung durch irgendein uns derzeit noch unbekanntes Organ, vielleicht durch die Schilddrüse erfolgt. Merkwürdigerweise finden sich hier auch regelrechte Ausfallserscheinungen, nach Mandl und Bürger in 47%, während Molimina menstrualia nur in 26% vorhanden sind. Diese also ziemlich häufigen Erscheinungen sind bei Aufrechterhaltung der derzeit herrschenden Theorie schwer zu erklären. Sie treten zu der Zeit auf, wo die Welle gerade nicht sehr hoch ist, oder ein plötzliches Ansteigen und ein ebenso plötzlicher Abfall nachweisbar ist. Man nimmt in diesen Fällen zu der wahrscheinlich richtigen Ansicht Zuflucht, daß hier die Ovarien nicht funktionieren. So richtig auch die Tatsache an sich sein mag, in das Schema paßt sie logischerweise nicht hinein. Wenn die Steigerung der Sekretion der Ovarien, beziehungsweise die Anhäufung der Stoffe zu den Molimina menstrualia führt, kann doch unmöglich der Wegfall eben dieser Stoffe die gleichen Erscheinungen hervorrufen. Denn zwischen Molimina menstrualia und Ausfallerscheinungen besteht nur der Unterschied, daß erstere in und zu bestimmten Zeiten wiederkehren, letztere aber häufiger und in unregelmäßigen Intervallen.

Vollends unverständlich bleiben aber bei dieser Theorie die Tatsachen, die sich aus der Mitentfernung der Ovarien ergeben. Bei Mitentfernung der Ovarien treten in 73% Symptome auf. Man sollte annehmen, daß wenn das Organ, welches tatsächlich bei stärkster

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Sekretion die Symptome erzeugt, entfernt wird, keine Symptome mehr auftreten, welche wir sonst auf die Sekretion dieses Organs beziehen. Es sollten also logischerweise bei Entfernung des Uterus samt Adnexen im Gegenteil keine Ausfallserscheinungen auftreten. Belassen wir eines oder beide Ovarien, so finden sich in 67% Symptome. Ich möchte nun meinen, daß der Unterschied so gering ist, daß man eigentlich den Ovarien fast keinen Einfluß auf die Ausfallssymptome zuschreiben sollte. Aber vielleicht ist die Heftigkeit der Symptome eine geringere. Heftige Symptome bei Belassen der Ovarien in 15%, bei Wegnahme der Ovarien in 19%. Bei Belassen des vergiftenden Organs also auch höher als bei Wegnahme desselben. Und tatsächlich haben wir ja alle die auf größere oder geringere Erfahrungen gestützte Ansicht, daß die Ovarien die Ausfallserscheinungen mildern. Die Tatsachen stehen in strengstem Widerspruch zu der Theorie der vergiftenden Wirkung des Ovariums. Wir fühlen uns unbedingt zu der Ansicht gedrängt, daß das Ovarium ein entgiftendes Organ ist. Dann ist auch der Ausdruck Ausfallserscheinungen gerechtfertigt. Es ergebe also diese Anschauung die Theorie, daß im Körper Stoffwechselvorgänge sich abspielen, die zu einer allmählichen zunehmenden Intoxikation mit begleitender Blutdrucksteigerung führen, die schließlich die Sekretion des Ovariums auslösen; diese Sekrete wirken nun auf den Uterus ein, der durch äußere Sekretion und Blutung die giftigen Stoffe ausscheidet; vielleicht werden durch das Ovarialsekret direkt die giftigen Stoffwechselprodukte entgiftet. Blutdrucksenkung ist die Folge.

Prüfen wir diese Theorie an einigen Tatsachen. Totalexstirpation des inneren Genitales muß zu Ausfallserscheinungen führen, da die Entgiftung durch das Ovarium fehlt. Größtenteils treten aber andere Organe ein, so anerkanntermaßen die Schilddrüse, deren Tätigkeit die Entgiftung besorgt. Werden beide Ovarien belassen oder auch nur eines, so treten die Ausfallserscheinungen etwas seltener auf; das Ovarium sezerniert ja und kann die Stoffwechselprodukte entgiften, es fehlt die Sekretion des Uterus und die Blutung, daher kommt es doch und zwar nicht so selten zu Symptomen. Die Welle bleibt erhalten, Molimina treten auf, denn die Entgiftung erfolgt periodisch. In diese Theorie passen auch sehr gut die Beobachtungen Gellhorns und van de Veldes, daß Ovarialtabletten die Menstruationsblutung auslösen können.

Aber auch diese Theorie kann nicht voll befriedigen. Es ist zunächst auffallend, daß der Körper beim geschlechtsreifen Weibe zur Entgiftung noch des Ovariums bedarf, während vorher und nachher die anderen entgiftenden Drüsen ausreichen. Man könnte dies auf ein Plus an toxischen Stoffen beziehen. Tatsächlich spricht ja vieles

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dafür. Niemals erreicht im normalen Zustand der Blutdruck die Höhe wie zur prämenstruellen Zeit. Hierzu kommen die prämenstruellen Vergiftungserscheinungen. Es liegt nun nahe, in der eireifenden Tätigkeit dieses Plus zu suchen, derart, daß durch das Heranreifen der Eier toxische Produkte gebildet wurden. Dagegen spricht so manches, vor allem die zeitliche Unabhängigkeit der Eireifung von der Periode. Ferner beginnt nicht selten die Eireifung vor der Menstruation und überdauert dieselbe. Wir sehen bei Haustieren oft die Brunst eintreten ohne Reifung von Eiern. Nach Hensen fallen bei Kaninchen brünstige Erregung und Eiablösung nicht immer zusammen. Bei den Fledermäusen[1]) sind Brunst und Ovulation durch Monate voneinander getrennt. v. Winckel[2]) erwähnt eine Frau mit 17 Schwangerschaften, die gerade zu der Zeit, wo sie nicht menstruierte, schwanger wurde. Sprungreife Follikel beobachtete man im Klimakterium, Schwangerschaft vor Eintritt der Menstruation sowohl in der Pubertät, wie auch in der Laktationsperiode. Dann ist es kaum glaublich, daß die Follikelflüssigkeit — und nur um diese kann es sich handeln — giftig sei, da doch das Ei in ihr aufwächst.

Ich meine, daß dieses Plus an Toxinen vom Uterus geliefert werden könnte. Der Uterus würde sonach zu den innersekretorischen Organen vergiftender, blutdrucksteigernder Natur zu rechnen sein. Wir werden sehen, daß sich mancherlei Erscheinungen dadurch zwanglos erklären lassen, daß sich dann das Sekretionspaar Uterus-Ovarium mit Leichtigkeit in die Serien der anderen innersekretorischen Organe einfügen läßt, daß wir so zu einer allen Erscheinungen gewachsenen Menstruationstheorie kommen, und daß schließlich einige Tatsachen für die innersekretorische Funktion des Uterus zu sprechen scheinen.

Nicht ganz uninteressant ist diesbezüglich die Tabelle II von Mandl und Bürger: Supravaginale Amputation, Totalexstirpation des Uterus per laparotomiam mit Belassung eines oder beider Ovarien. Die Fälle von supravaginaler Amputation geben in 57%, die von Totalexstirpation in 42% Ausfallserscheinungen; dies ist um so auffallender, als sich bei der Nachuntersuchung die Ovarien bei supravaginaler Amputation stets in gutem Zustande befanden, während sie bei Totalexstirpation des Uterus 4mal nicht palpabel waren, 1mal sich eine in ihrer Größe nicht genau bestimmbare Resistenz vorfand. Dies spricht wohl sehr dafür, daß das schlechtere Resultat auf den Uterusrest zu-

1) R. Müller, Sexualbiologie. Berlin, L. Marcus, 1907.

2) Handbuch d. Geburtsh. I.

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rückzuführen ist. Dieser Befund ist auch deshalb von großer Bedeutung, da nach Mandl und Bürger ein Abhängigkeitsverhältnis der Ovarien vom Uterus nicht bestehen soll, wir also nicht annehmen dürften, daß der Unterschied in dem Zustande der Ovarien gelegen ist. Dem ist aber nicht so; wir werden später sehen, daß die Sekretion des Ovariums doch vom Uterus bis zu einem gewissen Grade abhängig sein dürfte, und ich sehe gerade die obigen Nachuntersuchungen als einen Beweis dafür an, daß eine solche Abhängigkeit besteht. Wäre ferner das Ovarium das vergiftende Organ, so wäre wohl die größere Zahl der Ausfallserscheinungen bei erhaltenem Ovarium verständlich; da es aber ein entgiftendes Organ ist, so müssen wir die größere Zahl der Ausfallserscheinungen auf den Uterusrest beziehen.

Auf den ersten Blick spricht nur eine Tatsache gegen die hier vertretene Ansicht, nämlich die Tatsache, daß Totalexstirpation des inneren Genitale in $^3/_4$ der Fälle mit Ausfallserscheinungen einhergeht; da der Uterus als vergiftendes Sekretionsorgan wegfällt, so sollten auch Ausfallserscheinungen fehlen. Diese Überlegung ist nicht richtig; denn ich setzte oben auseinander, daß das Ovarium nicht allein entgiftend auf das eventuelle Uterussekret wirke, sondern auch auf andere Stoffwechselprodukte, daß das Uterussekret nur ein Plus darstelle. Der Wegfall der Ovarien schlägt aber gewaltig Bresche in die Reihe der entgiftenden Organe, die nun plötzlich die Aufgabe des Ovarium übernehmen müssen, das bis dato die übrigen erfolgreich unterstützt, vielleicht sogar vertreten hat. Können die anderen durch Hypersekretion den Ausfall decken, beispielsweise die Thyreoidea, die sehr häufig vergrößert befunden wird, dann fehlen Ausfallserscheinungen gewöhnlich. In einer großen Zahl von Fällen sind diese aber in mäßiger Weise, nur in 19% in heftiger Weise vorhanden. Es ist nämlich noch weiter zu berücksichtigen, daß die Hypertrophie der Thyreoidea, wie wir später sehen werden, auch nicht ohne Konsequenzen bleibt, daß wieder antagonistische Organe die verstärkte Tätigkeit der Schilddrüse zum Teil wettmachen. Ist einmal in das Konzert der sekretorischen Organe ein Mißton gekommen, dann gelingt es kaum mehr, volle Harmonie zu erzielen.

Im Widerspruch mit diesen Ausführungen stehen die drei Beobachtungen Abels[1]), wo bei Erhaltensein von Korpusschleimhautteilen die Menstruation fortdauerte und Ausfallserscheinungen fehlten. Die Zahl der Fälle gegenüber denen aus der Klinik Schauta und anderen

1) Dauerfolge der Zweifelschen Myomektomie. Arch. f. Gyn. Bd. 57.

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ähnlichen später zitierten, welche die gegenteilige Erfahrung ergeben, ist so gering, daß man hier nur von einer Zufälligkeit sprechen kann.

Deutlicher ergibt sich die Richtigkeit der Theorie aus folgenden weiteren Erfahrungen. Es muß hier zunächst auf das Verhalten der Ovarien nach Exstirpation des Uterus näher eingegangen werden. Durch die Untersuchungen von Mandl und Bürger, Burckhard[1]), Henckel (Zeitschr. f. Geb., Bd. 58, H. 3) u. a. wurde der Anschein erweckt, als ob die Ovarien vollkommen intakt blieben. Keitler fand, daß etwa 12—13 Monate nach vollzogener Uterusexstirpation reifende und sprungreife größere und kleinere Follikel, Corpora lutea und Primärfollikel vorhanden waren. Anders sahen die Befunde nach 3 Jahren aus. Mandl und Bürger fanden Degenerationserscheinungen am Ei und auffallend viele zystisch veränderte Follikel. Es war eine gewisse Hemmung in der Eireifung vorhanden. Ähnliche Befunde zeigten 4 Fälle Holzbachs (Arch. f. Gynäkol. 80, 2). Es war eine „hinreichende Menge funktionstüchtigen Ovarialgewebes" vorhanden. Dennoch hatten alle 4 Frauen Beschwerden. Trotz dieser Befunde sind alle Autoren der Ansicht, daß diese Ovarien punkto Sekretion normal sind. Nimmt man an, daß die Sekretion des Ovariums von den Luteinzellen abhängig ist, daß diese sich aber nur nach dem Sprunge eines reifen Follikels bilden, so muß man insbesondere in Rücksicht auf die Befunde von Mandl und Bürger sagen, daß hier sehr wenig Material für die Bildung der Luteinzellen vorhanden war. Wenn fast alle größeren Follikel zystisch entarten, wobei die Eier zugrunde gehen, dann muß es logischerweise höchst selten zur Follikelreife, zum Follikelsprunge und zur Bildung von Luteinzellen kommen. Für Anhänger der Luteinzellen-Sekretionstheorie müßten solche Ovarien wohl als funktionsuntüchtig gelten. Es käme da nur ein Befund in Betracht, der freilich von den Autoren nicht erwähnt und wohl auch nicht gemacht wurde. Ich muß hier auf meine demnächst erscheinende Arbeit über die Tätigkeit des Ovariums in der Schwangerschaft[2]) hinweisen. Ich fand nämlich, konform mit anderen Autoren, daß in der Schwangerschaft die größeren Follikel fast regelmäßig zystisch degenerieren, daß sich aber gleichzeitig die Theca interna in Luteinzellen, Follikelluteinzellen will ich sie nennen, verwandelt (Wallart, Seitz usw.). Diese Follikelluteinzellen haben nun meiner Ansicht nach sekretorische Funktionen. Wäre die Bildung solcher Follikelluteinzellen an den zurückgelassenen Ovarien beobachtet worden, so könnte man immerhin von einer relativen Funktionstüchtigkeit

1) Über Ausfallserscheinungen nach abdomineller Myotomie mit Zurücklassung der Ovarien. Berliner Gynäkologenkongr.

2) Arch. f. Gynäk.

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sprechen. Dieser Befund scheint aber nicht vorzuliegen, denn er wurde nicht erhoben.

Ich verfüge über kein derartiges Präparat, von Menschen stammend. Aber ich röntgenisierte einmal den trächtigen Uterus eines Kaninchens unter möglichster Abdeckung der Ovarien, es kam zum Abortus, zur Atrophie des Uterus, und nach einigen Monaten nahm ich die Ovarien heraus. Sie zeigten ein ähnliches Bild, wie dasjenige, welches Neumann und ich bei Bestrahlung der Ovarien trächtiger Kaninchen erhoben haben[1]): zystische Degeneration der größeren Follikel; einzelne Follikel zeigten aber einen Follikelluteinzellenbesatz. Dieser Befund läßt sich leider nicht verwerten, weil er erstens und vor allem noch vereinzelt ist, weil einige Trächtigkeiten vorausgegangen sind, und weil bei dem großen Reichtum der Kaninchenovarien an Follikelluteinzellen nicht zu entscheiden ist, ob die Zellen wirklich erst auf Grundlage der Degeneration der Follikel entstanden sind.

Bei dem derzeitigen Stand scheint es also festzustehen, daß von einer normalen Sekretion der zurückgelassenen Ovarien auf Grundlage der Luteinzellensekretion nicht die Rede sein kann.

Steht aber die innersekretorische Funktion mit den interstitiellen Zellen in Zusammenhang, so wären die bisher erhobenen Befunde in keiner Hinsicht beweisend; denn auf diese wurde keine Rücksicht genommen. In dem oben erwähnten Kaninchenovarium ließen sich vielleicht Merkmale einer Degeneration auffinden. Die Zellen waren kleiner, das Protoplasma vakuolisiert, die Kerne hin und wieder kariolytisch verändert, Befunde ganz ähnlich denen, wie wir sie an den röntgenisierten Ovarien erheben konnten. Sieht man von den Zysten ab, so war das Ovarium entschieden kleiner als de norma. Aber ich will aus diesem vereinzelten Befund keine Schlüsse ziehen. Nur das eine will ich nochmals hervorheben, daß die bisher erhobenen Befunde keinen Schluß zulassen über die Funktionstüchtigkeit der interstitiellen Zellen, daß also alle Autoren nicht berechtigt waren, aus den von ihnen erhobenen Befunden auf eine normale Sekretion der zurückgelassenen Ovarien zu schließen, daß vielmehr einige von ihnen erhobenen Befunde und die meinigen das Gegenteil wahrscheinlicher machen.

Auffallend ist es nun andererseits, daß sich vielfach bei Nachuntersuchungen, wie in den oben zitierten Fällen aus der Klinik Schauta, das Ovarium verkleinert erwies oder überhaupt nicht

1) Der Einfluß der Röntgenstrahlen auf die Eierstöcke trächtiger Kaninchen und auf die Trächtigkeit. Zeitschr. f. Heilkunde Bd. 28, H. 3.

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aufgefunden werden konnte. Dies ist um so auffallender, als, da die zystische Degeneration anfangs, oft auch später überall angetroffen wurde, eigentlich das Ovarium vergrößert sein sollte. Man muß also trotz der freilich pathologischen Follikelreife mit einem Zugrundegehen der Eierstöcke rechnen, wenigstens in vielen Fällen. Dieses Zugrundegehen, das sich ja auch innersekretorisch durch die Ausfallserscheinungen dokumentiert, wird nun von vielen Autoren mit der Gefäßversorgung in Zusammenhang gebracht. Mir scheint es nicht recht plausibel, daß nach Wegnahme des Uterus das Ovarium so schlecht versorgt werden sollte, daß es zugrunde gehen müsse. Es spricht wohl alles dafür, daß sich hier in diesem gefäßreichen Bezirk, ebenso wie anderwärts, Kollateralbahnen entwickeln, worauf schon Henckel hinweist. Und dann ist noch ein zweites Moment zu berücksichtigen, auf das auch Henckel aufmerksam macht, daß nämlich degenerative Veränderungen (am eireifenden Parenchym) frühestens ein Jahr nach Entfernung des Uterus festzustellen sind. Es besteht also keine Abhängigkeit von der Gefäßversorgung. Ich meine, daß sich diese Befunde vielleicht durch eine Abhängigkeit der innersekretorischen Funktion des Ovariums von der hypothetischen des Uterus erklären ließen.

Diese Abhängigkeit zeigt sich noch anderwärts. Bei Myomen kommen stark vergrößerte Ovarien vor, und der histologische Befund spricht wohl für eine Steigerung der Sekretion dieser Ovarien. Ich fand in diesen Myomovarien neben stark vergrößerten Blutgefäßen, stark vermehrtem Bindegewebe eine große Zahl von Follikeln in Zysten umgewandelt. Über den Epithelbesatz einzelner dieser Zysten läßt sich streiten, die Begrenzung vieler wird aber von vielen Lagen protoplasmareicher Zellen gebildet, deren Abstammung aus Körnerzellen der Theka unzweifelhaft ist, wie auch die völlige Analogie mit den Follikelluteinzellen im Ovarium Schwangerer. Manche Autoren suchen nun den Grund für diese Steigerung in einer gesteigerten Toxinbildung im Myom; es ist fraglich, ob dieses wirklich zu einem vermehrten Stoffwechsel Anlaß gibt, es liegt meiner Ansicht nach vielleicht näher, die Ursache in einer gesteigerten Sekretion der Uterusschleimhaut zu suchen. Daß bei Myomen die Uterusinnenfläche an und für sich vergrößert ist, brauche ich kaum zu erwähnen, außerdem ist sehr häufig die Schleimhaut mächtiger, und der relative Drüsenreichtum größer, auch ist der Intervall zwischen den Blutungen häufig ein kürzerer, also ein rascheres und stärkeres Ansteigen der Toxizität. Demgegenüber hypertrophiert meiner Vermutung nach das Ovarium, seine stärkere Funktion ist an der starken, langandauernden Blutung zu erkennen. Daß die Ursache nicht im Myom, sondern in der Schleim-

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haut gelegen ist, dafür spricht der zeitweilige Erfolg der Auskratzung; dieselbe kann unmöglich auf die Stoffwechselvorgänge im Myom einen Einfluß ausüben, sondern auf die Schleimhaut selbst, indem diese längere Zeit braucht, um dieselbe Mächtigkeit wie früher zu erlangen. Einen weiteren Beweis für die Abhängigkeit des Ovariums vom Uterus sehe ich in der Verkleinerung der Myomovarien nach Enukleation der Myome.

Man könnte zwar gerade diese Erfahrungstatsache zugunsten der Ansicht Henckels verwerten, daß die Steigerung der Funktion des Ovariums direkt auf die Myome bzw. ihre Kapsel zu beziehen ist. Aber mit Rücksicht auf die oben angeführte Beobachtung hinsichtlich der Auskratzung ist wohl nur die Erklärung zulässig, daß die Ausschälung auf die Uterusschleimhaut einwirkt, wofür wir ja Beweise genug haben, und daß die Rückbildung der Schleimhaut die Rückbildung der Ovarien zur Folge hat. Henckel führt als weitere Stütze seiner Ansicht an, daß Ausfallserscheinungen nach Exstirpation myomatöser Uteri sehr häufig sind, daß er aber nach Exstirpation von karzinomatösen Uteri diese sehr selten beobachtet hat. Demgegenüber ist es aber auffallend, daß wir nach Exstirpation von Uteri aus anderen Gründen, beispielsweise bei Prolaps, Adnextumoren, doppelseitigen Ovarialzysten Ausfallserscheinungen sehr häufig sehen. Es läßt sich daher, glaube ich, aus diesen Beobachtungen nur das eine schließen, daß der Uterus irgendeinen Einfluß auf die Ausfallserscheinungen hat. Des weiteren meint Henckel, daß in den Fällen von Erhaltung der Menstruation durch Konservierung eines Uterusrestes bei gleichzeitig bestehenden Ausfallserscheinungen der vorhandene Uterusrest groß genug war zur Erzeugung der Antikörper, um der bis dahin gesteigerten Ovarialfunktion entgegenzuwirken. Gerade hier zeigt sich die Unzulänglichkeit der Henckelschen Erklärung, da damit die Lehre von der antitoxischen Funktion der Ovarien, die durch zahllose Versuche und therapeutische Anwendung des Ovarialextraktes sichergestellt zu sein scheint, bankrott erklärt wird. Meiner Ansicht nach liegt die Erklärung viel näher, daß das Fehlen der Ausfallserscheinungen in einem Teil der Fälle darauf zurückzuführen ist, daß das Stückchen Uterusrest genügte, um die Ovarialfunktion zu erhalten, daß aber in den Fällen, wo Ausfallserscheinungen auftraten, die Ovarialfunktion nur soweit erhalten blieb, daß es zwar zur Blutung, aber nicht zur genügenden Entgiftung des Organismus kam. Dies stünde in Analogie mit den Ausfallserscheinungen, die wir auch meist gelegentlich der Menstruation beobachteten und auf Hypofunktion des Ovariums zurückführen. Daß meine Erklärung wahrscheinlicher zu sein dünkt, wird auch durch die Beobachtung

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Henckels bestätigt, daß nämlich nach Enukleation von Myomen Ausfallserscheinungen nicht vorkommen. Wenn tatsächlich das Muskelgewebe um die Myome antitoxisch wirkt, müßte der Wegfall dieses Gewebes — nach Henckel wird das Myom mitsamt der Kapsel entfernt — insbesondere bei der gesteigerten Ovarialtätigkeit zu Ausfallserscheinungen führen. Henckel stellt in einer weiteren Arbeit die interstitiellen Myome in Gegensatz zu den subserösen und submukösen und meint von letzteren, daß hier die Menstruation zur regelmäßigen Zeit erlischt und daß ferner hier keine spezifischen Veränderungen an den Ovarien vorkommen. Dem kann ich nicht zustimmen. Unter den 6 von mir untersuchten Myomovarien fand sich 1 bei rein submukösem Myom. Es zeigte ganz dieselben Veränderungen wie die anderen, ganz abgesehen davon, daß im allgemeinen doch die Ansicht vorherrscht, daß bei submukösem Myom die Menstruation nicht zur gesetzmäßigen Zeit erlischt. Ich ging auf diesen Gedankengang Henckels genau ein, da er doch mit ziemlicher Deutlichkeit darauf hinweist, daß dem Uterus eine innere Sekretion zukommen dürfte.

Außerdem möchte ich noch kurz erwähnen, daß sich diese Abhängigkeit des Ovariums vom Uterus auch noch sehr deutlich in der Schwangerschaft dokumentiert, worauf ich in einer anderen Arbeit zurückkommen werde; nur so viel sei hervorgehoben, daß ich dort den Nachweis dafür zu bringen versuche, daß sich das Ovarium in der Schwangerschaft in dem Zustande erhöhter sekretorischer Tätigkeit befinde.

Läßt sich nun die hypothetische sekretorische Funktion des Uterus auch histologisch nachweisen? In der prächtigen Arbeit von Hitschmann und Adler[1]) ist von einer sekretorischen Tätigkeit der Uterusdrüsen im prämenstruellen Stadium die Rede. Dies ist aber eine Sekretion nach außen, die ihren Höhepunkt kurz vor der Blutung erreicht. An ein einfaches Durchwandern des Sekretes durch das Epithel ist wohl nicht zu denken. Die Granulierung und Vakuolisierung des Protoplasmas, die ich auch an analogen eigenen Präparaten nachweisen konnte, läßt sich nur so deuten, daß gewisse Stoffe dem Blute entnommen werden und vermittels der Granula die Permeabilität erlangen. Können wir nun annehmen, daß es sich hier einfach um Ausscheidung blutdrucksteigernder, toxischer Substanzen handelt, die irgendwo im Körper gebildet werden? Also um eine einfache Filtration? Ich glaube, daß damit die Tatsachen nicht übereinstimmen. Aus den Beschreibungen sowohl als auch aus eigenen Befunden geht hervor, daß bereits in der prämenstruellen Zeit die sekretorische

1) Monatsschr. f. Geburtshilfe, Januar 1908.

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Tätigkeit eine ganz bedeutende ist, und gerade zu dieser Zeit steigt der Blutdruck noch immer bedeutend an, gerade zu dieser Zeit zeigen sich Intoxikationserscheinungen. Es wäre nun denkbar — und vielleicht sprechen die Vakuolen an der Basis der Zellen dafür —, daß durch die Tätigkeit dieser Zellen, ähnlich anderen sekretorischen, dem Blute Stoffe entnommen werden, die schließlich zur Ausscheidung kommen, wobei die Schlacken dieses Stoffwechselvorganges das gesuchte Toxin darstellen. Der Höhepunkt der äußeren Sekretion während der Menstruation stellt nach der Beschreibung der Autoren geradezu ein Zugrundegehen dieser Zellen dar. Und so wäre es leicht erklärlich, warum in der prämenstruellen Zeit, wo die sekretorische Tätigkeit der Drüsen einsetzt, ein Ansteigen der Toxizität statthat, und mit dem Höhepunkt der Sekretion, in dem nicht allein relativ viel ausgeschieden wird, sondern die die Toxizität bedingenden Zellen zugrunde gehen, die Toxizität abnimmt, ganz abgesehen von der antagonistischen Wirkung des Ovarialsekretes. Noch ein weiterer Befund an der Uterusschleimhaut fällt auf, und das ist die starke Granulierung der Bindegewebszwischenzellen. Ich will hier nur auf die Versuche Schückings hinweisen, der aus dem Fruchthalter ein Metrotoxin darstellte, das nicht allein giftig wirkte, sondern auch Blutungen erzeugte. Sieht man in der Deciduazelle den Ausdruck für die Hypertrophie der Bindegewebszelle infolge gesteigerter Sekretion, wie in der Follikelluteinzelle die Steigerung der Sekretion der Thekazelle, so könnte man vielleicht auch an eine sekretorische Funktion der Bindegewebszwischenzellen denken, die in der Gravidität zu Deciduazellen hypertrophiert sind. Man würde dann begreiflich finden, warum gerade zur Zeit der Menstruation sich Deciduazellen finden und warum gerade die Dysmenorrhöe mit einer gesteigerten Deciduabildung einhergeht. Man könnte die nervösen Erscheinungen der Dysmenorrhöe als Folge der starken Intoxikation infolge der gesteigerten Sekretion durch Hypertrophie der Bindegewebszellen bis zur Deciduazelle ansehen. Ein sicherer Schluß aus der Histologie der Uterusschleimhaut auf das Bestehen der inneren Sekretion des Uterus läßt sich freilich derzeit nicht ziehen.

Ein Bestätigung dieser Ansicht von der Sekretion des Uterus und weiterhin von der Abhängigkeit der Ovarialsekretion und Ovarialerhaltung von der uterinen Sekretion wäre gegeben, wenn wir ähnliche Befunde beim Manne erheben könnten. Tatsächlich liegen solche Versuchsergebnisse vor. Sarralach und Pares[1]) haben Hunden die Prostata entfernt. Hierauf fehlten alle Sekretionserscheinungen an den übrigen

1) Zur Physiologie der Prostata und des Hodens. Société de biol. de Paris 28. XII. 1907.

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Geschlechtsorganen und es trat Atrophie des Hodens auf. Verfütterte man Glyzerinextrakte der Prostata, so kam es nicht zur Atrophie des Hodens, und auch die Ejakulation war möglich. Spermatozoen zeigten sich im Sperma. Es wäre also dann die Prostata innersekretorisch dem Uterus, die Hoden dem Ovarium gleichzustellen.

Aus allen diesen Erfahrungen ergibt sich die Vermutung, daß der Uterus intern sezerniert, daß dieses Sekret einen Teil jener Toxine bilde, welche das Ansteigen der Welle bewirken, daß das Ovarium entgiftet, und daß zwischen beiden Sekretionen und Organen ein Abhängigkeitsverhältnis besteht. Dies stimmt recht gut mit den Erfahrungen Grammatikatis[1]), daß die Molimina menstrualia nach Zurücklassen der Ovarien mit viel schwereren Erscheinungen einhergehen, daß hier die Störungen viel schwerer sind als nach Kastration. Ähnlich lauten auch die Äußerungen von Fehling[2]) und Alterthum[3]). Aus einer Arbeit von Buschbeck[4]) geht hervor, daß bei Zurücklassen der Ovarien in der ersten Zeit zwar Beschwerden vorhanden waren, daß sie später aber aufhörten. Wenn bei Zurücklassen der Ovarien Störungen vorhanden sind, so kann man dies nur so erklären, daß diese Ovarien, obwohl sie vielleicht äußerlich und auch mikroskopisch nichts Abnormes, insbesondere hinsichtlich der Eireifung (obwohl auch hier gegenteilig zu deutende Befunde vorliegen) boten, doch sekretorisch insuffizient waren, wie ich vermute deshalb, weil die antagonistische Sekretion des Uterus fehlte. Allmählich treten andere Organe für das Ovarium ein, und so wird das Gleichgewicht wieder hergestellt. Auf einige hier nicht zitierte Arbeiten will ich nicht eingehen; sie stützen sich entweder auf anatomische Befunde, wobei das Erhaltensein der Ovulation als beweisend für das Erhaltensein der Sekretion aufgefaßt wird, eine Annahme, der man sicherlich nicht zustimmen kann, zum Teil stützen sie sich auf eine so kleine Zahl von Fällen, daß sie dem Vergleich mit dem großen und sorgfältig beobachteten Material aus der Klinik Schauta nicht standhalten. Und deshalb nahm ich im vorhergehenden insbesondere auf dieses Rücksicht.

Ein Punkt wird zwar vielfach hervorgehoben, aber nicht besonders berücksichtigt, und kommt in den Statistiken nicht zum Ausdrucke: Es ist das Alter der Pat. Es wurde vielfach beobachtet, daß bei älteren Pat. die Kastration zumeist ohne Beschwerden verläuft. Von

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1) Zentralbl. f. Gyn. 1889, VII.
2) Beitr. zur Geburtsh. Bd. 1.
3) Beitr. zur Geburtsh. Bd. 2 und I. D. Freiburg 1895.
4) Arch. f. Gyn. Bd. 56.

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diesem Gesichtspunkte aus sollten ältere Frauen aus den Vergleichsstatistiken ganz ausgeschieden werden, was nirgends geschieht. Obiges Faktum ist natürlich leicht zu erklären; die interne Sekretion bei diesen alternden Organen dürfte eine allmählich abnehmende sein, und andere Organe springen, soweit dies notwendig ist, für sie ein. Wird hier kastriert, so werden minderwertige, mehr oder minder bereits nebensächliche Organe entfernt, die Ausfallserscheinungen fehlen oder sind sehr gering.

Von neueren Arbeiten sei zunächst die von Burckhard[1]) erwähnt. Zurücklassung der Ovarien macht das Auftreten der Molimina häufig (22 %), ändert nichts an der Häufigkeit der Ausfallserscheinungen. Für uns ist besonders bemerkenswert, daß die supravaginale Amputation ungünstigere Resultate in bezug auf die Ausfallserscheinungen gibt als die Totalexstirpation, also völlige Übereinstimmung mit den Schlüssen, die ich oben aus Tabelle II der Arbeit von Mandl und Bürger gezogen habe.

Aus der Arbeit Werths[2]) sei folgendes hervorgehoben. Bei Mitnahme der Ovarien in 84% Ausfallserscheinungen. Bei supravaginaler Amputation mit Belassung der Ovarien in ca. 65% Ausfallserscheinungen. Die nach 3 Jahren untersuchten Fälle ergaben aber in 85% Ausfallserscheinungen. Die geringe Verminderung der Zahl der Ausfallserscheinungen kurz nach der Operation läßt sich leicht dahin erklären, daß entsprechend der relativ geringeren Sekretion aus dem Uterusstumpf die Ovarialsekretion sich zunächst mitunter als suffizient erwies; später reichte entweder die Sekretion des Uterus nicht mehr aus, um die der Ovarien zu erhalten, oder die Sekretion des Uterusstumpfes sistierte, wodurch es auch zum Funktionsstillstand des Ovarium kam, und so traten Ausfallserscheinungen auf und zwar in gleicher Häufigkeit wie bei Mitnahme der Ovarien. Daß in 6 Fällen Menstruationsblutungen bei gleichzeitigen Ausfallserscheinungen bestanden, spricht wohl zugunsten der hier vertretenen Theorie, daß die Sekretion der Ovarien abhängig ist von der Sekretion des Uterus. Da letztere hier nur eine geringe ist, kann nach der hier vertretenen Theorie erstere auch nur gering sein, und es fehlt daher oft eine ausreichende Entgiftung. Die übrigen hier nicht erwähnten Arbeiten (s. diesbezüglich das Buch von Mandl und Bürger) geben zu ähnlichen Schlüssen Anlaß wie die zitierten.

Was die Zeit des Eintrittes der Ausfallserscheinungen anlangt, so ist es klar, daß bei Mitentfernung der Ovarien, dem plötz-

1) Zeitschr. f. Geburtsh. Bd. 43.
2) Klin. Jahrb. 1902, Jena.

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lichen Wegfall eines entgiftenden Organes, die Ausfallserscheinungen zumeist sofort einsetzen (48%), während bei Belassung erst die allmählich einsetzende sekretorische Insuffizienz der Ovarien die Erscheinungen entstehen läßt (30% gleich nach der Operation).

Nur nebenbei sei erwähnt, daß die viel zitierte Statistik eine Abhängigkeit der Atrophie des äußeren Genitals von der Sekretion kaum erkennen läßt. Wenn die Atrophie bei Belassung der Ovarien in 25%, sonst in 36% auftritt, so liegt es wohl näher an mangelhafte Ernährung infolge der schlechten Gefäßversorgung zu denken. Die Atrophie müßte sonst doch häufiger sein.

Einzelne Details in der Menstruationstheorie blieben noch zu erläutern. Wodurch wird die Blutung hervorgerufen, durch das hypothetische Uterus- oder Ovarialsekret? Ich meine durch letzteres, welches Störungen im Uteruskreislauf zu erzeugen imstande ist. Ich verweise diesbezüglich auf die Beobachtungen Gellhorns, der durch Ovarialtabletten Blutungen hervorrief, ebenso auf die Erfahrungen van de Veldes. Daß es sich hier nur um Blutüberfüllung handelt, zeigt die Mitteilung Temesvarys[1]), die sich auf viele Erfahrungen anderer Autoren stützt, daß man nämlich durch Senfkataplasmen auf die Brust die Menstruation (sc. Blutung) hervorrufen könne. Ich verweise auf die schon erwähnten Versuche von Ficarelli und Holterbach und jene von Deales, Hervorrufung der Brunst durch Yohimbin betreffend. Von Interesse ist auch der Fall von Blondel und Sendral[2]). Nach Verlust der Menses kam es zu einem Glaukom, das auch die Iridektomie nicht beseitigen konnte. Durch Ovarienkapseln, Apiol, Aloe und Eisentartarat gelang es die Menses hervorzurufen und das Glaukom zu heilen. Hierher gehören auch wohl die Erfolge der Organotherapie bei Chlorose.

Auch die langdauernden Blutungen bei Myom lassen sich auf die verstärkte Sekretion der Myomovarien zurückführen. Es könnte zwar auch die vermehrte Sekretion der hyperplastischen Uterusschleimhaut die starken Blutungen verursachen; aber Röntgenbestrahlungen der Ovarien bei Myom mit ihrer guten, sofortigen Wirkung auf die Blutungen (Faveau de Courmelles, M. Fränkel usw.) sprechen wohl entschieden für die Anschauung, daß es das Ovarialsekret ist, das die Blutungen erzeugt. Es sind überhaupt die Resultate der Röntgenbestrahlungen, die mit aller Deutlichkeit darauf hinweisen, daß das Ovarialsekret die Blutungen hervorruft. Man könnte zwar bei obigen Versuchen an Myomovarien meinen, daß die Verringerung der Sekretion der Ovarien auf die Uterusschleimhaut einwirke, und

1) Journ. of obstetr. 1903.
2) La Gynéc. Févr. 1904.

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daß so erst die Blutungen beeinflußt werden. Aber solche Rückbildungen der Uterusschleimhaut — und nur darum könnte es sich handeln — auf Grund des verringerten trophischen Einflusses der Ovarien brauchen erfahrungsgemäß erst längere Zeit, die Schwächung der Blutung tritt aber sofort ein. Und so möchte ich wohl annehmen, daß die Blutung durch das Ovarialsekret hervorgerufen wird.

Nur die obenerwähnten Versuche von Schücking scheinen dagegen zu sprechen; denn das Metrotoxin erzeugt auch Blutungen. Vielleicht erfolgt die Blutung auf dem Umwege über die Ovarien, indem durch das Metrotoxin das Ovarium zur Sekretion angeregt wird, und so die Blutung einsetzt; ebenso wie ich mir vorstelle, daß durch die starke Vergiftung des Organismus von seiten des hypothetischen Uterussekretes die Sekretion des Ovariums maximal gesteigert wird und die Blutung erzeugt.

Nicht zu entscheiden ist derzeit die Frage, ob die interstitiellen Zellen des Ovariums oder die Luteinzellen die Träger der Sekretion sind. Es scheint doch ein kleiner Unterschied zwischen diesen Zellen zu bestehen. Denn Lampert[1]) beobachtete eine hohe Giftigkeit des Extraktes der gelben Körper, eine Giftigkeit, welche die Eierstöcke ohne gelben Körper nicht besitzen. Für die Abhängigkeit der Menstruation von den Luteinzellen sprechen neuerdings die Untersuchungen von P. Ancel und F. Villemin[2]). Sie fanden eben gesprungene Follikel nur bei Frauen, welche 14 Tage vorher die Regel hatten, wo also die nächste Periode in 12 Tagen hätte stattfinden sollen. Der gelbe Körper bilde sich zur Zeit der Periode aus und degeneriere unmittelbar nachher. Zu ähnlichen Resultaten führen die beiden Autoren auch Versuche mit Ektopie des Ovariums[3]), Versuche, die freilich nicht ganz einwandsfrei sind, ebensowenig wie die Deutung. Die Bemerkung, daß die Zellen der Glande interstitielle keine Veränderung zeigten, ist wohl absolut nicht verwertbar. Zu anderen Schlüssen kommen Leopold und Ravano[4]). Es kann (freilich größtenteils auf Grund makroskopischer Beobachtungen) Ovulation ohne Menstruation vorkommen und Menstruation ohne Ovulation. Die letztere fand in mehr als einem Drittel der Fälle nicht gleichzeitig mit der Menstruation statt. Immerhin ergibt sich meines Erachtens doch in der Mehrzahl der Fälle eine gewisse Abhängigkeit der Menstruation von der Ovulation, andererseits neige ich zu der Ansicht, daß die interstitiellen Zellen gleichfalls innersekretorische

1) Comptes rendus de la Soc. de biol. Janv. 1907.
2) Soc. de biol. Paris Juillet 1907.
3) Soc. de biol. Paris Août 1907.
4) Soc. de biol. Paris Juillet 1907.

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Kraft besitzen, vielleicht in gleichem Sinne, aber in relativ vermindertem Maße wie die Luteinzellen. Ich verweise diesbezüglich und hinsichtlich der sonstigen Literatur auf meine anderen einschlägigen Arbeiten[1]). Auch die Untersuchungen von Regaud und Dubreuil (Soc. de biol. de Paris Févr. 1908) können mich nicht vom Gegenteil überzeugen. Insbesondere auf Grundlage des makroskopischen Aussehens und des Gewichtes der Ovarien kommen die beiden Autoren zu dem Schlusse, daß zwischen der Glande interstitielle und der Brunst ein Zusammenhang nicht bestehe. Es handelt sich hier jedenfalls um Sekretionserscheinungen, und diese lassen sich wohl zur Zeit der Brunst und kurz nachher nicht mehr nachweisen.

Ich würde daher der Ansicht zuneigen, daß die Entgiftung des Organismus durch die innere Sekretion der interstitiellen Zellen und der Luteinzellen erfolgt. Die Tätigkeit der interstitiellen Zellen dürfte eine geringe sein und während der ganzen Dauer des Intervalls bestehen, da diese Zellen zur Zeit der Menstruation nicht wesentlich anders aussehen als sonst. Vielleicht erfährt diese Sekretion zur Zeit der Menstruation eine Steigerung, doch fehlt hiefür noch jeder histologische Beweis; nur der Umstand, daß es eine Menstruation ohne Eireifung gibt (Leopold und Ravano, Hensen, Müller u. a.) spricht für diese Annahme. Außerhalb der Menstruation ist natürlich diese sekretorische Tätigkeit der interstitiellen Zellen sehr gering, sie vermag der vermuteten Tätigkeit des Uterus nicht kräftig entgegenzuarbeiten. Unabhängig von diesen Vorgängen reifen Eier. Hat ein Follikel eine entsprechende Größe erreicht, so springt er, die Granulosazellen, welche das Ei bisher ernährt haben, gehen zugrunde, und der starke Blutzufluß zum Follikel, der bisher von den Granulosazellen absorbiert wurde, führt zur Hypertrophie der Thekazellen; die Luteinzellen sezernieren und vermehren sich, sobald die maximale Toxizität des Blutes ihre Sekretion anregt, also zur prämenstruellen Zeit, kurz nachher degenerieren sie. Diese Theorie ist also nicht an eine bestimmte Zeit des Follikelsprunges gebunden und trägt der Erfahrung Rechnung, daß der Follikelsprung zumeist vor

1) O. O. Fellner, Neuere Ergebnisse aus den Forschungen über das Corpus luteum. Med. Klinik 1906, 42. — O. O. Fellner und F. Neumann, Über Röntgenbestrahlung der Ovarien in der Schwangerschaft. Zentralbl. f. Gyn. 1906, 22. Der Einfluß der Röntgenstrahlen auf die Eierstöcke trächtiger Kaninchen und auf die Trächtigkeit. Zeitschr. f. Heilkunde Bd. 28, H. 7. — Über den Einfluß des Cholins und der Röntgenstrahlen auf den Ablauf der Gravidität. Münchener med. Wochenschr. 1907, 23. Ferner meine demnächst erscheinenden Arbeiten: „Über die Tätigkeit des Ovariums in der Schwangerschaft" und „Zur Histologie des Ovariums in der Schwangerschaft".

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der Periode erfolgt. Auch dieser Umstand ist nunmehr leicht zu erklären, da die zunehmende Sekretion der interstitiellen Zellen zu einer zunehmenden Blutfülle der Ovarien und somit zu einer allmählich sich steigernden besseren Ernährung des Eies führt.

Sollte sich die Annahme einer Uterussekretion weiterhin bestätigen, so ergibt sich folgende Theorie der Menstruation, die freilich noch einiger starker Stützen bedarf: Durch die innersekretorische Tätigkeit des Uterus kommt es zu einer allmählich sich steigernden Vergiftung des Organismus, welche wahrscheinlich die Sekretion der interstitiellen Ovarialzellen steigert, welche Steigerung wieder zu einer stärkeren Blutfülle in den Genitalgefäßen und somit zur Reifung der Follikel führt, und ihr Maximum vor der Periode erreicht, demnach die Berstung des Follikels gewöhnlich vorher eintreten läßt. Durch den Wegfall des Eies werden die Thekazellen besser ernährt und entwickeln sich zu Luteinzellen. Die maximale Vergiftung des Organismus löst eine starke Sekretion, vielleicht der interstitiellen Zellen, sicher aber der Luteinzellen aus. Diese Sekretion wieder entgiftet nicht allein den Organismus durch ihre antagonistische Wirkung, sondern führt auch zu Blutungen aus dem Genitale, wodurch ein weiterer Faktor der Entgiftung gebildet wird. Schließlich löst sie auch die äußere Sekretion der inzwischen herangewachsenen Drüsen aus. Was hier ausgeschieden wird, ist fraglich. Mag sein das uterine Sekret, ein weiterer Faktor der Entgiftung, mag sein das ovarielle Sekret; vielleicht auch beide. Daß ovarielles Sekret auch mit sezerniert wird, dafür spricht der Umstand, daß wie Bell[1]) nachgewiesen hat, durch den Uterus Kalk ausgeschieden wird, und andererseits die Tätigkeit des Ovariums mit Kalkausscheidung einhergeht.

Dies ist die Theorie, die sich auf Grund eigener Untersuchungen und der bisherigen Forschungsergebnisse ergibt. Manches bedarf natürlich noch sehr der Bestätigung, andererseits kann jede neue Tatsache die Theorie ganz oder zum Teil umstürzen, aber soweit die heutigen Kenntnisse reichen, scheint sie meiner Ansicht nach alles zu erklären.

II. Die innersekretorischen Organe und das Ovarium.

Um das Verhältnis der innersekretorischen Organe zum Ovarium verstehen zu können, muß man sich vorerst über das Verhältnis der einzelnen Organe zueinander im allgemeinen klar sein. Bei der

1) Brit. med. Journ. 1907, 2716.

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genauen Durchsicht der Literatur drängen sich folgende Leitsätze auf, deren Richtigkeit durch die Literaturangaben bewiesen werden soll.

Wir haben im wesentlichen 3 Paare von sekretorischen Drüsen zu unterscheiden: Ovarien und Uterus, Schilddrüse und Nebenniere (es dürften da antagonistisch noch andere Drüsen in Betracht kommen, doch erwähne ich der Einfachheit halber nur die bedeutendste) und die Hypophysis. Letztere sezerniert sowohl blutdrucksteigernde als auch blutdrucksenkende Substanzen, doch überwiegt zumeist die erstere, wie in allen anderen Paaren. Die Tätigkeit dieser Drüsen ist wohl so aufzufassen: Im Organismus gehen eine Reihe von Stoffwechselvorgängen vor sich, unter denen giftige, blutdrucksteigernde das Übergewicht haben. Um hier regulierend einzuwirken, sind bei den höheren Tieren und beim Menschen innersekretorische Organe eingeschaltet, die diese Stoffe unschädlich machen, was insbesondere hinsichtlich der Psyche und der Intelligenz von Wichtigkeit ist, die aber gleichzeitig den Blutdruck herabsetzen. Da dies aber natürlich das Leben gefährden würde, sind noch blutdrucksteigernde Organe vorhanden. Schaltet man ein blutdrucksteigerndes Organ aus, so müßten natürlich die anderen hypertrophieren. Hier fehlen größtenteils entsprechende Versuche, und hinsichtlich des inneren Genitales muß man auf die hier herrschenden speziellen Verhältnisse Rücksicht nehmen, da Exstirpation der Ovarien zur Atrophie des Uterus führt, und wahrscheinlich umgekehrt Exstirpation des Uterus mit der Zeit häufig die sekretorische Insuffizienz oder allgemeine Atrophie der Ovarien zur Folge hat, wie ich oben ausgeführt habe.

Steigerung der Tätigkeit eines blutdrucksteigernden Organes hat verstärkte Tätigkeit nicht allein des zugehörigen antagonistischen Organes, sondern aller anderen zur Folge z. B. Myom—Myomovarium (Metrorrhagien) — Struma—Myomherz, unter der Voraussetzung, daß obige Hypothese betreffs der Uterussekretion richtig ist. Steigerung der Tätigkeit eines entgiftenden Organes kann Steigerung aller blutdrucksteigernden und konsekutiv auch aller entgiftenden Drüsen zur Folge haben: Basedow — verstärkte Menstruation. Doch ist hier zu bedenken, daß die Steigerung der sekretorischen Tätigkeit der Schilddrüse die Entgiftung des Organismus konstant in so starkem Maße besorgt, daß die Tätigkeit des Ovariums kaum angeregt wird, und es daher zum Ausbleiben der Menstruation und schließlich zur Atrophie des Genitales kommen kann. (S. weiter unten.) Und so sehen wir Basedow bald mit verstärkter Blutung, bald mit Atrophie der Genitalien einhergehen. Wegfall oder Verminderung der Tätigkeit eines entgiftenden Organes führt zur kompensatorischen Hypertrophie der anderen entgiftenden Drüsen — Kastration (Klimax) — Struma.

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Fällt die Tätigkeit eines entgiftenden Organes zur Zeit fort, in der die Sekretion der Genitalien noch keine Rolle spielt, so können diese nicht hypertrophieren. Die gleichzeitige Hypertrophie der anderen entgiftenden Organe und die konsekutive Verminderung der Sekretion der antagonistischen Drüse macht wahrscheinlich die Sekretion der Genitaldrüse, welche später einsetzen sollte, überflüssig. Es kommt zur Atrophie des Genitales.

Vergleicht man die physiologischen Wirkungen des Uterus- und Ovarialsekretes, so stößt man auf große Schwierigkeiten, da die verschiedenen Literaturangaben insbesondere hinsichtlich der Ovarien zumeist diametral entgegengesetzt sind. Es ist dies auch nach dem obenerwähnten leicht verständlich. Bei der Unkenntnis einer innersekretorischen Tätigkeit des Uterus wurde manches auf die Ovarien bezogen, was vielleicht auf den Ausfall beider Organe oder nur des Uterus zu beziehen ist. Ich will mich im folgenden bemühen, mich auf die Angaben zu stützen, die verwertbar sind, das sind die Erscheinungen nach Einverleibung von Ovarialsubstanz, oder unmittelbar nach der Kastration. Zwischen Tyreoidea und Ovarien herrscht eine gewisse Übereinstimmung der Funktionen. Thyreoidea steigert den Sauerstoffverbrauch, jedoch bei weitem weniger als Ovarialextrakt (Perrin und Blum[1]). Thyreoidea verringert den Fettgehalt des Organismus, Kastration und Klimakterium gehen mit starker Fettbildung einher. Thyroidea setzt den Blutdruck herab[2]) und ebenso das Ovarium. Schilddrüse begünstigst das Haarwachstum, ebenso die Hoden. Thyreoidea erzeugt Drucksteigerung der Diurese[3]), Senator, Parkon und Papinian sahen Vermehrung der Ausscheidung der Urate nach Darreichung von Eierstockspräparaten, Verminderung nach Entfernung der Eierstöcke, das gleiche beobachtete Jeandelize nach Thyreoidektomie. Thyreoidea vermindert die Zurückhaltung das Kalziums (Moraczewski, Parkon und Papinian), ebenso das Ovarium. Erweiterung der Gefäße durch Schilddrüse und Eierstock (Hallion[4]). Was die Ausscheidung der Phosphate anlangt, so wird diese durch Darreichung von Ovarin vermehrt (Caratullo und Tarulli, Gomez), durch die Kastration vermindert, ebenso ist sie bei Myxödem vermindert, nur Jeandelize fand eine Vermehrung der Phosphate nach Thyreoidektomie. Worauf dieser Widerspruch beruht, ist mir

1) Rev. méd. de l'est Janv. 1906.

2) Oliver und Schata, Haskoock, Gley, Langlois.

3) Lobenstine, Bull. of the lying in hosp. of the City of New York. Dec. 1905.

4) Soc. de biol. de Paris Juillet 1907.

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nicht klar. Was den Einfluß auf das Knochensystem betrifft, so sehe ich auch hier völlige Übereinstimmung: ich komme darauf später zurück.

V. Eiselsberg fand, daß junge Tiere nach Schilddrüsenexstirpation atrophische Genitalorgane bekamen. Hofmeister beobachtete nach Entfernung der Schilddrüse Frühreife der Follikel. Lanz berichtet, daß Böcklein wie Zicklein in der ersten Jugend tyreoidektomiert die Fortpflanzungsfähigkeit absolut einbüßen; im fortpflanzungsfähigen Alter thyreoidektomierte Tiere können die Fortpflanzungsfähigkeit völlig verlieren. Hierher gehören auch die Versuche von Bleibtreu, der nach Bestrahlung der Schilddrüse Rückgang der Trächtigkeit beobachtete, ebenso wie Neumann und ich nach Bestrahlung der Ovarien [1]).

Ob die frühzeitige Follikelreifung als Versuch einer kompensatorischen Hypertrophie, der alsbald aufgegeben wurde, anzusehen ist, will ich dahingestellt sein lassen, sicherlich geht aus allen anderen Versuchen hervor, daß Verlust der Schilddrüsenfunktion in früher Jugend mit Atrophie der Ovarien einhergeht, entsprechend der obigen Deutung. Hinsichtlich der Einbuße der Fortpflanzungsfähigkeit bei älteren Tieren und hinsichtlich des Rückgangs der Trächtigkeit sei auf eine andere Arbeit (Tätigkeit der Ovarien in der Schwangerschaft) verwiesen.

Daß umgekehrt Kastration Hypersekretion der Schilddrüse zur Folge hat, ist bereits erwähnt. So beobachteten Parkon und Goldstein [2]) eine 21jährige Frau, die 6 Monate nach der Kastration eine Vergrößerung der Schilddrüse auf das Dreifache aufwieß. Perrin und Blau [3]) berichten über einen Fall von Morbus Basedowii, der nach Kastration aufgetreten war, und durch Darreichung von Ovarienextrakt zurückging. Texione [4]) fand, daß Exstirpation der Eierstöcke bei Hunden zur vermehrten Tätigkeit der Thyreoidea, schließlich aber zur Atrophie führt. Von großem Interesse ist ein Fall von Grimsdale. Es trat erhebliche Besserung des bestehenden Morbus Basedowii nach

1) O. O. Fellner und F. Neumann, Der Einfluß der Röntgenstrahlen auf die Eierstöcke trächtiger Kaninchen und auf die Trächtigkeit. Zeitschr. f. Heilkunde Bd. 28, H. 7; Dieselben, Über den Einfluß des Cholins und der Röntgenstrahlen auf den Ablauf der Gravidität. Münchner med. Wochenschr. 1907, 10; Über Röntgenbestrahlung der Ovarien in der Schwangerschaft. Zentralbl. f. Gyn. 1906, 22.

2) Spitalul 1907, 22.

3) Revue méd. de l'est Janv. 1906.

4) North of Engl. obstetr. Soc. May 1904.

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doppelseitiger Salpingo-Oophorektomie auf, die wegen schwerer Dysmenorrhöe ausgeführt wurde. Die Struma blieb. Ausfallserscheinungen stellten sich nicht ein. Wir haben also ursprünglich eine Störung des sekretorischen Gleichgewichtes gehabt, nämlich eine übermäßige Funktion der entgiftenden Organe, insbesondere der Schilddrüse, aber auch des Ovariums (Dysmenorrhöe!); der Wegfall eines Organes führte zur besseren Bilanzierung, also zur erheblichen Besserung der Symptome; die Struma blieb, denn das Ovarium wurde durch die Schilddrüse ersetzt, so gut, daß Ausfallserscheinungen fehlten. Cessa[1]) beobachtete gleichfalls Hypertrophie der Schilddrüse nach Abtragung der Testikel wie der Ovarien, ebenso Mathieu das Auftreten von Basedow nach Kastration.

Über die Steigerung der ovariellen Sekretion (Osteomalazie) wird in einer anderen Arbeit gesprochen werden. Doch sei hier auf das Anschwellen der Schilddrüse während der Menstruation hingewiesen, wobei es bis zum Ausbruch von Basedowsymptomen kommen kann, wie dies Hermann Cohn in seiner Dissertation beschreibt.

Bei Hyposekretion des Ovariums müssen entsprechend den obigen Auseinandersetzungen Störungen ähnlich den Basedowsymptomen auftreten, da die Schilddrüse zum Teil die Aufgabe des Ovariums übernimmt. Tatsächlich berichtet Dalché[2]) über solche Krankheitsfälle, die mit vasomotorischen Störungen und solchen nervöser und dyspeptischer Natur einhergingen. Die Behandlung mit Ovarialtabletten gab gute Resultate.

Auch die Chlorose wird als eine Hyposekretion des Ovariums aufgefaßt (Prenant, Charrin, Spillmann und Etienne und Demange[3]). Ferner sei auf die Untersuchung von Laclère und Levat, die eine Erhöhung der Toxizität des Blutserums Chlorotischer fanden, hingewiesen. Von besonderem Interesse sind die Versuche von R. Breuer und von Seiler[4]). Bei Kastration nimmt die Anzahl der roten Blutkörperchen und die Menge des Hämoglobins ab. Wird hernach der Uterus entfernt, so steigt die Zahl der Blutkörperchen und die Menge des Hämoglobins wieder. Dieses Ergebnis stimmt recht gut mit dem oben vermuteten Antagonismus zwischen Ovarien und Uterus. Dementsprechend hatte die ovarielle Therapie ziemlich günstige Erfolge (Muret, Spillmann und Etienne, Jacobs, Jayle). Es muß ferner bei Chlorose Hypertrophie der Schilddrüse nicht

1) Soc. méd. de Boul. Mars 1904.
2) Gazette des hôp. Juillet 1908.
3) Thèse de Nancy 1898.
4) Gesellsch. der Ärzte, Wien. Juli 1903.

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selten sein (Hayem, Perrin und Blum[1]). Auch die bei Chlorose auftretende Neuritis optica kann nach Berger und Loewy[2]) auf die Hypersekretion der Schilddrüse zurückgeführt werden, da Coppez (Soc. Belge d'ophthalmologie 1903) als Folge von starker medikamentöser Darreichung von Extrakt der Thyreoidea das Auftreten von Neuritis optica beobachtete.

Hierher gehören auch die Fälle von Basedowscher Krankheit, die während des Klimakteriums entstanden sind, so 3 Fälle von Parisot (Bull. méd. Janv. 1906).

Wenn auch manche Autoren die Basedowsche Krankheit mit einer Degeneration der Schilddrüse in Zusammenhang bringen wollen, so fehlen doch hierfür strikte Beweise, und wir wollen in folgendem daran festhalten, daß diese Krankheit mit einer Steigerung der Sekretion der Schilddrüse einhergeht. Über das Inkonstante des Befundes an den Genitalien und die Erklärung hierfür habe ich mich schon oben geäußert. Kleinwächter[3]) hat Atrophie der Genitalien mit Ausnahme des Uterus beobachtet; gerade dieses Erhaltenbleiben des Uterus stimmt recht gut mit obiger Theorie. Sänger konnte zwar einen derartigen Zusammenhang nicht finden.[4]) Daß tatsächlich häufig weder eine Atrophie des Uterus, noch zum mindesten eine solche des eireifenden Parenchyms auftritt, beweisen die Fälle von Schwangerschaft bei Morbus Basedowii trotz Ausbleibens der Regel, wie ich[5]) einen solchen publiziert habe. Unter 72 Kranken Kochers hatten nur 3 Patientinnen normale und starke Regeln. Von 8 Fällen Wilsons (Lancet Nov. 1906) war bei dreien die Blutung eine schwache oder fehlte ganz, während 2 sehr starke Blutungen hatten. In einem dieser Fälle ging eine Atrophie des äußeren und inneren Genitales mit dem Ausbleiben der Menstruation und dem Auftreten der Basedowsymptome einher.

Gerade solche Fälle lassen die Vermutung aufkommen, daß mitunter vielleicht das Ausbleiben der Sekretion des Ovariums das Primäre und die Basedowsymptome das Sekundäre sind. Denn tatsächlich kommen ja in anderen Fällen stärkere Blutungen vor (Branwell, Simpson). Langdauernde Blutungen (Gillebert d'Hercourt, Finck, Cheadle, Haby, Kelly, Tillaux, Collin, Sansom, Sallier), ja

1) Rev. méd. de l'est Janv. 1906.

2) Über Augenerkrankungen sexuellen Ursprungs bei Frauen. Wiesbaden, Bergmann, 1906.

3) Zeitschr. f. Geb. 1889, 16.

4) Geb. Gesellsch. Leipzig, Mai 1889.

5) O. O. Fellner, Die Beziehungen innerer Krankheiten zu Schwangerschaft usw. Wien, Deuticke, 1903.

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selbst Dysmenorrhöe sind, beispielsweise von Wilson publiziert. Im übrigen möchte ich nochmals auf das oben Gesagte hinweisen, daß Basedow höchstwahrscheinlich deshalb zum Ausbleiben der Menstruation führt, weil die Hypersekretion der Schilddrüse eine kontinuierliche, so starke Entgiftung des Organismus herbeiführt, daß es nicht zur Auslösung einer genügenden Sekretion des Ovariums kommt (normaler Uterus). Daß diese Inaktivität schließlich zur Atrophie führt, ist selbstverständlich.

Der Wegfall der Tätigkeit der Schilddrüse — Myxödem — zu einer Zeit, wo die Genitalien noch keine Rolle spielen, führt, wie bereits oben erwähnt und erklärt, zu Aplasie der Keimdrüsen.

Was die Dercumsche Krankheit betrifft, welche von Sicard und Russy[1]) nach Ovariotomie beobachtet wurde, so können die Erscheinungen wohl auf den Ausfall der Eierstöcke bezogen werden. Loening[2]) denkt an Schilddrüsenveränderungen. Bei der Seltenheit dieser Erkrankung könnte man sich das Entstehen derselben so erklären, daß in diesen Fällen die Schilddrüse von vornherein nicht imstande war, die sonst zumeist auftretende Hypersekretion nach Exstirpation der Ovarien zu leisten und daher atrophierte.

Es wäre schließlich auch noch auf die Erfolge der Opotherapie[3]) hinzuweisen. Es ist hier darauf Rücksicht zu nehmen, daß es fast gleichgültig zu sein scheint, von welchem entgiftenden Organ man bei Steigerung einer blutdrucksteigernden Sekretion oder bei Ausfall bzw. Verminderung einer entgiftenden Sekretion das Präparat benützt. Darauf wurde zu wenig Rücksicht genommen, und es wurden oft aus den Erfolgen der Opotherapie direkte Schlüsse auf die Erkrankung jenes Organs gezogen, von dessen Darreichung man Erfolge sah; und dies wird man sicherlich bei Berücksichtigung der hier vertretenen Theorien für nicht zutreffend finden. Wenn Amenorrhöe bei fetten Personen durch Schilddrüse günstig beeinflußt wird (Philipps[4]), so muß diese Amenorrhöe nicht in der Schilddrüse ihre Ursache haben, sondern es wird gleichzeitig eine Steigerung der Schilddrüsenfunktion und somit entsprechend den obigen Theorien eine Steigerung der Sekretion aller entgiftenden Drüsen herbeigeführt. Wenn Blutungen bei Darreichung von Schilddrüse mitunter geringer werden, sobald es sich um Myome handelt, so müssen wir berücksichtigen, daß durch

1) Soc. méd. des Hôpitaux de Paris Oct. 1903.

2) Kongreß f. innere Med. München, April 1906.

3) Siehe auch O. O. Fellner, Die Opotherapie in der Gynäkologie. Therap. Rundschau 1907, 29.

4) Lancet Mai 1901.

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die reichliche Zufuhr entgiftenden Sekretes die durch die freilich hypothetische Hypersekretion der myomatösen Uterusschleimhaut herbeigeführte Intoxikation nicht jene Höhe wie sonst erreicht, und infolgedessen auch die Blutung eine geringere ist. Auch Perlsee[1]) hatte mit Schilddrüse bei abnormer menstrueller Blutung guten Erfolg.

Aus dieser Zusammenstellung ergibt sich eine völlige Analogie der Funktionen des Ovariums und der Schilddrüse. Von einem Antagonismus, wie ihn manche Autoren annehmen, kann wohl keine Rede sein.

Bezüglich der Hypophysis habe ich bereits oben erwähnt, daß ich mit Vincent[2]) annehme, daß sie zwei verschiedene Stoffe sezerniert, einen in Alkohol und Äther unlöslichen blutdrucksteigernden und einen löslichen blutdrucksenkenden; für gewöhnlich überwiegt ersterer. Wir haben es also mit der Vereinigung eines entgiftenden Organes und seines Antagonisten zu tun. Dementsprechend hat nach Mislawski Reizung der Seehügel eine starke Blutdrucksteigerung, Pulsverlangsamung, Stillstand der Herztätigkeit und Veränderung der Atmung zur Folge. Dieser Anschauung braucht es nicht unbedingt zu widersprechen, wenn durch Darreichung von Hypophysenextrakt die Phosphorausscheidung und ebenso die Stickstoffausscheidung vermehrt wird, freilich beide nur in unbedeutendem Maße; denn es ist zu bedenken, daß hier zwei Antagonisten verabreicht werden.

Nach Rogowitsch[3]) tritt die Hypophysis vikariierend für die entfernte Schilddrüse ein. Dies schließt man aus der Vergrößerung derselben. Es kann sich dabei selbstverständlich nur um jene Teile der Schilddrüse handeln, die entgiftender Natur sind. Zerstörung der Hypophysis hat nach Vassale und Sacchi dieselben Erscheinungen wie die Entfernung der Schilddrüse zur Folge; auch hier müssen wir auf den Wegfall des entgiftenden Teiles des Organes Rücksicht nehmen. Andererseits hat, wie Fichera angibt, Entfernung der Keimdrüse Vergrößerung der Hypophysis zur Folge, ganz entsprechend den obigen Leitsätzen. Tandler und Groß bestätigen diese Angabe. Es ist interessant, daß, wie die beiden Autoren angeben, diese Vergrößerung in einem Falle keine akromegalischen Symptome zur Folge hatte, wohl der beste Beweis dafür, daß es sich um eine vikariierende Vergrößerung handelt.

Akromegalie nennt man eine Erkrankung, die mit einer Vergrößerung der Hypophysis einhergeht, und welche heute zumeist auf

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1) Prager med. Wochenschr. 1906, Bd. 24.
2) Journ. of exper. Med. 1898.
3) Zieglers Beiträge 1889.

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eine Überfunktion der Hypophysis zurückgeführt wird. Hierfür sprechen auch jene Fälle, wo nach Entfernung des Tumors die Erscheinungen der Akromegalie schwanden (Hochenegg). Es fragt sich nun, ob die Akromegalie mit einer Überfunktion des entgiftenden oder des blutdrucksteigernden Teiles des Gehirnanhangs zusammenhängt. Man sah gute Erfolge mit Schilddrüsenbehandlung (Fröhlich, v. Frankl-Hochwart). Dies würde dafür sprechen, daß in diesen Fällen die blutdrucksteigernde Komponente vermehrt ist. Doch ist, wie Bartels[1]) erwähnt, zu bedenken, daß die Besserung gerade in solchen Fällen (Berger, Bartels) dann eintritt, wenn das Präparat nicht mehr verabreicht wird. Da das nicht in allen Fällen vorkommt, so könnte man sich die Besserung so erklären, daß durch die Darreichung des Schilddrüsenextraktes die antagonistische Komponente, also die blutdrucksteigernden Drüsen zur Tätigkeit allmählich angeregt werden, und daß die überdauernde Überfunktion dieser Drüsen die Besserung herbeiführt; es wäre dann die Akromegalie die Folgeerscheinung einer Vergrößerung der Hypophysis, die vor allem auf einer vermehrten Funktion der blutdrucksenkenden Komponenten beruht. Schwer zu erklären ist dann, wieso die Darreichung von Hypophysensubstanz zu einer Besserung der Symptome führt (Axenfeld, Elschnig, Fleischl, Uthof). Man müßte sich dann vorstellen, daß in diesem Tumor das ganze blutdrucksteigernde Gewebe zugrunde gegangen ist, und daß die Zuführung der entgiftenden Substanz durch Hypophysenextrakt zur Herbeiführung einer Entgiftung genügt, was nicht sehr plausibel erscheint, wie denn überhaupt die Erklärung der therapeutischen Erfolge auf Grund der vorerwähnten Annahmen der Schwierigkeiten genug bietet.

Geht man aber, was logischer zu sein scheint, von der letzteren Erfahrung aus, so ist anzunehmen, daß bei der Vergrößerung der akromegalischen Hypophysis das blutdrucksteigernde Gewebe zugrunde gegangen ist, und daher die Zuführung von Hypophysenextrakt von Nutzen ist. Die Besserung nach Aufhören der Behandlung mit Schilddrüsenextrakt ist dann viel leichter in logischer Weise zu erklären. Während der Darreichung von Schilddrüsenextrakt werden die der Schilddrüse direkt antagonistischen Drüsen zur vermehrten Tätigkeit angeregt und hypertrophieren. Der augenblickliche Effekt auf die Akromegalie wird daher, da sich das künstlich zugeführte Sekret mit der durch die Darreichung erzeugten Hypersekretion der Antagonisten so ziemlich das Gleichgewicht hält, ein sehr geringer sein. Setzen wir die Schilddrüsenmedikation aus, dann dauert die Sekretion der hyper-

1) Naturwissenschaftlicher Verein Straßburg, Nov. 1907.

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trophierten antagonistischen Drüsen noch eine Zeitlang an, ohne daß entgiftende Substanz zugeführt wird, und der Überfunktion des blutdrucksenkenden Sekretes der akromegalischen Hypophysis wird dadurch entgegengearbeitet. Dieser Anschauung entsprechend, die ich nach dem heutigen Stande der Erfahrungen akzeptieren möchte, ohne mich aber, da mir eigene Untersuchungen diesbezüglich nicht zu Gebote stehen, dafür einzusetzen, würde die akromegalische Hypophysis einer Drüse entsprechen, welche vorwiegend entgiftendes Sekret produziert; sie wäre also, was ihren Einfluß auf die anderen Drüsen betrifft, der Schilddrüse und dem Ovarium gleichzustellen.

Wir finden dementsprechend bei der Akromegalie die Schilddrüse sehr häufig vergrößert, und wie Pineles (Volkmanns Sammlung klin. Vorträge 242) beobachtet hat, finden sich unter den konstanten Erscheinungen der Akromegalie eine Anzahl Störungen, die sich beim Morbus Basedowii wiederfinden. Mendel beschreibt einen Fall von Hypophysentumor, Struma, Persistenz der Thymus, Mastitis und zystischen Tumor der Ovarien, A. Fränkel[1]) einen Fall von Hypophysentumor, Struma, knotiger Verdickung im Pankreas und Ovarialzyste. Ebenso findet sich nach Claude[2]) bei Morbus Basedowii Akromegalie. Dies stimmt mit den obigen Leitsätzen, daß Hyperfunktion einer entgiftenden Drüse die der andern zur Folge hat. Ebenso hat auch Überfunktion der Schilddrüse das gleiche an der Hypophysis zur Folge. Ja, es gibt Autoren, wie Petrén[3]) und Mendel[4]), welche in Fällen von Akromegalie ohne Veränderung der Hypophysis annehmen, daß hier die Schilddrüse dieselbe Rolle spielt, wie die Hypophysis. Die Möglichkeit wohl zugegeben, ist zu berücksichtigen, daß wir auch mikroskopisch kaum in der Lage sind, zu entscheiden, ob die Hypophysis normal sezerniert hat. Diese gleichzeitige Veränderung an der Schilddrüse und Hypophysis erklärt auch den Befund Holmgrens[5]), daß Patienten im Pubertätsalter mit Morbus Basedowii eine abnorm große Körperlänge erreichen. Ferner kommt, wie ich hier vorwegnehmen will, in der Graviditat, die — darüber soll in einer anderen Arbeit ausführlich die Rede sein — mit vermehrter Tätigkeit des Ovariums einhergeht, nicht selten eine Hypertrophie der Hypophysis vor. Ich verweise diesbezüglich auf die von Halban und erst vor kurzem von Tandler und Groß erwähnten akromegalischen Symptome in der Schwangerschaft.

1) Soc. de biol. 1905, t. 59.
2) Virchows Arch. 1907.
3) Verein für innere Med. 1. April 1901.
4) Berliner klin. Wochenschr. 1900.
5) Hygiea Bd. 18.

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Es wäre ferner noch auf Zeichen der Atrophie an den Genitalien bei Akromegalie hinzuweisen. Verschiedentlich wird angegeben, daß Akromegalie mit Aufhören der Menstruation einhergeht (siehe oben). Man darf dies nicht ohne weiteres als eine Atrophie des Genitales auffassen, denn es wird bei der Amenorrhöe der Akromegalischen der Uterus meist normal angetroffen. Ich möchte daher diese Amenorrhöe ähnlich wie die Basedowamenorrhöe auffassen. Durch die Steigerung der entgiftenden Tätigkeit der Hypophysis (vermehrt durch die gleichzeitige Hypersekretion der Tyreoidea) wird eine solche Entgiftung des Organismus herbeigeführt, daß es nicht zur Auslösung einer genügenden Ovarialsekretion, einer Menstruation kommt (normaler Uterus!). Schließlich kann auch hier die Untätigkeit zur Atrophie führen. Schmidt-Rimpler[1]) hat den ersten anatomisch festgestellten Fall von Hirntumor beobachtet, der unter Erscheinungen der Amenorrhöe und Sehnervenatrophie einherging. Hier handelte es sich aber um einen Uterus infantilis. In anderen Fällen, so in denen von Axenfeld[2]), Yamagucki[3]), Bayerthal[4]), Abelsdorf[5]), L. Müller[6]), Karafiath[7]), ist zu mindest das gleichzeitige Auftreten der Amenorrhöe mit der Erkrankung des Hirnanhangs wahrscheinlich. Patellani[8]) hat 200 solcher Fälle zusammengestellt. Hierzu stehen natürlich die Befunde von Tandler und Groß nicht im Widerspruch. Diese fanden pathologische Veränderungen des Epithels der Samenkanälchen und der Zwischenzellen, sowie totale Rückbildung der Primordialfollikel. Die beiden Autoren neigen mehr zu der Ansicht, daß die primäre Veränderung in der Keimdrüse sitzt, weil in sehr vielen Fällen die gestörte Funktion der Keimdrüse die erste Manifestation der Akromegalie ist. Man muß hier, glaube ich, sehr vorsichtig sein. Die Störung der Menstruation ist ein augenfälliges Symptom; ich wüßte aber nicht, welches Symptom der Akromegalie in der ersten Zeit so augenfällig wäre. Nur Röntgenuntersuchungen gleich bei Ausbleiben der ersten Regel könnten hier vielleicht eine Entscheidung bringen. Gegen die von Tandler und Groß favorisierte Annahme würde außer der normalen Beschaffenheit des Uterus die Beobachtung sprechen, daß Akro-

1) Die Erkrankungen des Auges im Zusammenhange mit anderen Erkrankungen. Wien, Hölder, 1898.
2) Vereinig. Südwestdeutscher Irrenärzte 1902.
3) Klin. Monatsbl. f. Augenheilk. XLI.
4) Sém. Méd. 1905, t. 47.
5) Arch. f. Augenheilk. XXI.
6) Sém. Méd. 1905, t. 47.
7) Ibid.
8) Gyn. Ges. Mailand, Sept. 1906.

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megalie nicht selten von einer Schwangerschaft her datiert wird, was auch Tandler und Groß erwähnen. Denn die Schwangerschaft geht, wie ich an anderer Stelle zu erweisen suche, mit einer vermehrten Tätigkeit des Ovariums einher. Es würde daher die Akromegalie von einem Zeitpunkte ihren Anfang nehmen, wo die Tätigkeit des Ovariums verstärkt ist. Bei solchen Angaben muß man aber, wie ich schon in früheren Arbeiten erwiesen habe, sehr vorsichtig sein. Vor allem pflegen Frauen ihre ganze Zeitrechnung auf Schwangerschaften einzurichten: Das war damals, als ich zum drittenmal schwanger war. Dann aber ist zu bedenken, daß nicht allein Krankheitserscheinungen in der Schwangerschaft aggravieren, sondern auch unter anderen in der Schwangerschaft regelmäßig auftretenden Störungen solche sind, die zu dem Bilde der Akromegalie gehören. Was die Fettleibigkeit bei Hypophysenerkrankung betrifft, auf welche A. Fröhlich aufmerksam gemacht hat, so muß man hier wohl zwischen der Akromegalie und der Unterentwicklung der Hypophysis unterscheiden. Der Fall von traumatischer Verletzung der Hypophysis (Madelung) gehört in die letztere Gruppe. Bei Hypofunktion oder Wegfall der gesamten Hypophysissekretion haben wir denselben Zustand vor uns wie nach Totalexstirpation des Genitales. Wegfall einer entgiftenden Drüse führt zur Fettentwicklung, ebenso wie dies bei Myxödem der Fall ist. Fettentwicklung bei Akromegalie aber, wo es zu einer Hypofunktion des Ovariums oder gar schließlich zur Atrophie des Genitales gekommen ist, muß notwendigerweise auf das Ovarium zurückgeführt werden. Es läßt sich also auch aus diesem Verhalten kein Schluß auf das Primäre der Hypofunktion des Genitale ziehen. Am meisten widerspricht aber dieser Ansicht das gleichzeitige Vorkommen der Akromegalie und der Schwangerschaft. War jene vor dieser vorhanden, dann ist sicher die Atrophie des Genitales nicht das Primäre. Entstand die Akromegalie aber in der Schwangerschaft, dann kann sie erst recht nicht mit einer Unterfunktion des Ovariums in Zusammenhang gebracht werden. Ich neige also mehr zu der Ansicht, daß die Veränderung der Hypophyse das Primäre ist.

Streng hiervon zu trennen ist der Riesenwuchs bei Infantilismus und bei frühzeitig Kastrierten. Tandler und Groß legen bei dem Knochenwachstum das Schwergewicht auf die Schilddrüse. Tatsächlich spielen frühzeitige extra- und intrauterine Erkrankungen der Schilddrüse beim Zustandekommen des Infantilismus eine große Rolle. Dagegen wird von Sanctis und anderen Autoren angeführt, daß es schwere und leichte Infantilismusformen gibt, bei denen die Schilddrüse wohlerhalten ist. Es muß auch auf die anderen Drüsen hinsichtlich des Knochenwachstums Rücksicht genommen werden, vor allem auf die

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Thymus. Basch hat gezeigt, daß Tiere nach Exstirpation der Thymusdrüse einen viel grazileren Knochenbau aufwiesen, und daß nach Knochenbrüchen die Kallusbildung eine sehr geringe war. Ich zitiere ferner Tandler und Groß: Es ist eine längst bekannte Tatsache, daß die Thymus des Menschen durchschnittlich nach dem 2. Lebensjahre im Wachsen stehen bleibt, während ihre Involution knapp vor dem Beginn der Pubertät anfängt. Es ist mehr als wahrscheinlich, daß die Reifung der Geschlechtsdrüse die Involution der Thymus hervorruft. Bei kastrierten Rindern bleibt die Thymus länger bestehen als bei nichtkastrierten (Calzolari, Henderson, Goddal). Wir selbst konnten feststellen, daß bei früh kastrierten Tieren die Thymus persistiert. Aber auch Einwirkung der Thymus auf die Keimdrüse wurde schon beschrieben. Noël Paton hat mitgeteilt, daß bei Meerschweinchen nach Entfernung der Thymus angeblich eine Gewichtszunahme der Testikel zu konstatieren sei. Dies spricht meiner Ansicht nach wohl mit Deutlichkeit dafür, daß die Thymus in die gleiche Ordnung wie Schilddrüse und Ovarium zu stellen ist, daß die Aufgabe der Thymus, freilich in verstärktem Maße, später vom Ovarium übernommen wird. Es darf daher nicht wundernehmen, daß bei Infantilismus, sowie bei frühzeitig Kastrierten die Thymus persistiert.

Pierre Marie, Lannois u. a. haben nachgewiesen, daß die Hypertrophie der Hypophysis während des Wachstums Riesenwuchs, nachher Akromegalie hervorrufe. Tandler und Groß legen großes Gewicht auf die Schilddrüse. Ich meine, daß hier alle innersekretorischen Organe in ihrer gegenseitigen Wechselwirkung von Belang sind.

Es läßt sich hier eine Skala aufstellen: je geringer die Kraft der entgiftenden Organe, desto geringer das Wachstum; hierbei ist natürlich auf die möglicherweise bestehende antagonistische Wirkung des Uterus Rücksicht zu nehmen. Hinsichtlich der Akromegalie ist auch beispielsweise Indemans[1]) der Ansicht, daß nicht die Hypophysis allein die Ursache ist, sondern auch Störungen des Thyreoidalsystems und der Genitaldrüsen. Beim chondrodystrophischen Zwerg haben wir vielleicht eine normale Hyphophysis, eine in ihrer Sekretion schwache Schilddrüse, normales Ovarium, normales Genitale. Der kretinistische Zwerg hat eine Vergrößerung der Hypophysis, eine mangelhaft funktionierende Schilddrüse, Hypoplasie des Genitales. Beim normalen Menschen kommt die normale Schilddrüsensekretion, das normale Genitale hinzu und fällt die Vermehrung der Hypophysissekretion fort. Die Osteomalazie zähle ich auch zu den Erkrankungen, wo ein

1) Nederl. Tijdschr. v. Geneesk. 2. III. 1908.

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Knochenwachstum stattfindet.[1]) Hier haben wir verstärkte Tätigkeit der Hypophyse, der Schilddrüse, des Ovariums und des Uterus. Bei Akromegalie verstärkte Tätigkeit der Hypophysis, Schilddrüse, fehlende des Ovariums und wahrscheinlich fast normale des Uterus. Stärkstes Längenwachstum beim Kastrierten: verstärkte Tätigkeit der Hypophyse, der Schilddrüse, normale der Thymus, fehlende des Ovarium und Uterus. Freilich ist hier zu bemerken, daß die Angaben nicht eindeutig sind. So hat Cesca (Soc. méd. chirurg. de Boulogne, Mars 1904) nach Abtragung des Testikels wie des Ovariums Hypertrophie der Schilddrüse und der Nebenniere beobachtet, während Parathyreoidea, Hypophysis und Thymus keine Veränderungen zeigten.

Was das Verstreichen der Epiphysenfugen betrifft, so scheint dies als Reifeerscheinung an das Übergehen der Sekretion von der Thymus auf das Genitale gebunden zu sein. Mit der Pubertät verschwinden die Epiphysenfugen. Bei frühzeitig Kastrierten bleiben diese bestehen und ebenso beim kretinistischen Zwerg, der ein hypoplastisches Genitale und häufig Persistenz der Thymus aufweist; während beim chondrodystrophischen Zwerg die Epiphysenfugen frühzeitig verstreichen, jedenfalls infolge frühzeitiger Sekretion des Genitales. Vielleicht spielt beim Verstreichen der Fugen die beginnende Sekretion des Uterus eine Rolle.

Es wäre schließlich noch darauf hinzuweisen, daß manche Autoren, so Berger und Loewy[2]), die Amenorrhöe nicht mit der Hypophysis in Zusammenhang bringen, sondern mit dem Hydrocephalus internus. Sie berufen sich hierbei auf 2 Fälle von Herbst[3]) mit Stauungspapille, Amenorrhöe und Hydrozephalus. In einem derselben schwand die Stauungspapille nach der Lumbalpunktion; gleichzeitig trat eine Besserung der Sehschärfe und das Wiedererscheinen der Menstruation auf. Mit Rücksicht aber auf die obigen Tierversuche und Krankengeschichten liegt es wohl näher anzunehmen, daß der Hydrozephalus Stauung oder Kompression in der Hypophysis und so ähnliche Veränderung in der Sekretion derselben zur Folge hatte wie Hirntumoren und solche der Hypophysis selbst.

Ebenso wie Unterfunktion der Ovarien zur Hyperfunktion der anderen entgiftenden Organe führt, so ist dies auch bei Myxödem der

1) Siehe O. O. Fellner, Die Beziehungen innerer Krankheiten zur Schwangerschaft usw. Wien, Deuticke, 1903 und die demnächst erscheinende Arbeit: Über die Tätigkeit des Ovariums in der Schwangerschaft.

2) l. c.

3) Wiener klin. Wochenschr. 1902.

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Fall, und finden wir auch hier Akromegalie (Pineles[1]), Huisman[2]), Greeve[3]).

Die Thymus steht in vollkommener Analogie zur Schilddrüse und zum Ovarium. Auszüge der Thymus wirken nach Svehla[4]) pulsbeschleunigend und blutdruckerniedrigend. Die Thymus verringert auch die Zurückhaltung des Kalziums. Über ihre Wechselbeziehung zum Ovarium wurde bereits oben das Wichtigste angeführt. Nach all dem bisher Angeführten kann es nicht wundernehmen, daß wir bei Basedow nicht gar so selten Persistenz und Hypertrophie der Thymusdrüse finden. Gierke[5]) konnte 42 solcher Fälle aus der Literatur zusammenstellen. Auch bei Akromegalie müßte man a priori annehmen, daß sich öfters Thymuspersistenz findet. Ich verweise auf die früher erwähnten Fälle von Mendel und A. Fränkel. Nagel gibt an, daß die Autoren bald den Schwund, bald die Persistenz der Thymus angeben.

Abtragung des Testikels wie des Ovariums hat nach Cecca nebst Hypertrophie der Schilddrüse auch eine solche der Nebennieren zur Folge. Ebenso fand Marchand bei einem Falle von Hermaphroditismus femininus Atrophie der Ovarien und Hypertrophie der Nebennieren. Théodossiew[6]) beobachtete bei Tieren 10 Monate nach der Kastration vor allem Hyperplasie der Rindenzellen. Es sei ferner auf die Hypersekretion der Nebennieren in der Schwangerschaft hingewiesen. Auf den Antagonismus zwischen Tyreoidea und Nebennieren haben Loeper und Courzow aufmerksam gemacht.

Auch das Pankreas gehört in die Reihe der entgiftenden Drüsen. Bei Akromegalie findet sich vielleicht Hypertrophie des Pankreas (Mendel, A. Fränkel). Andererseits führt Exstirpation des Pankreas zu Hyperaktivität der Schilddrüse (Lorand[7]). Innig, wenn auch zu wenig beachtet, sind die Beziehungen des Diabetes zum Genitale. Die Periode wird immer schwächer, bis sie schließlich ganz erlischt. Gleichzeitig setzt eine allmählich zunehmende Atrophie des Uterus und eine solche, sekundäre, der Ovarien (Hofmeier) ein. Eine Erklärung für diese Tatsache wage ich nicht auszusprechen, da sie derzeit zuviel der Theorien und Hypothesen enthalten würde. Das gleiche

1) Volkmanns Samml. klin. Vortr. 242.
2) Therapie der Gegenwart 1903, Bd. 44.
3) New York. med. Journ. vol. 82.
4) Arch. f. exper. Pathologie 1900, Bd. 43.
5) Münchner med. Wochenschr. 1907, Bd. 16.
6) Russki Wratsch Febr. 1906.
7) Pres. méd. 1904, 27.

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gilt für die Tatsache, daß Diabetes häufiger bei Männern vorkommt. Bouchardet[1]) berechnet 172 Männer auf 53 Frauen.

Von obigen Regeln einigermaßen abweichend sind die Angaben einzelner Autoren wie Kleinwächter[2]) und Jahreiß[3]), welche auch stärkere Blutungen bei Diabetes beschreiben. Es handelt sich hier aber meist um Myome, wie überhaupt bei Myomen Diabetes nicht selten ist (Naunyn[4]) 8 Fälle unter 400 diabetischen Individuen, Boas[5]) 12 unter 366). Mit aller Reserve möchte ich die Vermutung aussprechen, daß hier eine starke Sekretion des Uterus besteht, die vielleicht hinsichtlich der Glykosurie der Nebennierensekretion gleichzustellen ist. A. Glies gibt an, daß Glykosurie nach Entfernung des Myoms prompt schwindet.

Schwieriger zu erklären sind die Fälle von Imlach[6]), Beyea[7]), Giles[8]), Gottschalk[9]) und der meine[9]). Es handelte sich um Schwinden der Zuckerausscheidung nach Entfernen der Adnexe oder des Uterus. Hier kämen zwei Momente in Betracht, eventuell der Wegfall einer blutdrucksteigernden Drüse, andererseits der Umstand, daß vielleicht durch den Wegfall der Eierstöcke die anderen entgiftenden Drüsen, darunter auch das Pankreas, hypertrophieren dürften.

Ganz im Widerspruch mit den sonstigen Erfahrungen steht ein anderer von mir publizierter Fall, wo einige Zeit nach Exstirpation eines Myoms Diabetes auftrat.

Wenn ich das wichtigste Ergebnis dieser Studie kurz zusammenfasse, so haben wir in einem Teil der Hypophysis, in der Schilddrüse, dem Pankreas, der Thymus und der Ovarien blutdrucksenkende Drüsen vor uns, die sich gegenseitig vertreten können, und welchen ein Teil der Hypophysis, die Nebenniere und vielleicht der Uterus als blutdrucksteigernde Drüsen antagonistisch gegenüberstehen.

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1) De la glycosurie ou diabète sucré. Paris 1875.
2) Zeitschr. f. Gyn. 1900, Bd. 43.
3) Zentralbl. f. Gyn. 1901, Bd. 2.
4) Der Diabetes mellitus. Nothnagels Handb. 1878.
5) Hufeland-Gesellsch. 20. Nov. 1902.
6) Brit. med. Journ. July 1885.
7) Amer. Journ. of obstetr. 1900.
8) Brit. med. Journ. 1900.
9) O. O. Fellner, Über den Diabetes in der Chirurgie. Wiener klin. Wochenschr. 1903, Bd. 34.

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(Innere Medizin Nr. 153.)

Die Seekrankheit und ihre Verhütung.

Von

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Obgleich die Frage der Seekrankheit schon oft das Interesse der Forscher erregt hat, und die Literatur über diesen Gegenstand geradezu unübersehbar geworden ist, hat man doch nur sehr geringe Fortschritte gemacht in der Bekämpfung dieser höchst unangenehmen Krankheit. Auf zweierlei Weise hat man sie zu verhüten versucht, nämlich:

1. durch Mittel, die die unerwünschten Schiffsbewegungen möglichst zu vermindern suchen.

   Diese Mittel beruhen größtenteils auf dem Prinzip, das zuerst von Bessemer beschrieben ist in seinem: „Projet de Salon suspendu contre le mal de mer".

   Sie haben nur wenig Anwendung gefunden und sind in dieser Hinsicht von späteren Bestrebungen nicht übertroffen, die in anderer Weise automatisch die Schiffsbewegungen zu vermindern suchten (Schwerdt, Rosenbach u. a.).

   Diese Mittel beeinflussen am wenigsten die Bewegungen des Schiffes in senkrechter Richtung, die gerade von Seekranken am meisten gefürchtet werden. Sie haben große technische Schwierigkeiten und niemals den gewünschten Zweck ganz erreicht; sie werden deshalb hier außer Betracht bleiben.
2. durch Mittel, die die Empfänglichkeit des Individuums für die Schiffsbewegungen zu vermindern suchen.

Je nach den verschiedenen Vorstellungen, die sich die Schriftsteller über Seekrankheit hinsichtlich der Genese dieses Leidens

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gebildet haben, sind auch die von ihnen empfohlenen Maßnahmen zur Verhütung oder Bekämpfung verschieden. Ehe wir die verschiedenen Theorien der Seekrankheit besprechen, werden wir uns zuvor die Symptome der Krankheit kurz ins Gedächtnis zurückrufen:

In der übergroßen Mehrzahl der Fälle ist die Seekrankheit eine akute, selten länger als eine Woche dauernde Erkrankung. Sie fängt mit einer leichten Unlust an, als Vorläufer einer tief elenden psychischen Depression, und ist begleitet von objektiven Symptomen wie: Anämie, Gähnen, Speichelfluß, Nahrungsverweigerung und Erbrechen, Respirations- und Zirkulationsveränderungen, Störungen der Digestion (gewöhnlich Obstipation), kleiner schwacher Puls, und bei längerer Dauer: Abmagerung. Auch Verminderung der Harnabsonderung bei Erhöhung der Konzentration scheint ziemlich konstant zu sein. Reynolds[1]) berichtet, daß er wiederholt Zucker im Urin der Seekranken gefunden hatte. Auch scheint die Seekrankheit sehr oft auf die Menstruation und Schwangerschaft störend einzuwirken.

Schwerdt[2]) fand während einer Seereise bei sich selbst intraabdominale Druckschwankungen, die abhängig von den Schiffsbewegungen waren. Er meint, daß dadurch Zirkulationsstörungen entstehen müßten, die man willkürlich vermindern könne durch tiefes Einatmen beim Heruntergehen des Schiffskörpers und Ausatmen beim Heraufgehen desselben. Dadurch würde der „Inspirations-Klappenschluß“ (d. h. der Klappenschluß der Oberschenkelvenen bei der intraabdominalen Druckerhöhung während der Inspiration) zusammenfallen mit dem „Senkungs-Klappenschluß“ (d. h. dem Klappenschluß durch Rückstoß des Blutes am Ende der niedergehenden Bewegung des Schiffes). Ebenso werden dann „Exspirations-“ mit „Hebungs-Klappenöffnung“ zusammenfallen. Auf analoge Weise bewirkt man instinktiv ein Zusammenfallen von „Arbeits-Klappenschluß“ mit „Inspirations-Klappenschluß“ dadurch, daß man jede schwere Arbeit mit einer tiefen Inspiration anfängt. Schwerdts Darlegung gibt eine theoretische Erklärung zu der schon längst bekannten empirisch gefundenen Bedeutung der Atmungsregulierung nach den Bewegungen des Schiffes. —

Wenn eine heruntergehende Bewegung schnell und kräftig auftritt, erweckt sie bei den meisten Menschen reflektorisch eine krampfhafte Inspiration, die oft von einer Kontraktion der Bauchmuskeln begleitet

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1) T. T. Reynolds, On the nature and treatment of sea-sickness. Lancet 1884.

2) Dr. C. Schwerdt, Beiträge zur Ursache und Vorschläge zur Verhütung der Seekrankheit. Jena 1902.

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ist. Durch die Unregelmäßigkeit und Kompliziertheit der Schiffsbewegungen wird aber die praktische Anwendung dieser Atmungsregulierung gewöhnlich sehr schwer, wo nicht unmöglich.

Die subjektiven Erscheinungen der Seekrankheit sind sehr ernst. Zu welch einem bedauernswürdigen psychischen Elend sie führen, wurde von Riese[1]) treffend und schön wie folgt beschrieben:

„Die Vorstellungen sind außerordentlich matt und die Phantasie erschrecklich träge. Bleierne Trägheit lähmt auch die Glieder; eine bodenlose Interesselosigkeit hat sich unserer bemächtigt, eine grenzenlose Blasiertheit, in welcher nichts, was uns interessierte, irgendwelchen Reiz auf uns auszuüben vermag. Die ganze Welt erscheint grau in grau gemalt; es ist eine vollkommene Paralyse der Lebenslust. Dies ist das Stadium, wo der Mensch am liebsten allein ist, wo er jeder Konversation ausweicht, und schon in seinem konventionellen Standpunkt zu wanken beginnt. Mühsam sucht er noch den Gesetzen der Höflichkeit zu folgen und Teilnahme an der menschlichen Gesellschaft zu erheucheln, oder über einen Scherz zu lächeln, aber sein Gesicht, dessen Blässe mittlerweile mit dem seiner Gedanken zu wetteifern beginnt (denn es reiht sich jetzt eine zweite Symptomenreihe an die psychischen), hat einen eigentümlich starren Ausdruck erlangt, und sein Lächeln straft ihn Lügen; es ist ein Risor sardonicus, eine wahre Karikatur auf die Lustigkeit. Es liegt in diesem Zustand, in diesem vergeblichen Ankämpfen, welches der beginnenden Seekrankheit charakteristisch ist, gerade die eigentümliche Komik, welche dieselbe zum Schaden ihrer Opfer nicht verleugnen kann und die besonders an dem Starken und Selbstbewußten hervortritt. Gerade an ihm hat der allmähliche Übergang von stolzester renommistischer Sicherheit durch stillen Zweifel an den eigenen Kräften zur demütigsten Ergebung etwas ungemein Komisches für den, welcher nicht im Banne der Krankheit steht. Man sieht, wie immer mehr und mehr der Versuch, das Gefühl der persönlichen Würde auch in der äußeren Haltung auszuprägen, aufgegeben wird, und wie die stolzeste Gestalt zu einem kläglichen Bilde des Jammers zusammensinkt. Das Gesicht bekommt durch jenes ‚Erstarren des Muskelspiels', wie es Steinbach bezeichnet, etwas Fremdartiges, was man schwer beschreiben kann, etwas Wachsfigurenartiges; es ist, als wäre jeder geistige Zug aus dem Gesichte gewichen und nur gewissermaßen die Maske desselben geblieben. Gut bekannte Gesichter kommen uns in diesem Zustande fremd vor, am fremdartigsten dasjenige, welches uns der Spiegel zeigt."

Wer niemals seekrank war, und auch nie Gelegenheit hatte, an

1) E. Riese, Die Seekrankheit. Dissert. Berlin 1888. S. 15.

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ernsten Formen der Seekrankheit leidende Personen zu beobachten, wird sich kaum eine richtige Vorstellung dieses Zustandes der höchsten Verzweiflung bilden können. Des Dichters Wort:

„Only those who brave its dangers
Comprehend its mysteries"

gilt wohl zu allererst dem wunderlichen Stadium, in dem man gegen „dangers" wie gegen „mysteries" völlig gleichgültig ist. —

Riese vergleicht den psychischen Zustand von schwer Seekranken mit den schwersten Depressionszuständen der Melancholie; Fonssagrives[1]) beschreibt diese völlige Apathie wie folgt:

„Chez beaucoup de personnes il y a un tel brisement des forces, qu'elles gisent sur le pont comme des masses inertes sans s'inquiéter du lieu où elles sont, souillant leur vêtements de leurs évacuations et la vie cérébrale est tellement dominée par cet état d'angoisse, que les deux sentiments les plus puissants et les plus vivaces chez la femme, celui de la pudeur et celui de la maternité, sont quelque fois, comme on l'a fait remarquer, momentanément méconnus."

Auch hat die Seekrankheit viel Ähnlichkeit mit akuter Nikotinvergiftung, wie mancher aus Erfahrung wissen wird.

Mit der obigen Beschreibung der objektiven und psychischen Erscheinungen ist das Krankheitsbild noch nicht erschöpft. Dazu gehört noch: Schwindel, Übelkeit, oft auch Hyperästhesie für üble Gerüche, bisweilen auch für Geräusche, und als weniger konstante Symptome Kopfschmerz, Angst und Beklommenheitsgefühl.

Als Komplikationen werden von den meisten Forschern genannt:

1. Frühgeburt und Abortus. Wegen des geburtsbeschleunigenden Einflusses werden von verschiedenen befugten Autoren (u. a. Fonssagrives, Foucauet) Seefahrten den Schwangeren abgeraten.

2. der Ausbruch von Psychosen. Die Seekrankheit wird in diesen Fällen gewöhnlich als Gelegenheitsursache betrachtet. Dieselbe Rolle spielt sie in einigen anderen krankhaften Zuständen, wie z. B. bei der Inkarzeration von Hernien, oder beim Durchbruch eines Magengeschwürs.

Ein Symptom, der Schwindel, verdient vor allem besondere Aufmerksamkeit. Denn so oft wir Schwindel schnell und einigermaßen heftig auftreten sehen, wird er von anderen Erscheinungen begleitet, die mit zu dem Bilde der Seekrankheit gehören.

Unser Gleichgewichtsgefühl resultiert aus zahlreichen Empfindungen, die auf mehreren verschiedenen Wegen (Gesichtssinn, Labyrinth, Tastsinn, Muskel- und Gelenksinn) die Zentralorgane erreichen, und auf

1) Fonssagrives, Traité d'hygiène navale. Paris 1877.

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Stellungsänderungen unsers Körpers im Raume mit bestimmten, uns aus der Erfahrung bekannten Änderungen reagieren. Unter der richtigen Zusammenwirkung zahlreicher sensibler und sensorischer Empfindungen kann man sich eine gute Orientierung ungefähr so entstanden denken, wie durch Zusammenwirkung von mehreren Instrumenten harmonische Musik hervorgebracht werden kann. Die Orientierung einerseits, wie die Harmonie andererseits brauchen nicht durch Ausfall eines Teiles des Ganzen verloren zu gehen; jedoch ist dies der Fall durch unharmonisches Mitarbeiten eines der Konstituenten. „Wenn wir", sagt Hitzig[1]), „die mannigfaltigen Erscheinungen von Schwindel von einem ihnen gemeinschaftlichen Gesichtspunkte aus ins Auge fassen, so ergibt sich, daß sie sowohl durch Affektionen der peripheren Endapparate, wie der zuleitenden Bahnen, wie auch endlich des Zentralorgans, in dem sich alle diese Bahnen vereinigen, hervorgebracht, andererseits aber auch durch die Einwirkung jener verschiedenen exzito-motorischen Apparate bis zu einem gewissen Grade ausgeglichen werden können. Der Vestibularapparat, der Sehapparat und der kinästhetische Apparat spielen hierbei die gleiche Rolle. So verschieden die Folgen der Eingriffe in ein Ampullarsystem, der Totalexstirpation des Kleinhirns, die Lähmung eines Augenmuskels, ein Kleinhirntumor, oder die graue Degeneration der Hinterstränge auch erscheinen mögen, so sind sie ebensowohl wie ihre Ausgleichung auf dasselbe Prinzip zurückzuführen."

Bei einer ersten Seefahrt werden in allen oben genannten Bahnen desorientierende Reize den Zentralorganen zugeführt. Kein einziger Punkt des ganzen Gesichtsfeldes bleibt unbeweglich, alles wirbelt in unregelmäßigster Weise durcheinander, was anfänglich den Eindruck eines ganz ungewöhnlichen Wirrwarrs macht. Inzwischen stören zahlreiche ungewöhnliche Reize, vom Vestibularapparat ausgehend, die normale Funktion des Kleinhirns. Der kinästhetische Apparat bereitet uns gleichfalls die größten Überraschungen. Die sonst so konstante Druckempfindung gegen die Fußsohlen ändert sich jetzt fortwährend. Beim Gehen hat man kaum seine Muskelinnervation für eine bestehende Neigung richtig dosiert, so geht es uns dabei wie jemandem, der das Pappmodell eines schweren Gewichtes hebt. Ehe man sich von einem Fehlgriff Rechenschaft geben kann, ist man schon wieder das Opfer eines folgenden geworden. Diese Reize bewirken durch ihren fortwährenden Wechsel eine vollständige Desorientierung, welche zu einer an Ratlosigkeit grenzenden Verwirrung führen kann. Bei

1) Hitzig, Der Schwindel (Vertigo). Spez. Path. und Ther. von H. Nothnagel Bd. 12, T. 2.

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großer Anstrengung gelingt es anfänglich noch das Gleichgewichtsgefühl zu behalten, dadurch nämlich, daß man soviel wie möglich die ungewöhnlichen Reize zu entwirren versucht. Diese Wahrnehmung wurde schon 1874 von Th. Abrahamsz[1]) gemacht, die er ungefähr wie folgt beschreibt:

„Nicht durch Zerstreuung im Gespräch oder Spiel während der ersten Anwandlung gelingt es dem Erbrechen zuvorzukommen, sondern dadurch, daß man genau acht gibt, und die Schiffsbewegungen studiert in allen ihren verschiedenen Phasen und Abweichungen. Nur auf diese Weise gelang es uns, das Erbrechen zu verhüten, indem wir jedoch auch jetzt noch, nach langer Erfahrung, ein Gefühl von Unbehagen empfinden, wenn wir uns bei mehr oder weniger stürmischem Wetter mit Sachen beschäftigen, die uns verhindern, die Bewegungen zu observieren. Jede Ableitung rächt sich; sobald man sozusagen nicht mehr weiß, wo das Schiff in seiner Bewegung im Raume geblieben ist, fühlt man sich schwindlig."

Auch Riese machte an sich selbst eine übereinstimmende Beobachtung und sagt:

„Bis zum Abend des ersten Tages kämpfte ich erfolgreich gegen die feindliche Macht, dann erfolgte die Übergabe; doch ich habe das deutliche Bewußtsein, daß ich dieselbe durch ein willkürliches Widerstreben ziemlich lange hinausschob. Ich habe das Gefühl, daß mir dies dadurch gelang, daß ich mir der Schwankungen des Schiffes und des eigenen Körpers so weit als möglich bewußt blieb.

Ich suchte mich also in den verschiedenen, stets wechselnden Lagen im Raum zu orientieren und mir meiner augenblicklichen Stellung zu demselben in jedem Moment möglichst klar zu bleiben. Ich kann versichern, daß ich darin ganz wesentlich unterstützt wurde durch ein beständiges Festhalten des Horizontes mit den Augen.

Solange mir dies gelang, wurde ich nicht seekrank; erst als es mir bei den immer komplizierteren Bewegungen des Schiffes nicht mehr möglich war, mir innerlich über meine Schwankungen klar zu bleiben und sie gleichsam gegen den stabilen Raum zu normieren, erfaßte mich ein eigentümliches Schwindelgefühl: es war nicht mehr, als ob ich im feststehenden Raume schwankte, sondern der Raum schien mit einem Male um mich sich schwankend zu bewegen, er schien seinen festen Bestand verloren zu haben, und jede Möglichkeit, die Bewegungen des eigenen Körpers zu kontrollieren, hörte mit einem Male auf."

Und weiter auf S. 70 seiner Arbeit über: „Die Seekrankheit": „Dieser Moment des Insuffizientwerdens des Orientierungssinnes ist

1) Nederlandsch Tijdschrift voor Geneeskunde 1874, 10. Jaarg. II, p. 237.

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dadurch charakterisiert, daß der Beobachter das Gefühl hat, als drehten sich die Gegenstände um ihn, und, auch wenn er die Augen schließt, als drehe sich der Raum um ihn. Äußerlich ist dieser Moment des einsetzenden Schwindels charakterisiert durch ein Starrwerden des Blicks, hervorgerufen (wie Purkyne nachgewiesen hat) durch eine plötzliche Fixation des Augapfels, der bis dahin (d. h. solange eine Orientierung im Raume möglich war) ruckweise den Achsendrehungen des Körpers gefolgt ist." —

Beim Entstehen des Desorientationsgefühles tritt auch das dringende Bedürfnis auf, sich hinzulegen. Die Stabilität ist im Liegen gesicherter und die desorientierenden Reize sind geringer an Zahl und Stärke. Ziemlich schnell kann sich dann das Wohlbefinden ansehnlich bessern, und besonders ist dies bei denjenigen der Fall, denen ein erfrischender Schlaf zu Hilfe kommt, das ausgestandene Elend zu vergessen und das gestörte Nervengleichgewicht zur Ruhe zu bringen.

Anfänglich wird aber noch jeder Versuch, sich aufzurichten, das Gefühl hervorbringen, daß die Gewöhnung an die Schiffsbewegungen nicht so schnell eintritt, als man sich beim Liegen dachte. Denn bei einem Wetter, das eine mäßige Bewegung des Schiffes unterhält, gewöhnen sich die meisten Seekranken nur sehr allmählich, im Laufe einiger Tage, ans Meer.

Die Prognose der Seekrankheit ist günstig. Sterbefälle als Folge der Seekrankheit allein sind niemals mit Sicherheit wahrgenommen; zurückbleibende Störungen ebensowenig. Wo in der Literatur einige Male der Tod eines Patienten ausschließlich der Seekrankheit zugeschrieben wurde, lassen sich doch gegen die Diagnose ernste Bedenken erheben. Guépratte, der in den „Bulletins de l'académie de médicine IX" den Zusammenhang zwischen Seekrankheit und zwei von ihm angeführten Sterbefällen an Enzephalitis und Gastroenteritis bestreitet, behauptet: „On a vu survenir chez quelques personnes des hématémèses dont la terminaison a été funeste", ohne aber einen bestimmten Fall zu beschreiben. H. Otto Schultze beschrieb: „Two cases of fatal hemorrhage from laceration of the mucosa of the œsophagus by vomiting" (Proceedings of the New-York pathological Society, Nov. 1906, n. s. vol. 6, no. 6).

Bei beiden Patienten war der Blutung langwährendes Erbrechen vorangegangen, bei einem der beiden infolge von Seekrankheit. Bei der Obduktion wurden in beiden Fällen zwei longitudinale Risse in der Schleimhaut des Ösophagus gefunden, obwohl die Schleimhaut bei mikroskopischer Untersuchung nichts Abnormes darbot. Auch wenn man gegen diesen sonderbaren Fall keine Bedenken erheben

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will, wird man mit Rücksicht auf die ungeheuere Zahl der Opfer, die die Seekrankheit seit Hunderten von Jahren erfordert, die günstige Prognose nichtsdestoweniger aufrecht erhalten müssen. Nur in betreff der Dauer ist die Prognose nicht immer günstig. Zwar verschwinden die Erscheinungen der Krankheit immer sehr schnell bei Beendigung der Ursache (bei Beendigung oder Unterbrechung der Seereise), aber bei weiten Reisen findet man nicht selten, daß einige Leute sich sehr langsam und schwer ans Meer gewöhnen. Es tut wenig zur Sache, ob man die Seekrankheit in solchen Fällen als chronische bezeichnen will oder nicht. (Riese hat gegen diese Benennung Bedenken, weil die Krankheit verschwindet, sobald ihre Ursache endet.) Aber hinsichtlich der Erscheinungen, die viel weniger heftig sind wie im Anfang der Seekrankheit, ist die Unterscheidung in akute und chronische Formen ganz berechtigt. Für Berufsseeleute ist eine solche Überempfindlichkeit eine schlimme Eigenschaft, die jede neue Seefahrt zu einer neuen Quälerei macht. Denn ein solcher chronischer Seekranker ist auf dem Meere fast immer verstimmt, er leidet an Digestionsstörungen, Obstipation, hat wenig oder gar keinen Appetit und fühlt je nach der Witterung mehr oder weniger Neigung zum Erbrechen; er ist anämisch und leidet oft an Kopfschmerz. Jedoch kann er meist im Gegensatze zu dem akut Seekranken seine Arbeit verrichten und nimmt auch, obschon mit Unlust, Teil an der Unterhaltung. Daß sogar eine solche Überempfindlichkeit die Möglichkeit nicht ausschließt, eine ruhmreiche Laufbahn als Seemann zu machen, bewiesen Nelson und Tegetthoff, die beide zu dieser Rubrik der Überempfindlichen, die von Steinbach[1]) auf 6—8% der Menschheit geschätzt wird, gehörten.

Havelberg[2]) sucht die Ursache der Überempfindlichkeit in einer Magenneurose. Da aber die mir bekannten überempfindlichen Seeleute im Hafen und auf dem Lande eine tadellose Magenfunktion hatten, kann mich diese Erklärung für die mir bekannten Fälle nicht befriedigen. Mir sind ein Dutzend solcher überempfindlichen männlichen Personen bekannt, an denen mir auffiel, daß keiner von ihnen rauchte bzw. priemte. Die Hälfte davon waren Berufsseeleute, bei denen übrigens das Verschmähen des Genußmittels Tabak selten ist. Mehrere Autoren berichten von Überempfindlichkeit bei hysterischen Personen; bei den mir bekannten zwölfen konnte aber auch von hysterischer Anlage nicht die Rede sein.

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1) Steinbach, Zur Pathologie der Seekrankheit. Wiener Med. Presse 1878.

2) Havelberg, Hyperazidität und Seekrankheit. Festschrift f. Salkowski, Berlin. S. 181.

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Bei den Überempfindlichen fängt auf der ersten Reise die Seekrankheit unter den heftigsten Erscheinungen an.

Fournier beschrieb: „Un cas grave de mal de mer“ in den „Archives de méd. navale 1874“. Er fand die Symptome so gefahrdrohend, daß er es für nötig hielt, das von Forget genannte „remède spécifique et unique du mal de mer“ anzuwenden, d. h. den Patienten ans Land zu setzen.

Ich hatte selbst Gelegenheit, einen Patienten zu untersuchen, der 3 Wochen lang seekrank gewesen war, bevor er in meine Behandlung kam. Es war ein junger Matrose, der eine Seefahrt von Holland nach Westindien gemacht hatte. Dieser 18jährige Seemann hatte während der Überfahrt sehr wenig Nahrung zu sich genommen und sich sehr oft erbrochen. Er wurde wie ein Schwerkranker von zwei Gehilfen ins Schiffslazarett getragen, und war duselig, schläfrig, apathisch, sehr anämisch und abgemagert, und konnte vor Schwäche nicht stehen. Die Körpertemperatur schwankte den ersten Tag zwischen 38,2 und 38,7° Celsius und blieb während der folgenden Tage noch etwas erhöht. Die Zunge war fleckig belegt, fuliginös, und der Patient zeigte noch deutlich Speichelfluß. Der Puls war kaum fühlbar und sehr frequent, aber regelmäßig. Das Herz war etwas dilatiert und an der Herzspitze hörte man ein systolisches Geräusch. Die Muskeln waren äußerst schlaff, die Reflexe stark erhöht. Die Haut zeigte starke Dermographie. Der Harn enthielt keinen Zucker, aber eine Spur von Eiweiß. Jedoch hatte der Mann auch früher an Albuminurie gelitten, und es blieb auch nach seiner Wiederherstellung ein wenig Albumen in seinem Harn.

Auf vor Anker liegendem Schiffe, bei Bettruhe und kräftiger Nahrung besserte sich der Zustand des Patienten schnell. Während der ersten Tage schlief er fast fortwährend zwischen seinen Mahlzeiten. Am 12. Tage nach seiner Ankunft in Westindien fühlte sich der Patient wiederhergestellt, obgleich er noch ein wenig unsicher auf den Füßen stand und es ihm schwindlig war. Er wog damals 101 Pfund, und hatte schon sichtlich an Gewicht zugenommen. Zwei Monate später wog er 115 Pfund. Bei nur wenig bewegtem Schiffe wurde dieser Matrose später immer wieder seekrank. Er fühlte sich dann unbehaglich, hatte Kopfschmerz, und mußte sich oft erbrechen. Aber doch hatte er sich so weit ans Meer gewöhnt, daß er fast augenblicklich nach dem Erbrechen wieder essen konnte, sich nicht mehr zu stützen brauchte, um sein Gleichgewicht zu behalten, auch hatte er das unangenehme Gefühl, als ob sich ihm alles vor den Augen drehte, verloren. Nur bei stürmischem Wetter konnte er sich oft nicht be-

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herrschen. Er wurde dann irgendwo liegend im Schiffe gefunden und war so apathisch, daß er auf Fragen kaum antwortete.

Unempfindlichkeit für Seekrankheit ist bei Erwachsenen gewiß viel seltener wie Überempfindlichkeit. Ob unter normalen Umständen bei einer ersten Seefahrt auf kleinem, beweglichem Schiffe und bei stürmischem Wetter völlige Unempfindlichkeit überhaupt vorkommt, ist sehr zweifelhaft, ausgenommen bei Säuglingen und sehr kleinen Kindern. Denn dem Säuglingsalter scheint, wie fast alle Forscher einstimmig zugeben, Unempfindlichkeit für Seekrankheit eigen.

Riese, der besonders auf diesen Punkt geachtet zu haben angibt, hat niemals einen seekranken Säugling gesehen, dagegen oft „die schwer seekranke Mutter in absoluter Passivität und Hilflosigkeit, und daneben in eigentümlichem Gegensatz zu ihr das Kind, welches in naiver Rücksichtslosigkeit und völliger Frische unbekümmert an der Mutter herumspielt".

W. Hesse[1]) bestimmte auf einer Seefahrt die Veränderung des Körpergewichtes von 44 Passagieren, unter denen 2 Säuglinge und 1 2jähriges Mädchen waren. Er teilte die Seekranken nach der Heftigkeit der Krankheit in drei Gruppen ein und kam zu folgendem Resultat, welches die Gewichtsverluste während einer Woche bei leicht stürmischem Wetter angibt:

	Zahl	wiegen zusammen	verlieren zusammen	Jede Person verliert durchschnittlich
Nicht seekrank	12	1440,5 Pfd.	9 Pfd.	0,75 Pfd.
Seekrank 1. Grad	10	1328,5 „	15 „	1,5 „
„ 2. „	18	2486,5 „	86,25 „	4,8 „
„ 3. „	4	552 „	51 „	12,75 „

Nur 5 Personen zeigten Gewichtszunahme, am meisten (3 Pfund) einer der beiden Säuglinge. Der andere Säugling nahm 1 Pfund ab. Das 2jährige Mädchen war leicht seekrank und verlor 3 Pfund. Hesse bemerkt, wie gewöhnlich die Seekrankheit der Mutter auch dem Säugling schadet, erstens weil die Milchsekretion meist zu wünschen übrigläßt, und zweitens, weil seekranke Mütter oft ihre Kinder schrecklich vernachlässigen.

Die Therapie der Seekrankheit ist ebenso reich an Mitteln wie arm an Erfolg. Selbst wer sonst auf Arzneien schwört, pflegt gegen Seekrankheit kein Mittel zu brauchen, weil fast jeder Laie davon überzeugt ist, daß dagegen doch nichts zu tun sei, so daß die Mittel sogar

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1) Dr. W. Hesse, Ein Beitrag zur Seekrankheit. Arch. d. Heilkunde 1874, 25. Febr., XV, S. 134.

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ihren suggestiven Wert verlieren. Doch kann Ratschlägen, wie den folgenden, ein gewisser Wert nicht abgesprochen werden. Man soll wenigstens eine Stunde vor Anfang der Seefahrt eine kräftige Mahlzeit zu sich nehmen; beim Auftreten vom Schwindel sich hinlegen, für kräftige Ventilation Sorge tragen, außerdem noch versuchen, ob eine Leibbinde einige Erleichterung gewährt, wie einige Autoren behaupten an sich selbst empfunden zu haben. Einige Seekranke fühlen einen günstigen Einfluß von permanenter Bauchlage. Wenn das Schiff vornehmlich stampft, kann man demjenigen, welcher noch für solche Ratschläge empfänglich ist, anraten, die Respiration nach den Schiffsbewegungen zu regulieren, in der Weise, wie es schon im Anfang dieses Aufsatzes beschrieben wurde. Möglicherweise hat auch Kopfstauung nach der Bierschen Methode, wie es Simon[1]) empfiehlt, einigen Einfluß durch Milderung der von Hirnanämie abhängigen Erscheinungen.

Es würde zu weit führen, hier alle Theorien der Seekrankheit ausführlich kritisch zu besprechen. Kürze halber wird dieser ausgebreitete Gegenstand hier einigermaßen schematisch behandelt. Bei der Beurteilung dieser Theorien muß man nicht nur die Erscheinungen der Seekrankheit in Betracht ziehen, sondern auch die Unterschiede in der Empfindlichkeit und die der Gewöhnung berücksichtigen. In dieser Hinsicht sind folgende, von den meisten Forschern übereinstimmend konstatierte Tatsachen von Interesse:

1. Die Erscheinungen sind um so schlimmer, je größer, unregelmäßiger und zusammengesetzter die Schiffsbewegungen sind. Die Bewegung um des Schiffes Querachse (Stampfen) mit ihren großen Ausschlägen hat viel mehr Bedeutung als die um des Schiffes Längsachse (Rollen).

2. Liegende Stellung mildert die Erscheinungen.

3. Säuglinge und sehr kleine Kinder sind unempfindlich. Frauen sind im allgemeinen empfindlicher als Männer.

4. Bei weitem die Mehrzahl der Menschen gewöhnt sich im Laufe einiger Tage an die Schiffsbewegungen. Diese Immunität ist aber sehr relativ. Sie kann auf die Dauer beim Aufenthalt auf dem Lande wieder ganz verloren gehen und gilt nicht für viel kleinere Schiffe oder für weit stürmischeres Wetter.

5. Einige Leute sind überempfindlich. Die akute Seekrankheit nimmt bei ihnen eine mehr chronische Form an, und jede Seefahrt stört aufs neue das Wohlbefinden.

1) J. Simon, Biersche Kopfstauung als Mittel gegen Seekrankheit. Therapie der Gegenwart Jan. 1907.

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Und weiterhin dürfen die Theorien der Seekrankheit nicht in Widerspruch kommen mit zahlreichen angestellten Beobachtungen über das physiologische und pathologische Gleichgewicht.

Unter den zahlreichen aufgestellten Theorien der Seekrankheit gibt es viele, die so oberflächlich und ungenügend die Erscheinungen erklären, daß jede Kritik überflüssig erscheint. Zu dieser Rubrik können diejenigen Theorien gerechnet werden, die alle Erscheinungen der Furcht vor der Seereise zuschreiben, wie auch diejenigen, die in der Seekrankheit einzig und ausschließlich nur einen optischen Schwindel sehen wollen. Auch die Theorie, welche die Seekrankheit als eine Intoxikation betrachten will, infolge Einatmung von zerstäubtem Seewasser, gehört hierher, wie auch endlich die Theorie, die die Erscheinungen der Seekrankheit aus Irritationen der Valleixschen Druckpunkte ableiten will.

Cornelius[1]), der Autor der letztgenannten, noch ziemlich modernen Theorie, meint durch Massage der Druckpunkte oder durch sog. Festlegen derselben mittels Pelotten die Erscheinungen der Seekrankheit bedeutend mildern zu können, wenn nur, fügt er vorsichtshalber hinzu, das Wetter nicht zu stürmisch ist. — Nachdem er an sich selbst Untersuchungen angestellt hatte, konkludierte er, daß durch Druckpunktbehandlung in „kürzester Zeit eine sonst nicht zu erwartende Gewöhnung an die Schiffsbewegungen“ eintrat.

Die übrigen Theorien kann man übersichtlich in drei Gruppen teilen, zwischen denen aber viele Übergänge und Kombinationen bestehen. — Die erste Gruppe, die der mechanischen Theorien, meint, daß die Schiffsbewegungen durch Erschütterungen und Schwankungen in den Geweben einiger oder aller Organe mechanische Änderungen hervorbringen.

Die zweite Gruppe, die man Reflextheorien nennen kann, sieht in den Schiffsbewegungen Reize, die von den sensiblen Nerven und Sinnesorganen aufgefangen, auf reflektorischem Wege die Erscheinungen der Seekrankheit erwecken.

Die dritte Gruppe, die der psychischen Theorien, sucht die Ursache der Krankheit in einer bewußten Gleichgewichtsstörung, in einem Gefühl von Desorientierung im Raume. Zu den bekanntesten mechanischen Theorien gehören die von Fonssagrives, der in einer zentrifugalen Bewegung der Zerebrospinalflüssigkeit die Ursache der Seekrankheit suchte, ferner die von Larrey, der Seekrankheit als eine Art leichter Hirnerschütterung betrachtet, ein „ébranlement des

1) Cornelius, Berliner klin. Wochenschr. 1903, S. 673 und Jahresbericht über die Fortschr. der Neurol. und Psychiatrie 1903, S. 414.

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molecules du cerveau", sowie die von Kéraudren, der die Reibungen und Erschütterungen der Unterleibsorgane als Urheber der Seekrankheit ansah. Wollaston, der meinte, ein einfaches physikalisches Experiment auf eine komplizierte biologische Frage anwenden zu können, glaubte, das Blut werde bei niedergehender Bewegung des Schiffes (wie das Quecksilber in einem Barometer, das schnell sinkt) leicht dem Gehirn zufließen, indem der Rückfluß zu gleicher Zeit erschwert sei und infolgedessen entstünden Hirnkongestionen. Obgleich Wollastons Theorie einige Anhänger zählte, fand sie mehr Gegner, die in Hirnanämie die Ursache der Seekrankheit gefunden zu haben meinten. Auch fehlte es nicht an solchen, die den Mittelweg einschlugen, d. h., die die Erscheinungen der Seekrankheit einer beständigen Abwechslung von Anämie und Hyperämie des Gehirns zuschrieben. Diese Theorien der Zirkulationsstörungen sind nicht alle zu den mechanischen Theorien zu rechnen, denn viele dachten sich die Zirkulationsänderungen auf reflektorischem Wege entstanden. Die von Schwerdt gegebene Vorstellung der Zirkulationsstörungen, welche mit Wollastons Theorie einige Übereinstimmung hat, ist doch viel plausibler als jene, erstens, weil sie den Klappenapparat mit in Betracht zieht und zweitens, weil sie sich auf Teile des Zirkulationssystems bezieht, in denen nur sehr geringe Spannungen und kleine Stromgeschwindigkeiten herrschen. Außerdem aber sucht Schwerdt in jenen Zirkulationsstörungen nicht die einzige Ursache der Seekrankheit; er untersuchte sich selbst und war nicht seekrank. Pflanz[1]) fand manometrisch Blutdruckschwankungen, die abhängig von den Schiffsbewegungen waren. Sie waren aber nicht größer als diejenigen, welche die täglichen Bewegungen des Menschen begleiten, und wurden deswegen von ihm nur als Symptom, nicht als Ursache der Seekrankheit betrachtet.

Alle mechanischen Theorien haben den großen Nachteil, daß sie zwei wichtige Tatsachen (nämlich die Immunität der kleinen Kinder und die Gewöhnung an die Bewegungen des Schiffes) nicht befriedigend erklären können. Fonssagrives nannte selbst die Gewöhnung: „pierre d'achoppement de toutes les théories mécaniques du mal de mer" und verwarf aus diesem Grunde später wieder seine eigene Theorie.

Riese weist weiter darauf hin, daß so heftige mechanische Erschütterungen, wie diese Theorien voraussetzen, uns auf dem Meere nicht treffen. Ganz richtig bemerkt er: „Man lege den Kopf an die

1) O. Pflanz, Zur Ätiologie der Seekrankheit. Wiener klin. Wochenschr. 1903, S. 896.

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Wand eines schütternden Wagens oder selbst an die Bretterwand des verhältnismäßig leise dahingleitenden Eisenbahnwagens, und man wird ziemlich starke Erschütterungen empfinden, wenigstens mit denen verglichen, die man etwa verspürt, wenn man den Kopf an den Mast, an die Reiling oder sonst einen festen Bestandteil des Schiffes lehnt. Meist wird man hier überhaupt keine Erschütterung wahrnehmen."

Seeschiffe haben gewöhnlich eine so enorme Inertie, daß die Schiffsbewegungen sehr allmählich ineinander übergehen, ausgenommen sehr kleine Fahrzeuge, die oft heftig erschüttern und stoßen können.

Folgende Bemerkung Rieses:

„Das Bier bleibt auch bei starken [Schiffs-]Schwankungen ganz ruhig im Glase und fließt nur aus, wenn etwa das Glas zu stark geneigt wird; auch spritzt es weder hinaus, noch wird es hinausgeschleudert, es folgt eben einfach der Schwerkraft", ist im allgemeinen richtig, doch wo es sich um sehr kleine Schiffe oder sehr stürmisches Wetter handelt, kann ihre Zulässigkeit nur eine bedingte sein.

Aus seinen Wahrnehmungen schließt Riese, daß „die mechanischen Einwirkungen auf Schiffen weit geringer sind, als mancherlei mechanische Einwirkungen, welche der menschliche Körper bei den verschiedensten Gelegenheiten im gewöhnlichen Leben unbeschadet erträgt. Daß ein Reiter ein gefülltes Glas nicht wohl würde transportieren können, liegt auf der Hand, auch in einem gewöhnlichen Wagen würde der Inhalt wahrscheinlich durch mechanische Kräfte verschüttet werden, doch ich glaube, selbst beim gewöhnlichen Gange sind die Erschütterungen, denen der Körper ausgesetzt ist, größere, als sie ihm von dem Schiffe mitgeteilt werden."

Ungeachtet dieser zahlreichen und begründeten Einwände gegen jede mechanische Theorie der Seekrankheit, und obgleich völlig damit bekannt, hat vor kurzem Rosenbach[1]) aufs neue versucht, das Krankheitsbild mechanisch zu erklären. Nach ausführlichen Betrachtungen, in denen eine sachliche Widerlegung der oben genannten Bedenken meines Erachtens nicht zu finden ist, kommt Rosenbach zu dem Resultate (S. 161, Seekr. als Typus der Kinetosen):

„daß für die Entstehung des Symptomenkomplexes der Seekrankheit (und aller Kinetosen) vor allem intra- und interenergetische (intermolekulare) Störungen verantwortlich zu machen sind. Sie treten ein, wenn besonders starke und ungewohnte Impulse, in specie die

1) Prof. Dr. O. Rosenbach, Die Seekrankheit als Typus der Kinetosen. Wien 1896 und „Die Seekrankheit" in Nothnagels Path. und Ther. Bd. 12, T. 2.

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Schiffsbewegung, das durch eine besondere Form der Oberflächenspannung bewirkte künstliche (innere) Gleichgewicht des gesamten Organismus oder seiner einzelnen Teile (funktionellen Einheiten) so wesentlich in Frage stellen, daß die vorhandenen reaktiven Kräfte (die latente Reserveenergie) nicht imstande sind, die normalen Beziehungen der Teile wiederherzustellen. Wir nehmen also an, daß überall da, wo äußere oder innere Impulse zu einer starken, inter- und intramolekularen Erschütterung (Veränderung des dynamischen Gleichgewichtes) der kleinen und größeren Komplexe führen, die Störung zuerst in einer Anomalie der außerwesentlichen (intra- und interorganischen) Arbeit, d. h. in einer Veränderung der sämtlichen wahrnehmbaren (tonischen und sthenischen) Funktionen der betreffenden Organe respektive des ganzen Organismus, zum Ausdrucke kommen wird."

„Wir sind somit geneigt, die abnorme Sekretion des Magens, die abnormen peristaltischen Bewegungen, das Erbrechen usw., auf eine bestimmte mechanische Beeinflussung des Organgewebes selbst (eine Betriebsstörung) zurückzuführen, und glauben, daß Leber, Darm, Gehirn, Nervenplexus in derselben Weise direkt mechanisch in Mitleidenschaft gezogen werden, ohne daß dabei ein Nerveneinfluß primär im Spiele ist, obwohl wir die gleichzeitige (koordinierte) Alteration der Psyche und des Nervengewebes, das ja, wie jedes andere Gewebe ebenfalls mechanisch irritiert werden kann, durchaus nicht in Abrede stellen. Es kann natürlich in einem Falle das Gehirn allein, es können in einem anderen auch die Abdominalorgane für sich affiziert sein; doch sind wohl die Störungen in letzteren, sowie kombinierte Affektionen, die stärkeren und häufigeren."

Außer dieser mechanisch verursachten Form der Seekrankheit erkennt Rosenbach auch das Bestehen einer psychischen Form an, denn auf Seite 163 liest man weiter:

„Neben der Form der Seekrankheit, die auf durchaus realer Basis, also bei direkter Beeinflussung der sichtbaren Gehirnmasse durch grobe Schwingungen zustande kommt (somatische Form), müssen wir die auf rein psychischer Basis (oder höchstens durch geringe optische Sinnesreize) entstandene besonders berücksichtigen, da hier die Entstehung der Störungen auch ohne Zuhilfenahme eines Gleichgewichtszentrums allein aus der Erregung von Unlustgefühlen (Vorstellungen) erklärt werden kann, was namentlich dadurch bewiesen wird, daß die Krankheit auch bei vollkommen ruhiger See auftritt, und daß durchaus ähnliche Erscheinungen auch auf dem Festlande bei gewissen psychischen Erregungen beobachtet werden."

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Rosenbach geht in seinen energetischen Betrachtungen so weit, daß er die durch die Seekrankheit verursachten Störungen in einem Organismus einfach analog achtet mit einer Trennung der Kontinuität oder einer Veränderung des Aggregatzustandes bei nicht organisierten Massen:

„Wir bezeichnen den Effekt dieser Verschiebungen bei organisierten Komplexen als eine Veränderung des Tonus und der Funktion, bei nicht organisierten Massen als Veränderung des Aggregatzustandes." (S. 4, Seekrankheit als Typus der Kinetosen.)

Es liegt auf der Hand, daß sich diese Theorie schwer vereinigen läßt mit der Immunität der Säuglinge, was Rosenbach auch selbst eingesehen hat, und schon daraus hervorgeht, daß er die Säuglingsimmunität in ihrer Allgemeinheit leugnet. Soweit er aber doch die geringe Empfindlichkeit der kleinen Kinder zugeben muß, erklärt er sie wie folgt: (S. 200)

„Je kunstvoller also die Oberflächenspannung eines Körpers an sich ist, desto größer ist die Arbeit für die richtige Spannung der kleinen, die Komplexe der Organe zusammensetzenden Teile unter der Wucht eines mächtigen, ungewohnten Impulses, und darum werden die Störungen bei den überaus wirkungsvollen Schwankungen des Schiffes für die meisten Erwachsenen so beträchtlich. Die Bedeutung von Stößen, wie sie die Schiffsbewegung darstellt, ist dagegen für Körper ohne besondere Komplikation der Oberflächenspannung und des räumlichen Gleichgewichtes außerordentlich gering, und so empfindet der Säugling die Einwirkungen relativ am geringsten, da sein Organismus eben noch nicht die Einrichtungen des vollkommenen Präzisionsapparates für das Gleichgewicht aller Teile hat, wie der Organismus des Erwachsenen. Um ein Beispiel zu brauchen, so sind die kunstvollen Spannungen der Gewölbe der Paläste beim Erdbeben mehr gefährdet als die in einfachem Gleichgewichte konstruierten Hütten aus Holzfachwerk."

Auf Seite 17 derselben Arbeit gibt Rosenbach noch zahlreiche andere Argumente für die kindliche Immunität, die aber an Gehalt die obigen nicht viel übertreffen.

Rosenbachs philosophisch-energetische Betrachtungen machen oft den Eindruck, auf weit weniger festem Boden zu stehen, als ein Schiff im Sturm bietet. Wie er in der Tatsache der Gewöhnung an den Einfluß des stürmischen Meeres eine der wichtigsten Stützen für seine Theorie zu sehen vermag (S. 185, Seekr. als Typus d. Kinet.), ist mir aus seiner Darlegung nicht klar geworden.

Wie ich schon oben bemerkte, bilden die Theorien der Zirkulationsstörungen einen Übergang von den mechanischen zu den Reflex-

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theorien. Denn viele, die die Erscheinungen der Seekrankheit ableiten wollen aus einer Hyperämie oder Anämie des Gehirns, denken sich diese Zirkulationsstörungen auf mechanische Weise entstanden, wieder andere kümmern sich nicht um die Frage, wie die Anaemia cerebri zustande kommt, sondern konstatieren nur eine Übereinstimmung zwischen akuter Hirnanämie und Seekrankheit.

Hagen-Torn[1]) endlich (und mit ihm viele andere) sieht in der Seekrankheit: „eine Reflexerscheinung, bedingt durch die Unmöglichkeit der Anpassung an die stets sich verändernden Beziehungen des Körpers zur Umgebung. Das Ende der Reflexschleife ist die Kontraktion der Gehirngefäße, wie Prof. Binz es annimmt".

Binz[2]) betrachtet Anaemia cerebri als nächstliegende Ursache der Seekrankheit und beruft sich auf Kramer[3]), der bei 44 Seekranken anämische Fundus oculi fand. — Reynolds[4]) meint, die Erscheinungen der Seekrankheit wären bedingt durch Druckschwankungen in den Ampullen der Canales semicirculares, während Butler-Savory[5]) genau den ganzen Reflexbogen beschreibt, ausgehend von „the two terminal branches of the auditory nerve to the cochlea and semicircular canals. The upper division of the auditory nerve is connected at the base of the internal auditory meatus with the geniculate ganglion of the facial nerve and this latter in its turn gives off fibres which pass to the trunk of the pneumogastric by means of its auricular branch; there is thus a complete chain of nerve fibres starting from the terminal branches of the auditory and finishing with the terminal branches of the pneumogastric, distributed over both surfaces of the stomach. The stimulation of the auditory nerve would be caused by the movement of the endolymph, set up by the motion of the ship. From there the stimulation would pass through the various secretory and vase-dilator fibres of the fascial to the submaxillary and sublingual glands &c.«

Diejenigen Theorien, die die Seekrankheit als verursacht ansehen durch ungewöhnliche Reize des Labyrinths, stützen sich auf die Übereinstimmungen zwischen den Erscheinungen der Seekrankheit und denjenigen, welche durch künstliche Reize, Beschädigung oder Exstir-

1) O. Hagen-Torn, Über die Seekrankheit. Zentralbl. f. Inn. Med. 1903, S. 697.

2) Karl Binz, Über die Seekrankheit. Zentralbl. f. Inn. Med. 1903, Nr. 9 und 1904, Nr. 11.

3) L. Kramer, Über die Seekrankheit. Prager Med. Wochenschr. 1892, Nr. 40 und 41.

4) Reynolds, On the Nature and treatment of sea-sickness. Lancet 1884, p. 1161.

5) Butler Savory, British Med. Journal 1901, I, p. 767.

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pation der Canales semicirculares auftreten. Sie können ohne Zweifel am Krankheitsbilde viel erklären, aber vernachlässigen mit Unrecht diejenigen Reize, welche auf optischen und kinästhetischen Bahnen zur Entstehung von Desorientierung beitragen und die, wenn sie allein auftreten, ebenfalls einen der Seekrankheit ähnlichen Symptomenkomplex erwecken. So versuchte u. a. Abrahamsz das Bild der Seekrankheit ganz von letztgenannten Ursachen (ungewöhnliche Gesichts- und Gefühlsempfindungen) abzuleiten. Seine Vorstellung und die oben beschriebene ergänzen sich gegenseitig und zeigen dann große Übereinstimmung mit den von Riese gegebenen Darlegungen über die Entstehung der Seekrankheit. Nur wird seine Neigung zur Lokalisierung eines statischen Zentrums und eines pathologisch-anatomischen Ortes der Seekrankheit heute weniger Anhänger finden als damals, als er seine Inauguraldissertation schrieb und man mit Lokalisieren größeren Erfolg hatte als jetzt.

Anhänger von rein psychischen Theorien werden heute wahrscheinlich sehr selten sein, obgleich eine rein psychische Form der Seekrankheit ebensowenig verneint werden kann wie Schwindel auf ausschließlich psychischer Grundlage. Schwindel beim Hinabsehen von hohen Gebäuden, oder bei einseitiger Augenmuskellähmung, oder bei einer wohlbekannten Kirmesbelustigung, welche den fast in Ruhe bleibenden Besuchern die Illusion gibt, 360 Grad rundzuschaukeln (sog. Hexenschaukel), sind dafür einige aus vielen Beispielen. Daß hysterische Personen auf nahezu und sogar auch auf ganz unbewegtem Schiffe Seekrankheitserscheinungen zeigen können, wird niemand wundern, der mit Hysterie ein wenig bekannt ist. Aber auch bei Gesunden spielen wahrscheinlich psychische Einflüsse eine große Rolle. Dafür sprechen verschiedene Umstände, wie z. B. die schon zitierte Wahrnehmung einiger Schriftsteller, daß man willkürlich das Ausbrechen der Seekrankheitserscheinungen einige Zeit unterdrücken kann, oder man wenigstens den Eindruck hat, es zu können. Dafür spricht ferner der so oft konstatierte Einfluß psychischer Infektion, die besonders bei Seekrankheit von großer Bedeutung ist und vielen aus Erfahrung bekannt sein wird. Weiter ist die Tatsache bekannt, daß seekranke Neulinge unter den Matrosen unter der suggestiven Behandlung ihrer älteren Kameraden sich schnell zu gewöhnen pflegen. Déry[1]) meint, das bei alten Seeleuten für solche Fälle bekannte Rezept, die Darreichung von Seewasser in- und auswendig, im Notfalle aufgezwungen, solle sich gut bewähren. — Solange die Homöopathie dafür keine bessere Erklärung an die Hand gibt, wird man in dieser Therapie

1) Déry, Die Seekrankheit. Allgem. milit.-ärztl. Zeitung 1871.

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schwerlich etwas anderes als suggestive Wirkung vermuten können. — Und endlich das so häufige Bestreben, Empfindlichkeit für Seekrankheit zu verneinen, und in Seekrankheit nur eine Schwäche oder Mangel an Willenskraft zu sehen. „Man erhält", sagt Rosenbach, „von Kriegern und Seekranken nur selten wahrheitsgetreue Aufschlüsse", und Neuhaus meint: „Wollte man den üblichen Prahlereien Glauben schenken, so würden mindestens 90% nie seekrank", indem Riese hierzu bemerkt: „Es ist ein jedem Schiffsarzt bekannter Umstand, daß Passagiere, besonders Herren, die an nicht zu schwerer Seekrankheit leiden, dies nie gern wahrhaben mögen, sondern ihre Indisposition lieber auf alle möglichen anderen Dinge, Diätfehler usw., zu schieben pflegen."

Obige Übersicht der Seekrankheitstheorien kann in keiner Hinsicht auf Vollständigkeit Anspruch machen, es ist nicht viel mehr als eine Blumenlese aus der so umfangreichen Literatur. Man kann kaum einen Jahrgang von irgendeiner medizinischen Zeitschrift zur Hand nehmen, ohne darin auf neue Theorien der Seekrankheit zu stoßen. Es ist kaum möglich, die ganze Literatur zu übersehen, und ich habe nur nach der Aufspürung und Erwähnung der wichtigsten Grundlagen der zahlreichen Theorien getrachtet.

Wenn wir nun die Seekrankheit betrachten als eine bewußte Desorientation im Raume, kombiniert mit einer Reihe reflektorischer Störungen, eins wie das andere hervorgebracht von ungewöhnlichen optischen, kinästhetischen und Labyrinthreizen, dann werden die Seite 485 genannten Eigentümlichkeiten der Krankheit befriedigend erklärt werden können. Die sub 1 und 2 genannten Kriterien brauchen keinen weiteren Kommentar. Daß Säuglinge, die noch jeder Orientierung im Raume entbehren, immun sind für Desorientierung, kann unsere Betrachtung nur stützen, daß sie aber auch unempfindlich sind für die Labyrinthreize, die sie auf beweglichem Schiffe treffen, könnte nicht a priori erwartet werden. Es ist mir nicht gelungen, in der Literatur Wahrnehmungen beschrieben zu finden, die auf Unempfindlichkeit der Säuglinge für Achsendrehung, galvanische oder thermische Labyrinthreize hinweisen. Der hiesige Herr Professor Schutter, mit dem ich hierüber Rats pflog, war auf meine Bitte bereit, diese Frage mit mir zu untersuchen; wir fanden, daß 6 Kinder unter 1 Jahre alt keinen kalorischen Nystagmus zeigten. Untersucht wurde mit Wasser von 25° Celsius, wobei normale ältere Kinder eine kräftige und minutenlang nach dem Experimente nachbleibende Reaktion zeigten. Ein in der Entwickelung zurückgebliebenes 2jähriges Kind, das wegen kongenitaler Hüfteluxation nicht gehen noch stehen konnte, zeigte

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auch keinen kalorischen Nystagmus, während ein normales $1^1/_2$jähriges Kindchen eine Spur von Reaktion zeigte. Aus diesen Untersuchungen wird es wahrscheinlich, daß der Vestibularapparat in der Zeit funktionsfähig wird, wo die Kinder das Gehen erlernen.

Die für Seekrankheit größere Empfindlichkeit der Frauen ist in Übereinstimmung mit ihrer allgemeinen höheren Irritabilität, und demzufolge sind auch nervöse und hysterische Naturen besonders empfindlich.

Die Tatsache der Gewöhnung, und die Frage, wie diese zustande kommt, verdient besondere Aufmerksamkeit, insofern die Frage für die Prophylaxe der Seekrankheit große Bedeutung hat.

Vergleicht man den erfahrenen Seemann mit dem Neuling auf dem beweglichen Boden, dann fallen uns bedeutende Unterschiede auf. Der Neuling bewegt sich sowohl objektiv wie subjektiv in sehr unsicherem Gleichgewichte. Er muß sich fortwährend stützen; und will er sich von einem Ort zum anderen bewegen, so tut er es am liebsten in dem Augenblicke, wo das Verdeck durch die Horizontalfläche geht. Zur Fortsetzung seines beabsichtigten Rückganges wartet er wiederum eine gleiche Gelegenheit ab. Wenn er eine Schiffsbewegung mit einer zweckmäßigen Körperbewegung zu beantworten versucht, tut er dies nur selten in der richtigen Weise und erinnert dabei an den Schlittschuhläufer oder Radfahrer, der sich zum erstenmal an das Studium dieses Sports wagt. Der erfahrene Seemann hingegen ist vollkommen gut equilibriert. Beim Stehen, Gehen, Sitzen: fortwährend kompensiert er automatisch die Schiffsbewegungen derart, daß sein Oberkörper immer ungefähr die vertikale Lage behält. Der erfahrene Aufwärter z. B. trägt ohne Mühe ein Brett mit gefüllten Gläsern zu den Passagieren als etwas Selbstverständliches, ohne eine Miene zu verziehen. Auf Photographien, welche am Bord fahrender Schiffe aufgenommen sind, kann man sich leicht von diesem Equilibrieren der Seeleute überzeugen. Viele, wenn nicht die meisten, tun es ganz unbewußt, oder denken nur daran, wenn die Schiffsbewegungen sehr groß werden. Wo dieses Equilibrieren zeitweise unterlassen wird, z. B. wenn man sich in irgendeine Arbeit zu sehr vertieft, tritt bisweilen überraschend wieder ein Gefühl von Unbehagen auf, welches dadurch zu bekämpfen ist, daß man seine Arbeit auf einige Zeit unterbricht. So sah ich einmal einen erfahrenen, und sonst für Seekrankheit nicht besonders empfänglichen Heizer auf nur mäßig bewegtem Schiffe unwohl werden beim Bemalen eines Brettes, das zu einer festlichen Gelegenheit dienen sollte und von diesem Heizer (der, wie ich glaube, früher Anstreicher gewesen war) bei fahrendem Schiffe bearbeitet wurde. Beim Schreiben und Lesen während stürmischen

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Wetters bemerkte ich oft auch an mir selbst, daß dann und wann Unterbrechung dieser Arbeit nötig war, um mein Wohlbefinden zu behalten. Wie aus einer schon früher zitierten Bemerkung von Abrahamsz hervorgeht, machte dieser Forscher auch dieselbe Erfahrung. Das fortwährende Equilibrieren verursacht nach einer Seefahrt auf kleinem, beweglichem Schiffe bei ungünstigem Wetter beim Anslandtreten ein Gefühl in den Füßen, welches an dasjenige erinnert, das man bei den ersten Schritten nach einer Schlittschuhfahrt hat.

Das Equilibrieren ist ein wichtiges Wehrmittel gegen die Schiffsbewegungen, die dadurch als Labyrinthreize sehr geschwächt werden. Leider aber kann man die Bewegungen in senkrechter Richtung nicht kompensieren, und wahrscheinlich steht deswegen das „Stampfen" in weit schlechterem Rufe als das „Rollen" des Schiffes. Die Frage taucht jetzt auf, ob das schon beschriebene chronische Fortbestehen der Seekrankheits-Erscheinungen bei Überempfindlichen dem mangelhaften Erlernen des zweckmäßigen automatischen Kompensieren der Schiffsbewegungen zugeschrieben werden kann. Zur Beantwortung dieser Frage wurde von mir untersucht, ob Überempfindlichkeit für Seekrankheit zusammengeht mit Störungen der Empfindlichkeit für Neigungsänderungen. Mit Hilfe eines improvisierten Goniometers, das mit einer Winde geräuschlos bewegt werden konnte, wurden bei ungefähr 100 Schiffspersonen nacheinander folgende Größen bestimmt:

1. die Schwelle (Schw).

2. die kleinste Neigungsänderung, die nötig war, um aufs neue eine Empfindung zu geben (dR).

3. die Minimum-Neigungsänderung, nötig, um eine Empfindung zu geben, ausgehend von einer Neigung von 10° (dR^{10}).

4. derselbe Wert bei einem Neigungswinkel von 20° (dR^{20}).

Immer wurden die Personen ohne Schuhe, mit herabhängenden Armen, geschlossenen Augen und bei gleichem Stande der Füße untersucht. Die Werte für: Schw, dR, dR^{10} und dR^{20} wurden gemessen in zwei verschiedenen Stellungen, die die Stampf- und Rollbewegung eines Schiffes nachahmten. Da aber die für seitliche Neigungsänderungen gefundenen Werte nicht nennenswert verschieden waren von denen, die bei Neigungen nach hinten und vorn gefunden wurden, nehmen wir jedesmal aus beiden den mittleren Wert. Eine gleiche Geschwindigkeit von Neigungsänderung bei jeder Untersuchung wurde möglichst dadurch verbürgt, daß immer derselbe Seesoldat die Winde drehte. Die Forderung, daß die Bewegungen übereinstimmend sind, und daß man sie für genaues Ablesen jedesmal muß hemmen können, ohne daß dabei fühlbare Stöße entstehen, bestimmt die größte Geschwindigkeit, die man brauchen kann. Diese Geschwindigkeit wird ungefähr

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übereinstimmen mit derjenigen, die ein tüchtiges Seeschiff bei mäßig bewegter See zeigt.

Jeder gefundene Wert wurde einige Male kontrolliert, und aus mehreren Wahrnehmungen jedesmal ein Mittelwert berechnet. Oft geschah es, daß die erste Neigung erst bei 6, 8 oder mehr Graden perzipiert wurde, obgleich doch niemals die Schwelle so hoch war. Ließ man doch die Neigung wieder abnehmen, dann wurde schon „Nieder" gerufen, ehe wieder der Horizontalstand erreicht war, und ein zweites Mal wurde schon bei viel weniger als 6 Graden das „Auf" gehört. Ausgenommen die ersten unrichtigen Wahrnehmungen, stimmten die gefundenen Werte bei Wiederholung der Untersuchungen gut überein.

Aus 100 Wahrnehmungen wurden folgende Mittelwerte berechnet:

Schw:	dR	dR^{10}	dR^{20}
1,96°	1,45°	1,29°	1,23°
		1,53°	1,64°

Für dR^{10} und dR^{20} wurden zwei Werte gefunden, da sich zeigte, daß gewöhnlich die Empfindlichkeit für Neigungszunahme bedeutend größer war als für Abnahme. Die oberhalb des Striches stehenden Ziffern haben immer Beziehung auf Zunahme, die unterhalb desselben stehenden auf Abnahme der Neigung. Die untersuchte Schiffsmannschaft bestand aus Personal der niederländischen Marine und war zusammengesetzt aus Leuten von verschiedenem Alter und in Übereinstimmung damit von verschiedener Befahrenheit. Da die älteren dieser Seeleute oft auffallend hohe Ziffern zeigten, so lag es auf der Hand, die Untersuchten in zwei Altersklassen einzuteilen, wobei folgende Mittelwerte gefunden wurden:

Tabelle I.

	Geboren vor 1880 Mittelwerte aus 37 Fällen	Geboren nach 1880 Mittelwerte aus 63 Fällen
Schw:	2,20°	1,82°
dR	1,60°	1,36°
dR^{10}	1,35°	1,26°
	1,57°	1,52°
dR^{20}	1,27°	1,21°
	1,72°	1,59°

Die älteren, mehr befahrenen Seeleute (d. h. seetüchtige, mit Schiffsarbeiten vertraute Leute, die schon ozeanische Reisen gemacht haben; der Gegensatz dazu sind Halbbefahrene und Unbefahrene)

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zeigten also immer höhere Ziffern als die jüngeren. Ähnliche Unterschiede in Empfindlichkeit wurden auch gefunden, wenn man das Jahr 1885 nimmt als Grenze zur Verteilung in zwei Altersgruppen:

Tabelle II.

	Geboren vor 1885 Mittelwerte aus 60 Fällen	Geboren nach 1885 Mittelwerte aus 40 Fällen
Schw:	2,12°	1,73°
dR	1,54°	1,32°
dR^{10}	1,33° 1,61°	1,24° 1,41°
dR^{20}	1,27° 1,69°	1,17° 1,49°

In Westindien, wo ich diese Untersuchungen anstellte, hatte ich keine Gelegenheit, ganz Unbefahrene (Leute, die noch nie auf dem Meere waren), zu untersuchen, was nötig gewesen wäre, um ausfindig zu machen, ob im allgemeinen das Alter Einfluß auf die Empfindlichkeit für Neigungsänderungen ausübe, oder ob die geringere Empfindlichkeit der älteren Seeleute einer gewissen Abstumpfung als Folge des langen Aufenthaltes auf dem Meere zugeschrieben werden müsse.

Hier in Groningen hatte ich Gelegenheit, Unbefahrene zu untersuchen. Ich hatte nun ein Goniometer nach v. Stein zu meiner Verfügung und die mit diesem Apparate gefundenen Werte können nicht ohne weiteres mit denen verglichen werden, die mit Hilfe meines primitiven Goniometers gefunden wurden. Aber auf Unterschieden der Empfindlichkeit, abhängig von Alter und Geschlecht, konnte mit dem genauern Apparate um so besser untersucht werden. Aus 100 Wahrnehmungen bei Personen in dem Alter von 15—50 Jahren wurden folgende Mittelwerte berechnet:

Tabelle III.

	Geboren vor 1885 50 Fälle	Geboren nach 1885 50 Fälle
Schw:	0,9425°	0,94°
dR	0,869°	0,836°
dR^{10}	0,763° 0,988°	0,729° 1,063°
dR^{20}	0,663° 0,983°	0,674° 0,997°

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Wie aus obiger Tabelle hervorgeht, wurden bedeutende Unterschiede, die abhängig vom Alter waren, nicht gefunden. Offenbar sind die Ziffern von Tab. III bedeutend niedriger als die der beiden ersten Tabellen. Dieses wird wohl teils den Unterschieden der bei beiden Untersuchungsreihen benutzten Apparate zugeschrieben werden müssen, teils aber auch der Unbefahrenheit der zuletzt untersuchten Personen. Die auf mich selbst sich beziehenden Werte, die ich mit Hilfe des Goniometers nach v. Stein fand, waren etwas größer als die Hälfte der früher mit dem einfachen Apparate gefundenen Werte. Aber damals diente ich auf einem fahrenden Schiffe, während ich mich jetzt seit mehr als einem Jahre auf dem Lande aufhalte.

Bei Vergleichung beider Geschlechter wurden folgende Werte gefunden:

Tabelle IV.

	Männer 50 Fälle	Weiber 50 Fälle
Schw:	0,985°	0,844°
dR	0,92°	0,79°
dR^{10}	0,78°	0,729°
	1,034°	1,008°
dR^{20}	0,644°	0,654°
	0,952°	0,963°

Möglicherweise sind also die Weiber etwas empfindlicher für Neigungsänderungen als die Männer, aber die Unterschiede sind sehr klein und verschwinden ganz bei Zunahme der Neigung.

Vergleichen wir jetzt von 6 für Seekrankheit überempfindlichen Seeleuten die für sie gefundenen Werte mit denjenigen, die auf Grund ihrer Befahrenheit zu erwarten waren, dann finden wir:

Tabelle V.

	Schw.		dR		dR^{10}		dR^{20}	
	Erwart.	Wahrn.	Erwart.	Wahrn.	Erwart.	Wahrn.	Erwart.	Wahrn.
Nr. 1 geb. 1866	2,20	1,75	1,60	1,75	1,35 1,57	1,5 1,5	1,27 1,72	1,— 1,5
„ 2 „ 1870	id.	2,—	id.	1,5	id.	1,25 1,5	id.	1,— 1,75
„ 3 „ 1882	1,82	1,25	1,36	1,—	1,26 1,52	1,25 1,25	1,21 1,59	1,5 1,—
„ 4 „ 1882	id.	1,—	id.	1,—	d.	0,75 1,—	id.	1,25 1,5
„ 5 „ 1887	1,73	1,—	1,32	1,—	1,24 1,41	1,— 1,25	1,17 1,49	1,25 1,5
„ 6 „ 1888	id.	2,5	id.	1,75	id.	1,5 1,5	id.	1,5 1,—

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Daß im allgemeinen die für Seekrankheit Überempfindlichen für Neigungsänderungen nicht weniger empfindlich sind als andere, sondern eher empfindlicher, geht hieraus hervor. Die Unterschiede sind aber gering und außerdem schwankend. Nummer 6 bezog sich auf den Matrosen, dessen Krankengeschichte oben beschrieben wurde, und der von allen am meisten überempfindlich für Seekrankheit war und doch für Neigungsänderungen Unterempfindlichkeit zeigte. Für die Diagnose der Überempfindlichkeit für Seekrankheit sind diese Untersuchungen offenbar unzureichend. Es muß jetzt noch auf eine Besonderheit der Ziffern hingewiesen werden, die in der Tabelle mit einem * angedeutet ist. Obwohl gleich große Zahlen für Zu- und Abnahme der Neigung auch bei Nichtüberempfindlichen für Seekrankheit nicht selten waren, so wurde ein solches umgekehrtes Verhältnis, das auf größere Empfindlichkeit für Abnahme als für Zunahme der Neigung hinweist, nur bei diesen beiden für Seekrankheit Überempfindlichen gefunden. Ob es von Bedeutung ist, wage ich nicht zu entscheiden, jedenfalls aber wäre es eine Anregung, zu untersuchen, ob vielleicht auf einem Fahrstuhle ein ähnlicher inverser Typus deutlicher zum Vorschein kommen würde. Leider hatte ich dazu keine Gelegenheit.

Aus obigem können wir schließen, daß Überempfindlichkeit für Seekrankheit nicht mangelhafter Empfindlichkeit für Neigungsänderungen und infolgedessen unvollständiges automatisches Equilibrieren zugeschrieben werden kann. Dagegen spricht außerdem die Tatsache, daß chronische Seekranke oft zwar das Rollen, aber nicht das Stampfen des Schiffes vertragen. Es ist deswegen wahrscheinlicher, daß Überempfindlichkeit für Seekrankheit auf einer Reflexhyperästhesie beruht. Die bewußte Desorientierung, die immer die akute Seekrankheit begleitet, fehlt der chronischen, die möglicherweise ganz aus reflektorischen Störungen gebildet wird. Die Idiosynkrasie für Tabak, die die meisten Überempfindlichen für Seekrankheit zeigen, weist auch (mit Rücksicht auf die große Ähnlichkeit von akuter Nikotinvergiftung und Seekrankheit) in jene Richtung. Auch mäßige und sogar starke Raucher pflegen bei stürmischem Wetter oft den Gebrauch dieses Genußmittels zu mäßigen oder zeitweise ganz einzustellen, weil wahrscheinlich Nikotin die Reizbarkeit derjenigen Teile des Nervensystems erhöht, die auch von den Schiffsbewegungen irritiert werden. Aus später von mir eingezogenen Erkundigungen hat sich noch wiederholt ergeben, daß Überempfindlichkeit für Seekrankheit und für Nikotin oft zusammengehen. Die Antithese hierzu trifft aber nicht zu. Nichtraucher sind keineswegs immer besonders empfindlich, weder für Nikotin, noch für Seekrankheit. Starke Raucher scheinen aber

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selten oder niemals zu den für Seekrankheit Überempfindlichen zu gehören.

Prophylaxis.

Obgleich die Natur uns den Weg selbst gezeigt hat, auf welchem man die Verhütung der Seekrankheit erhoffen kann, so hat man in dieser Richtung doch nur selten ernstliche Versuche angestellt und niemals mit einiger Beharrlichkeit durchgeführt. — Die Versuche, sich gegen Seekrankheit mittels nachgeahmter Schiffsbewegungen zu immunisieren, datieren schon aus dem 18. Jahrhundert, als Erasmus Darwin verordnete, durch Schwingungen in einer Schaukel einige Wochen vor Beginn einer Seefahrt sich an ungewöhnliche Bewegungen zu gewöhnen. v. Rochlitz[1]) erfand einen Apparat, um die Schiffsbewegungen nachzuahmen, den er „Philatlanticum" nannte und der auf der Wiener Weltausstellung ausgestellt war. Rosenbach[2]) beschreibt einen Apparat, „Krinoline" genannt, welcher Glockenform hat. „Die mit äußeren Sitzen versehene, in der gebräuchlichen Weise aufgehängte Glocke (Krinoline) wird vermittelst eines im Innern befindlichen Seiles abwechselnd um ihre Längsachse gedreht und periodisch nach abwärts gezogen, so daß etwa eine schraubenförmige Schaukelbewegung mit recht beträchtlicher Höhe der Windungen resultiert."

Die Bewegungen, die von diesen Apparaten ausgehen, haben große Ähnlichkeit mit denjenigen des heutigen Karussells. Obgleich mit Hilfe dieser Apparate Erfolg bis jetzt noch nicht erreicht wurde, erwartete auch Rosenbach von ähnlichen Versuchen gute Erfolge und sieht in künstlicher Gewöhnung nichts „schlechtweg Unerreichbares", aber: „Diese Apparate geben, vielleicht mit Ausnahme des vorher geschilderten glockenförmigen, zwar die Form, aber durchaus nicht die genauen Verhältnisse der Schwankungen eines größeren Schiffes wieder; denn die Schwingungen und ihre Wucht sind zu klein."

In der Tat wird man von den Übungsapparaten einen genügend großen Ausschlag der Schwingungen fordern müssen. Von der Wucht aber und deren Bedeutung für die Frage der Seekrankheit scheint Rosenbach sonderbare Vorstellungen gehegt zu haben. Denn Seite 198 lesen wir: „Das Gefühl des Fallens beim Herabgleiten des Schiffes oder, richtiger, das Bewußtsein der Innervationsimpulse, die wir geben müßten, um den Fall aufzuhalten, und die reaktive Arbeit der unter diesen Umständen ohne Ichbewußtsein (reflektorisch) ihren Zusammenhang erhaltenden Massenteile (Organe) wird jedenfalls viel beträcht-

1) K. v. Rochlitz, Die Seekrankheit und das Mittel sie zu verhüten, das Philatlanticum. Pest 1873.

2) O. Rosenbach, Die Seekrankheit als Typus d. Kinetosen. S. 19.

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licher sein, als die Empfindungen und Bewegungsimpulse, die unter gewöhnlichen Verhältnissen bei einem kaum 10 Fuß tiefen Falle ausgelöst werden, da eben die besondere Wucht der fallenden Masse des Schiffes, die wir nolens volens mit unseren Reaktionsbestrebungen kompensieren müssen, [eine schwere Arbeit!] besonders in Betracht kommt." „Mit andern Worten: Die Wucht des mit dem Schiffe fallenden Organismus entspricht nicht der Körpermasse (und der Höhe des Falles), sondern der Körpermasse plus der Masse des Schiffes, so daß in der bekannten Formel $\frac{M v^2}{2}$ für M die Masse des Schiffes plus der des Körpers zu setzen ist."

Und etwas weiter: „Der Effekt des Impulses, den wir als Wucht der Bewegung bezeichnen, und von dem die Anforderungen an die Kompensationsfähigkeit des Organismus abhängen[1]), muß beim Aufsteigen wesentlich kleiner sein als beim Absteigen" usw.

Und endlich Seite 199:

„Die verschiedene Wucht der beiden Bewegungsformen, der kinetische Effekt resp. die Größe der abnormen Wirkung des Stoßes auf den Körper[2]) würde sich also etwa durch die Formel $\frac{(M+m)s}{2t}$ (beim Fallen) und $\frac{(M-m)s}{2t}$ (beim Steigen) ausdrücken lassen" usw.

Aus obigem geht genügend hervor, welche Bedeutung Rosenbach der kinetischen Energie des Schiffes für das Entstehen der Seekrankheit beimißt. In Übereinstimmung damit glaubt er auch mit Unrecht, daß man auf kleinen Schiffen weniger der Gefahr ausgesetzt ist, seekrank zu werden, als auf großen Schiffen, und daß „das Fahren im Kahne nur selten die Erscheinungen der Seekrankheit hervorruft". — Die Behauptung Abrahamsz', „daß derjenige, der vom Verdecke einer großen Fregatte lachend Stürme hat angesehen, oft ein ganz anderes Gesicht macht bei einer Kahnfahrt auf einer Reede bei stürmischem Wetter", wird bei erfahrenen Seeleuten ohne Zweifel mehr Glauben finden. Von einem im Dienste zur See grau gewordenen Kauffahrteikapitän erfuhr ich einmal, daß er, als er in einer Ferienzeit eine kleine Vergnügungsreise mit einem Fischer mitmachte, auf dem kleinen Fischerbote so seekrank wurde, daß der Schiffer die Vermutung aussprach: sein Gast sei wahrscheinlich noch nie auf dem Meere gewesen. Auch meine eigene Erfahrung ist ganz in Widerspruch mit der Meinung Rosenbachs. Meine Laufbahn zur See fing ich auf einem kleinen Schiffe an, einem

1) Von mir gesperrt.
2) Von mir gesperrt.

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120 Tonnen großen Schoner. Während der ersten und auch sofort etwas stürmischen Tage war ich ordentlich seekrank, und auch später fühlte ich mich auf diesem Schiffchen bei stürmischem Wetter oft unbehaglich. Bei Wechsel der Mannschaft wurden in der Regel die neu angekommenen Matrosen auf diesem Schiffe seekrank, auch dann, wenn es schon mehr oder weniger erfahrene Seeleute waren.

Auf großen Schiffen bin ich später niemals seekrank gewesen, obgleich ich oft stürmisches Wetter hatte. Während einer Kahnfahrt von einigen Stunden Dauer längs der Küste von Curaçao, bei schönem Wetter und nur mäßig bewegtem Meere, sah ich einmal von etwa einem Dutzend erfahrener Seeleute 3 seekrank werden. Diese, für Seekrankheit nicht Überempfindlichen, würden bei gleichem Wetter auf einem großen Schiffe nicht die geringste Spur von Seekrankheit gefühlt haben. Eine Wahrnehmung Rosenbachs, daß bei Passage auf Kähnen durch starke Brandung gewöhnlich niemand von der Seekrankheit ergriffen wurde, muß wahrscheinlich der zu geringen Dauer jener Kahnfahrten zugeschrieben werden.

Aber in einiger Hinsicht haben doch kleine Schiffe Vorzüge vor großen. Die Schiffsbewegungen sind auf kleinen Fahrzeugen besser zu übersehen, sie sind außerdem einfacher und die Orientierung ist deshalb leichter. Aber ein kleines Schiffchen wird jedesmal von nur einer Welle bewegt, und infolge der großen Leichtigkeit leistet es weniger Widerstand gegen den Wechsel der Bewegungen, so daß die verschiedenen Phasen schnell und oft stoßweise ineinander übergehen. Infolge der geringen Länge ist beim Stampfen der Hebelarm kurz, und ungeachtet sehr großer Winkelausschläge bleiben die Bewegungen in vertikaler Richtung relativ klein. Da aber kleine Schiffe von jeder Welle im Ganzen gehoben werden, wird ihnen dabei jedesmal eine Bewegung in vertikale Richtung mitgeteilt ungefähr so groß wie der Höheunterschied zwischen Wellengipfel und Wellental. Große Schiffe hingegen werden von mehreren Wellen gleichzeitig bewegt. Ihr enormes Trägheitsvermögen leistet jeder Bewegungsänderung großen Widerstand, und macht dadurch die Bewegungen sehr langsam und allmählich ineinander übergehend. Die resultierende Bewegung ist immer eine Funktion zahlreicher Faktoren, die teils abhängig sind von äußeren Umständen (die verschiedenen Wellenschlagslängen der Meere; die Kursrichtung des Schiffes im Verband mit der Wellen- und Windrichtung; und die zeitliche Beschaffenheit vom Winde und Meer), teils aber von Eigenschaften des Schiffes selbst, namentlich von Form, Größe, Masse und Geschwindigkeit des Fahrzeuges. Jedes Schiff hat deswegen einen ihm eigenen Bewegungsmodus, der auf kleinen Schiffen im allgemeinen schnell ist mit großen Winkelaus-

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schlägen; auf großen Schiffen träge und mit kleineren Winkelausschlägen. Die Reize, die uns auf kleinen Schiffen treffen, sind also im Vergleich mit denen auf großen Schiffen zahlreicher, der Übergang ist schneller, aber die Orientierung ist leichter und die Bewegungen in senkrechter Richtung sind möglicherweise etwas kleiner. Theoretisch kann man daraus nicht schließen was besser ertragen wird, die Bewegungen der kleinen, oder die der großen Schiffe, aber die Erfahrung spricht zugunsten der letztgenannten. Dieses hat für die Prophylaxe große Bedeutung. Rosenbach fordert von seinem Standpunkte zur künstlichen Gewöhnung an die Schiffsbewegungen Übungsapparate von so enormen Massen, daß sie in dieser Hinsicht mit großen Seeschiffen zu vergleichen sind. Von unserm Standpunkte aus ist jene Forderung ganz unnötig, um so mehr weil man sich auf kleinen Schiffen mehr oder weniger überimmunisieren kann für die Bewegungen der größeren, während das Umgekehrte nicht möglich ist. Nötig für eine Übungsschule wäre unseres Erachtens ein Zimmerchen von einigen Meter im Quadrat, worin man durch Aufhängen von Gardinen, Pendeltischen, Hängelampen usw. Sorge trägt für die nötigen optischen Reize. Das Zimmerchen muß bewegbar sein um zwei senkrecht aufeinander stehende Achsen, die im Gleichgewichtsstande beide in der Horizontalfläche liegen müssen. Es muß sowohl um jede Achse besonders als um beide gleichzeitig bewegbar sein. Um eine der beiden Achsen soll die Bewegung einen Hebelarm haben von wenigstens 12 m Länge, damit die Ausschläge genügend groß werden.

Technisch wird man dies dadurch erreichen können, daß man eine Bahn bildet bestehend aus einem Teil eines Kreisbogen, von 12, oder wo nötig, mehr Meter langem Radius, und längs demselben die improvisierte Schiffskajüte bewegen läßt. Man wird aber auch denselben Effekt erreichen können mit Hilfe eines Apparates, der außer Bewegungen in senkrechter Richtung, wie ein Fahrstuhl, gleichzeitig Drehbewegungen zuläßt, um zwei, im Gleichgewichtsstande beide in der Horizontalfläche gelegene, Achsen. Möglichst muß man die Bewegungen in Übereinstimmung zu bringen suchen mit den Schiffsbewegungen. Der erfahrene Seemann fühlt an einer Schiffsbewegung wie ungefähr die folgende ausfallen wird, auch dann, wenn er sich im Unterschiffe befindet. Wahrscheinlich wird dies darauf beruhen, daß nach einem Ausschlag in irgendeiner Richtung der aus der Vertikale bewegte Schwerpunkt des Schiffes dahin zurückneigt. Infolgedessen bestehen die Schiffsbewegungen aus einem Gemisch vieler interferierenden Pendelbewegungen, die jede absonderlich den Pendelbewegungstypus (Geschwindigkeit umgekehrt proportional dem Sinus des Ausschlags-

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winkels) deutlich zeigen würden, aber auch in ihrer Zusammenwirkung dessen Charakter immer mehr oder weniger beibehalten.

Schreitet man in systematischer Weise von einfachen zu mehr zusammengesetzten Bewegungen, so wird man das zweckmäßige Equilibrieren wahrscheinlich sehr schnell lernen können, und es wird leicht sein dies, wenn nötig, mit Hilfe irgendeines Indikators zu kontrollieren. Und die Gewöhnung, die in ungünstigem seekrankem Zustand in wenigen Tagen aufzutreten pflegt, wird unter günstigen Umständen wahrscheinlich in bedeutend kürzerer Zeit erworben werden können. Obgleich keinesweges blind für die technischen Schwierigkeiten die zur Errichtung einer zweckmäßigen Übungsschule überwunden werden müssen, halte ich andererseits einen übertriebenen Optimismus nicht für erforderlich um ein solches Unternehmen als ausführbar und aussichtsvoll zu erachten. Obgleich die Seekrankheit praktisch nicht große Bedeutung hat, wegen der, mit wenigen Ausnahmen, so schnell eintretenden Gewöhnung und der geringen Gefahren, so wäre es doch für diejenigen, die sich auf den schon seit längerer Zeit geplanten und wahrscheinlich in absehbarer Zeit zustande kommenden Seesanatorien einer Kur unterziehen wollen, von nicht geringem Vorteil, wenn sie sich vorher gegen die Folgen der unangenehmen Schiffsbewegungen immunisiert hätten.

Groningen, Juni 1908.

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(Gynäkologie Nr. 186.)

Beitrag zum Kampf gegen das Puerperalfieber.

Von

Dr. Emil Ekstein,
Teplitz (Böhmen).

Die dritten fünf Jahre geburtshilflicher Praxis[1]).

Die in rastlosem Eifer vor- und aufwärts strebende Wissenschaft rüttelt an allem Alten, an durch Dezennien, ja oft jahrhundertelang bestehenden Institutionen. Nichts bleibt vor diesem Ansturm des wissenschaftlichen Fortschrittes unberührt. Rücksichtslos wird alles ins Wanken gebracht, was nicht mehr auf zeitgemäßen Fundamenten steht, was althergebrachte Gewohnheit ohne logische Berechtigung fortbestehen ließ.

Diese reorganisatorische Strömung macht sich auf allen Gebieten geltend.

Auch auf dem Gebiete der altehrwürdigen Geburtshilfe, die in festgefügten Bahnen durch Jahrzehnte gelehrt und geübt wurde, macht sich diese Strömung geltend. Die Geburtshilfe als Wissenschaft κατ' ἐξοχήν und die Sozialhygiene der Geburtshilfe werden in neue Bahnen gelenkt, die vom streng wissenschaftlichen Standpunkte bislang zwar noch keine vollkommen einwandfreie Ebnung erfahren konnten, denn dazu ist eben noch die Zeit zu kurz.

Erfreuliches und Unerfreuliches wurde bei diesen reorganisatorischen Bestrebungen gezeitigt und es werden noch Jahre vergehen, ehe ein solcher Ruhepunkt gefunden werden wird, wie er eben durch Jahrzehnte einem Dolcefarniente gleich geherrscht hat.

1) „Die ersten und zweiten fünf Jahre geb. Praxis." Verlag von Carl Marhold, Halle 1901 u. 1904.

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In je ruhigere Bahnen die Schwesterwissenschaft der Geburtshilfe, die Gynäkologie gelangt, mit um so größerer Unruhe wird an der modernen Ausgestaltung unserer gesamten Geburtshilfe gearbeitet und fürwahr, es gibt da noch viel und vieles zu tun, um dem modernen fortschrittlichen Geist unserer Zeit gerecht zu werden.

Der geburtshilfliche Praktiker, ob Spezialist oder praktischer Arzt, der bislang mit einer gewissen Gemütsruhe seiner Tätigkeit zu obliegen gewohnt war, hat bei dieser Reorganisation der Geburtshilfe allerdings keinen leichten Stand mehr, wenn er den festen Grund unter sich immer mehr ins Schwanken geraten sieht. Das Gefühl der Sicherheit, das ihn in seiner Tätigkeit bislang begleitete, muß ihn verlassen, wenn er diesen neuen Werdegang in der Geburtshilfe nur so leichthin verfolgt, denn in erschöpfender Weise dies zu tun, ist auch beim besten Willen infolge des Übermaßes der einschlägigen Literatur nicht möglich.

Wenn auch das Gros der fundamentalen Lehren unserer Geburtshilfe bislang nicht ins Wanken geriet, so sind es doch die Indikationen für das geburtshilfliche Eingreifen und das ganze operative Vorgehen, last not least das ganze Verhalten in bezug auf Anti- und Asepsis, die heute mehr denn je in Diskussion stehen. Eine chirurgische Ära scheint bereits Basis in der Geburtshilfe fassen zu sollen und mit Messer, Säge, Dilatatorien u. a. m. werden der Frucht alle Hindernisse von seite der mütterlichen Geburtswege prompt aus dem Wege geräumt. Der lange nicht verstreichen wollende Zervikalkanal wird mit Fingern und Instrumenten gedehnt, Mißverhältnisse des Beckens zum kindlichen Schädel oder umgekehrt werden mittels Symphysen- oder Schambeindurchtrennung behoben, extraperitoneal wird die Frucht durch das untere Uterinsegment durch Cöliotomie oder Kolpotomie zutage befördert u. a. m. Es macht den Eindruck, wie ich an anderer Stelle schon hervorhob, als ob man den goldenen Geduldsfaden des Geburtshelfers ganz wesentlich verkürzen, die ganze Geburt mit der Raschlebigkeit unserer ganzen Zeit konform zu rascherem Ende als bisher führen wollte. —

Dazu kommt noch die moderne Auffassung der Entstehung der Infektion in bezug auf das Puerperalfieber und der dementsprechend umgestaltete Vorgang bei der Händedesinfektion, der geradezu Bruch mit allem Althergebrachten bedeutet. Wasser- und Seifenwaschung werden als überflüssig für eine Händedesinfektion, unser bislang souveränes Desinfektionsmittel Sublimat nach genannten Waschungen als wirkungslos bezeichnet, u. a. m. Und gerade diese letztere Umwälzung trifft den geburtshilflichen Praktiker ganz besonders hart und bringt ihn geradezu mit seinem Gewissen in Konflikt. Wo die Grenze zwischen

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Recht und Unrecht suchen, wo dieselbe finden, wenn noch dazu die Unbilden der Praxis mit Macht hereinstürzen?.

Das Schicksal ist ein teurer Lehrmeister für jeden Menschen. Für den ärztlichen Praktiker, hier den geburtshilflichen Praktiker, der längere Zeit bereits in Praxis steht, ist die Erfahrung zwar auch ein teurer Lehrmeister, aber trotz alledem ein nicht immer ganz zuverlässiger.

Im allgemeinen ist ja jedes Schematisieren vom wissenschaftlichen Standpunkte aus zu verdammen, speziell in der Geburtshilfe, sowie überhaupt in der Medizin ist dasselbe ganz undenkbar. Post hoc, ergo propter hoc besteht hier keineswegs zu Recht, und wenn es in noch so vielen Fällen den Anschein hat, als ob dem doch so wäre, darf post hoc, ergo propter hoc doch nicht zum Dogma werden.

Wenn ich in einer Reihe von Publikationen immer mit behauptet habe, daß die Geburtshilfe der Kliniken lehrt, wie dieselbe sein soll, und die geburtshilfliche Praxis lehrt, wie dieselbe ist, so muß im Lichte der neueren Forschung diese dogmaartige Anschauung zum Falle kommen, denn die geburtshilfliche Poliklinik hat einen ganz gewaltigen Beweis für die Hinfälligkeit dieser Behauptung erbracht.

Hier bin ich auf dem Punkte angelangt, wo über die angestrebten Wandlungen, die die Sozialhygiene der Geburtshilfe erfahren muß, gesprochen werden soll. Bevor ich mich darüber verbreite, möchte ich einer Arbeit Otto v. Herffs „Im Kampfe gegen das Kindbettfieber", ein Mahnwort an Ärzte[1]), eingehend gedenken und dieselbe in kurzen Zügen darlegen, was in erster Linie im Interesse des ärztlichen Praktikers, dem mit Spezialliteratur sich zu befassen keine Zeit übrigbleibt, geschieht.

Otto v. Herffs Arbeit bedeutet meiner an anderer[2]) Stelle aufgestellten Meinung nach den Beginn der Tätigkeit der Klinik auf dem Gebiete der Sozialhygiene der Hausgeburten und es wäre in erstem und einzigem Interesse der endlichen Bekämpfung des Puerperalfiebers in der geburtshilflichen Praxis wünschenswert, wenn alle geburtshilflichen Kliniker und Lehrer auf der Bahn, die Otto v. Herff betreten, demselben folgen würden. Dann würden auch alle Praktiker mit ganzer Kraft mit in die Schranken treten zum Kampf gegen das Puerperalfieber, die Vorschläge einzelner würden rascher und fester Wurzel fassen und nicht wie bisher einfach literarischen Wert besitzen.

Pasteur, Koch, Lister haben durch ihre Arbeiten die Bekämpfung des Kindbettfiebers in Bahnen gelenkt, auf denen geburtshilfliche

1) Sammlung klinischer Vorträge Nr. 487. Ambrosius Barth, Leipzig.
2) „Klinsiche und Hausgeburten", Gynäk. Rundschau 1908.

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Kliniken und Anstalten zu einem vollständigen Siege über das Kindbettfieber gelangt sind, wo die Zahl der Kindbettfieberfälle in der geburtshilflichen Praxis auf etwa ein Drittel (?) der früheren vermindert wurde. Weiter ist man aber in der geburtshilflichen Praxis nicht gekommen, es ist ein Stillstand eingetreten und Opfer über Opfer fallen dieser Krankheit alljährlich anheim, eine beschämende Tatsache für die Ärztewelt. Der Schlendrian bei den Vorbeugungsmaßregeln und die stoische Untätigkeit der Beteiligten trugen die Schuld an diesen Mißständen. In den Händen des Arztes liegt die Zukunft der Vorbeugung des Kindbettfiebers und es muß mit der Anschauung gebrochen werden, daß es für antiquiert gilt, sich mit dieser für Staat und Familie gleich wichtigen Frage zu beschäftigen. Die Anschauung über die Entstehung des Kindbettfiebers durch ex- und enanthrope Keime ist vollkommen gefestigt, die Selbstinfektion eine nicht zu leugnende Tatsache. Gegen beide Infektionswege muß gekämpft werden, gegen die exanthropen Keime durch das Streben nach Keimfreiheit aller mit der Gebärenden in Berührung kommenden Gegenstände. So leicht dies für die durch Hitze sterilisierbaren Gegenstände ist, so schwer ist dies für die Haut und Hände zu erreichen.

Alle Methoden, die die Haut aufweichen, arbeiten einer Keimfreiheit entgegen, darunter auch die Seifenwasser-Waschung.

Die Keimauswanderung wird eingeschränkt und selbst völlig gehemmt, je trockener die Haut ist, je mehr sie einschrumpft.

Alle Methoden, die dieser Forderung nicht gerecht werden, müssen ungünstige Ergebnisse zeitigen, in erster Linie Wasser und Seife, wie alle Maßnahmen, die mit einer Wasserwaschung schließen, denn sie arbeiten der Keimabsperrung entgegen.

Die Alkoholwaschung vereinigt die Prinzipien der mechanischen und chemischen Reinigung in sich.

Heißwasser und Seife allein gibt schlechte Ergebnisse, nur die mechanische Wegscheuerung mittels Sand, Marmorstaub, Kieselgur mit nachträglichem festen Abreiben mittels eines rauhen trockenen Handtuches vermindert die Zahl der Keime, durch das trockene Abreiben werden die losgelösten Spaltpilze gründlich entfernt. Heißwasser und Seife plus Desinfiziens im Wasser ist nicht besser als eine Heißwasserreinigung mit Sand, Saposilikseife plus Abreiben mit einem rauhen Tuch.

Diese Methode muß als unzuverlässig fallen gelassen werden, das mechanische Prinzip des Wegscheuerns der Oberflächenkeime, verbunden mit der Einsperrung der Tiefenkeime wird in Zukunft die Technik der Hautreinigung beherrschen.

Wenn durch die bisherigen Arten der Desinfektion, Heißwasser-

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Seifenwaschung plus Desinfiziens nicht mehr Infektionsfälle zustande kamen, so liegt dies daran, daß glücklicherweise die gewöhnlichen Hautkeime der Tageshand eine geringe Virulenz besitzen und nur selten schaden.

Die Hand eines Arztes, der sich nicht streng von Infektionsstoffen fernhält, ist unleugbar gefährlicher als eine noch so schmutzige Tageshand einer entarteten Hebamme. An ersterer Hand werden öfters gefährliche Keime haften, an letzterer brauchen sie nicht vorhanden zu sein.

Die großen Opfer, die das Kindbettfieber alljährlich fordert, müssen das ärztliche Gewissen schärfen, sie müssen dazu führen, die Methoden der Heißwasser-Seifen-Reinigung plus Desinfiziens, gelöst in Wasser, vollständig zu verwerfen. Asepsis, gewährleistet durch mechanische Maßnahmen, ist das Ziel, das erstrebt werden muß. Das mechanische Prinzip der Entfernung der Oberflächenkeime neben einer Einsperrung der Tiefenkeime durch Einschrumpfung und Entwässerung der Haut kann nur der Heißwasser-Seifenwaschung im Sinne Ahlfelds folgen, noch besser ist es, diese vollständig fallen zu lassen, weil sie dem Prinzipe der Eintrocknung der Haut entgegenarbeitet.

Die Desinfektionsmethode wird denkbar einfach und abgekürzt, sie wird zu einer Schnelldesinfektion ohne irgend an Zweckmäßigkeit einzubüßen. Ohne Alkohol keine irgendwie genügende Keimarmut oder Keimfreiheit. Die Alkoholwaschung muß daher bei jeder Desinfektion an den Schluß gesetzt werden. Nach Reinigung der Nägel wird die Tageshand (nach Schumburg) unter Verzicht auf Wasser und Seife mittels Watte, Gaze oder Flanell — keine Handbürsten! — mit einer Lösung von zwei Teilen Alkohol und einem Teil Äther bei einem Zusatz von $^1/_2$ proz. Salpetersäure durch 4—5 Minuten abgerieben. Diese Art der Desinfektion besitzt auffallend auch eine Dauerwirkung, was bei länger währender Operation in Betracht kommt. v. Herff benützt statt des Ätherzusatzes Azeton mit bestem Erfolg.

Die Erkenntnis früherer Jahre, daß eine Keimfreiheit der Hand durch die damals üblichen Desinfektionsmethoden in einer praktisch zu verwertenden Zeit nicht zu erzielen ist, führte zur Entdeckung und Einführung der Gummihandschuhe, sowie der Handschuhe in den verschiedensten Kombinationen von Gummi und Trikot, welche eben durch verschiedene Methoden keimfrei zu machen waren. So wurden Handschuhe als bestes Prophylaktikum gegen Kindbettfieber betrachtet, selbstredend insolange dieselben undurchlässig resp. undurchlöchert blieben.

Mit behandschuhten Händen zu untersuchen und zu operieren war und ist nicht jedermanns Sache; dies der eine Nachteil, die leichte

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Zerreißlichkeit der Handschuhe der zweite dauernde Nachteil, die eben dem Gebrauch der Handschuhe anhaften. Letzteren Nachteiles wegen blieb auch beim Handschuhgebrauch die strengste Desinfektion der Hände Conditio sine qua non.

Nachdem die Ergebnisse der Kliniken durch den Gebrauch der Handschuhe gebessert worden und die Alkoholmethode inzwischen zu Ansehen gelangte, so bedeutet der Gebrauch der Handschuhe eben nur einen Schutz der eigenen Hand gegen Beschmutzung mit infektiösem Material und Schutz gegen eigene Erkrankung (Syphilis) und bietet die Möglichkeit der eigenen Tätigkeit, wenn die Haut der eigenen Hände eben nicht ganz frei von Verletzungen, Rillen und Schrunden ist, wo sich eben nicht selten angriffsfähige Formen von Spaltpilzen mit Vorliebe aufzuhalten pflegen.

Der wahre Wert der Handschuhe liegt demnach in der sogenannten Noninfektion. Diese beabsichtigte Keimabsperrung der Hände durch Handschuhe wird aber auch durch Einreiben der Haut mit Öl, Fett, Vaselin, Jodipin, Jodbenzinlösung, Jodazeton, Gaudanin oder Chirosoter bewirkt, so daß die Handschuhe in der Tat entbehrlich scheinen.

Die Scham der Frau keimfrei zu machen ist schwer angänglich wegen der Zartheit der Haut. Die bisherige gründliche Desinfektion mit Sublimat illusorisch. Alkohol-Azeton-Reinigung, die nur in Narkose durchzuführen ist, gibt in bezug auf Keimverarmung gute Resultate, selbstredend nach erfolgtem Rasieren der Scham.

Die Forderung, die Scheide von Eigenkeimen zu befreien, läßt sich schwer erzielen, hingegen läßt sich eine Abschwemmung der lose sitzenden Spaltpilze eher erreichen.

Scheidenspülungen sind von Vorteil, die Berechtigung, ja der Nutzen vorbeugender Scheiden- und Gebärmutterspülungen scheinen durch klinische Erfahrung in jeder Beziehung sichergestellt zu sein.

Die Schwere des Geburtstraumas spielt eine wesentliche Rolle in der Entstehung des Kindbettfiebers. Durch das Geburtstrauma können ex- und enanthrope Keime der Wöchnerin gefährlich werden. Je geringer das Geburtstrauma, desto ungünstiger die Bedingung für die Infektion. Je ausgedehnter ein Operationstrauma ist, je näher dieses an der Plazentarinsertionsstelle liegt, desto größer die Gefahr für die Wöchnerin. Der ungünstige Einfluß des Traumas läßt sich nur durch tadellose Asepsis und Antisepsis paralysieren.

Übersehen und Geringschätzen des Traumas ist neben ungenügender Desinfektion eine der Hauptursachen, daß das Kindbettfieber nicht abnehmen will.

Also keine Eingriffe, keine Operation ohne strengste Anzeigen, d. h. also nur bei eintretender Gefahr für Mutter und Kind, kein über-

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flüssiges Hineinmischen des Arztes, mehr Konservatismus in der Geburtshilfe! Abwartende Geburtsleitung, gestützt auf vorbeugende Eingriffe, Geduld und weitgehendstes Vertrauen in die Naturkräfte ist in ihren Endresultaten nicht nur völlig gleich, sondern muß auf die Dauer besser sein als die abwartende Geburtsleistung, gestützt auf Kaiserschnitt und Beckenspaltung.

Abgesehen von der Einführung einer zuverlässigen Desinfektion und der Verminderung des Geburtstraumas muß die Nachgeburtsperiode sorgfältiger geleitet, die Ausstoßung der Nachgeburt geduldig den Naturkräften überlassen werden, denn dadurch nur wird die manuelle Plazentarlösung um so weniger oft vorgenommen zu werden brauchen. Bei Wehenschwäche der Gebärenden soll nur dann operiert werden, wenn Gefahren für Mutter und Kind vorhanden sind. Durch Darreichung von Sekale muß dann für eine rasche Kontraktion des Uterus zwecks Vermeidung von Nachblutung gesorgt werden.

Verhaltene Plazentarteile sind unter allen Umständen zu entfernen, Eihautreste, ja selbst den retinierten ganzen Eihautsack zu entfernen ist unnötig und gefährlich.

Im Wochenbett ist durch Ergotin und heiße Scheidenspülungen für eine rasche Rückbildung des Uterus Sorge zu tragen.

Während des Wochenbettes ist, wenn irgend möglich, keinerlei operativer Eingriff, insbesondere kein Eingriff innerhalb der Gebärmutter vorzunehmen.

Der Kampf gegen eine mißverstandene chirurgische Ära der Geburtshilfe muß aufgenommen werden, denn die Vieltuerei mancher Ärzte stellt nur allzuhäufig die Hauptursache des Stillstandes, ja der Zunahme des Kindbettfiebers dar.

Die Regierung möge aber auch ihrerseits sich ihrer Pflichten auf diesem Gebiete erinnern und mit ihren Machtmitteln die Ausmerzung bedenklicher Auswüchse auf dem Gebiete der Vorbeugung des Kindbettfiebers, insbesondere bei den Desinfektionsvorschriften für Hebammen erleichtern.

Dieser kurze Bericht der geradezu fundamentalen Leitsätze aus der Arbeit v. Herffs wird so manchem Geburtshelfer zu denken geben und ich gestehe, daß ich bei Veröffentlichung meiner „zweiten fünf Jahre geburtshilflicher Praxis“ schon eine gewisse Vorahnung hatte, daß große Dinge in unserer Geburtshilfe sich vorbereiten.

Meine Ahnungen sind, wie die Arbeit v. Herffs zur Genüge dokumentiert, vollkommen berechtigt gewesen.

Daß wir Ärzte mit unserer Anti- resp. Asepsis, wie sie bisher üblich war, durch die Erfolge der neueren Forschung, trotz der oft langjährigen Erfahrungen in gutem Sinne, brechen werden müssen,

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dafür ist die Autorität v. Herffs entscheidend und nach unseren Erfahrungen in der geburtshilflichen Praxis auf dem flachen Lande, unter den denkbar schlechtesten sozialen Verhältnissen unserer Gebärenden, ist es nach den Ausführungen v. Herffs gewiß der Noninfektion der Beteiligten zuzuschreiben, daß die Erfolge noch so sind, wie sie eben sind, und nicht direkt unserer Anti- resp. Asepsis, in deren Betätigung sich unwillkürlich so manche Lücke einstellen mußte, wodurch dieselbe eben nicht mehr einwandfrei wurde. Der Vergleich der geburtshilflichen Poliklinik mit der geburtshilflichen Praxis κατ' ἐξοχήν und die in derselben für die neue Desinfektionsmethode in Anspruch genommenen guten Resultate, dürfte jedoch nicht so ganz richtig sein, wie v. Herff dies gemeiniglich anzunehmen beliebt.

Städte, die heute Universitätspolikliniken besitzen, weisen in bezug auf die Armengeburtshilfe denn doch weit salubrere soziale Verhältnisse auf, als dies in unseren Arbeiterwohnungen auf dem flachen Lande der Fall ist. Der Faustschlag, den jede hygienische, ja oft geradezu nur menschliche Anforderung an eine Wohnung, geschweige denn Einrichtung hier erhält, ist vielmals zu wuchtig, als daß man da auch nur im geringsten daran denken könnte, mit Anti- oder Asepsis einen Erfolg erreicht zu haben. Der Erfolg ist da und ganze Reihen von Erfolgen, die man in den Jahren unter solchen ganz tristen Verhältnissen erzielt, bestärken in der Ansicht, daß es eben die Noninfektion ist, die hier den guten Erfolg zeitigt. Ich führe diese Tatsache hier nur wiederum an, um der Behauptung v. Herffs in bezug auf Noninfektion vom Standpunkte des in schwierigsten Verhältnissen oft arbeitenden Praktikers die ihr gebührende Würdigung zu verleihen. Damit möchte ich aber auch das herbe Urteil v. Herffs über die Ärzte in bezug auf Mangelhaftigkeit im Vorgehen in der geburtshilflichen Praxis im allgemeinen in gewisser Beziehung etwas abschwächen.

Wie allgemein bekannt werden ca. 90—92% aller Geburten durch Hebammen geleitet und besorgt, ohne daß überhaupt ein Arzt zugezogen wird, und selbst zugegeben, daß dieses Prozentverhältnis noch zu hoch gegriffen ist, so muß mir jeder geburtshilfliche Praktiker zugestehen, daß gerade in der Wochenstube die Intelligenz des Arztes am wenigsten zur Wirkung gelangen kann. Wie schon De Lee ganz richtig bemerkt, steht man in Laienkreisen auf dem Standpunkt, daß die Niederkunft als ein natürlicher Vorgang keiner speziellen Für- und Vorsorge bedarf und nur sehr spärlich hat sich bislang dank der minderwertigen und unwürdigen Propaganda des Gros unserer unintelligenten Hebammen das Gegenteil dieser Anschauung noch bei weitem nicht den Eingang verschafft, wie dies v. Herff verlangt und wie er es eben von uns Ärzten verlangt. Ich stimme v. Herff vollkommen

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bei, wenn er die Teilnahmslosigkeit der Ärzte gegenüber einer Sanierung unserer ganzen Geburts- und Wochenbettshygiene entsprechend ins Treffen führt, aber von einer Schuld der Ärzte an dem Nichtaufhören des Kindbettfiebers kann füglich nicht in dem Maße gesprochen werden, wie dies v. Herff tut. Die Erfahrung lehrt, daß bei der in erster Linie herrschenden Unintelligenz des Gros der Hebammen und bei der im Laienpublikum noch so weit verbreiteten Indolenz ein geradezu nimmerermüdender Enthusiasmus dazu gehört, immer wieder für Reformen sich fruchtlos einzusetzen, wobei noch dazu die Staatsbehörden diesen Bestrebungen gegenüber sich konsequent passiv verhalten. Nur die harte Notwendigkeit, seinem Gewissen zu folgen, läßt gegenüber der bekannten Morbidität und Mortalität die Feder nicht zur Ruhe kommen. Der geburtshilfliche Praktiker, der aber noch allgemeine Praxis zu treiben gezwungen ist, kann, weder der Not gehorchend, noch dem eigenen Triebe, unmöglich reformierend wirken, denn dazu hat er weder Zeit noch Gelegenheit, seine volle Kraft dafür einzusetzen. Der Spezialist, der ja heute in ganz wesentlicher Stärke vertreten ist, kann und muß sich mit all diesen Fragen befassen und nur die Fruchtlosigkeit dieser Bemühungen in bezug auf praktische Erfolge hat die Reihe der vorkämpfenden Spezialisten bislang nicht allzugroß werden lassen. Dazu kommt noch, daß es bis vor nicht zu langer Zeit an einer Organisation zur Bekämpfung des Kindbettfiebers gefehlt hat, die in der Vereinigung zur Förderung deutschen Hebammenwesens eben erst ihren Anfang genommen zu haben scheint.

Der Vorwurf v. Herffs muß in dieser Beziehung die Ärzte hart treffen und dürfte in Rücksicht auf die unumgängliche Mitarbeiterschaft der Ärzte gewiß eine Milderung erfahren müssen, was schon in Anbetracht der geschilderten Sachlage nur gerecht erscheint.

Soll im Kampfe gegen das Kindbettfieber die Vorbeugung desselben in Zukunft in den Händen der Ärzte liegen, worin als Grundprinzip das Fernhalten der Gefahren der Fremdkeime und Eigenkeime gelegen ist, dann könnte dies, wie die Dinge heute liegen, nur in der Weise erreicht werden, wenn die Forderung, „die Geburt gehört dem Arzte", realisiert werden könnte. Insolange aber als geburtshilfliches Heilpersonal Hebammen fungieren, deren Ausbildung Unintelligenz nicht ausschließt, insolange wird auch jede Tätigkeit des Arztes in genannter Weise von vornherein als fruchtlos bezeichnet werden müssen.

Anders wird es und muß es sich selbstredend gestalten in bezug auf das Fernhalten der Eigenkeime was die eigene Person des Arztes anbelangt. Wenn die Ärzte im allgemeinen in der Aufnahme neuer Methoden etwas skeptisch geworden sind, so liegt dies zumeist in der

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vorschnellen Enunziation derselben durch ihre Erfinder, sei es die Klinik oder der Arzt selbst. Die Erfahrung lehrt uns diesbezüglich zur Genüge, daß dieser Skeptizismus in der Tat seine Berechtigung besitzt. Die neuen Lehren, wie sie v. Herff in einer ganz apodiktischen Weise enunziert, deren schlagende Erfolge die Klinik aufzuweisen hat, verdienen in allererster Linie die höchste Anerkennung des Praktikers, wird doch damit klinisch und bakteriologisch erprobt eine Methode der Händedesinfektion empfohlen, die in einer praktisch zu verwertenden Zeit eine Keimfreiheit oder hochgradige Keimverarmung mit Sicherheit zu erreichen ermöglicht, ja geradezu garantiert.

Hatte der Praktiker bislang keine Veranlassung, in die ihm an der Klinik gelehrten Methode der Händedesinfektion, Heißwasser-Seifenwaschung plus Desinfektionslösung, gewisse Zweifel bezüglich ihrer Wirksamkeit zu setzen und wurde ihm diese ganze Prozedur zur starren Methode, so kann und darf derselbe nach den v. Herff gemachten Darlegungen keinen Augenblick mehr schwanken, sich dieser neuen Richtung zu verschließen.

Von dem Gedanken einer Abkürzung der Heißwasser-Alkohol-Sublimatdesinfektion ausgehend habe ich selbst den Versuch gemacht, in meiner Praxis in dem bekannten Kölner Wasser Sublimat, Salizyl- und Borsäure wirksam zur Lösung zu bringen und nach erfolgter Heißwasser-Seifenwaschung Hände und Arme damit abzureiben[1]). Zersetzungsvorgänge, die sich bei längerem Stehen dieser Lösung einstellten, machten einen weiteren Gebrauch aber unmöglich und es erscheint nicht ausgeschlossen, daß bei längerem und intensivem Gebrauch Intoxikationserscheinungen auftreten würden. Jedenfalls aber hatte ich bei diesem Desinfektionsversuche die Keimabsperrung bezweckt, der Wegscheuerung der Keime aber nicht Rechnung getragen. Und gerade die Wegscheuerung der Keime erscheint mir das Hauptmoment; dieselbe ist aber bei der in der Praxis üblichen Heißwasser-Seifenwaschung in einem gewöhnlichen Waschbecken von vornherein ausgeschlossen. Durch die Art der Waschung in den gebräuchlichen Waschbecken mit oder ohne Bürste werden doch die Hände immer wieder mit den Keimen in Kontakt gebracht, geradezu imprägniert. Anders verhält es sich mit dieser Waschung beispielsweise bei Laparatomien. Hier werden die Hände und Arme mit Schmierseife und Marmorsand vorerst durch einige Minuten gründlich abgerieben, massiert. Hernach werden Hände und Arme in strömendem Wasser von diesem Seifen-Marmorsandgemisch befreit, in Sublimat und her-

1) Zentralbl. f. Gyn. 1907.

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nach erst in Alkohol abgerieben. So habe ich die Desinfektion bis jetzt geübt und in der letzten Serie von 80 Laparatomien keine Infektion, keinen Todesfall erlebt. Trotz alledem werde ich nach Abscheuern der Haut der Hände und Arme die Wasserabspülung ausschalten, desgleichen die Sublimatdesinfektion und mich nur des Alkohols bedienen. Jedenfalls wird aber auch in der geburtshilflichen Praxis die Heißwasser-Seifenwaschung im stehenden Wasser von nun an fallen, und die von v. Herff angegebene Methode benützt werden. Wie ich später darlegen werde, habe ich in allen meinen in den dritten 5 Jahren von mir durchgeführten Fällen von Abortus und Geburten keinen einzigen Fall von Infektion zu verzeichnen. Dazu muß ich aber bemerken, daß ich auf das strengste den Standpunkt der Auto-Noninfektion stets zu wahren suche. Ich beschränke mich in meiner Praxis striktest auf mein Gebiet, Geburtshilfe und Gynäkologie, und komme dabei nur höchst selten in die Lage, eitrige oder infektiöse Erkrankungen zu behandeln, die dann aber nur gynäkologischer Art sind. Peinlichste Körperpflege und ständige peinliche Reinhaltung der Hände sind selbstverständlich. Unter diesen Umständen habe ich immer das sichere Gefühl, auch ohne jedwede vorherige Desinfektion geburtshilflich gynäkologisch tätig sein zu können. Empfiehlt Foges in seiner bekannten Arbeit den Septikus, so möchte ich dem entgegen den Aseptikus empfehlen. Der praktische Arzt, der allgemeine Praxis treibt, kann bei seinem Dienste im allgemeinen und speziell als Kassenarzt in erster Linie, wo er oft 40—50 Kranken zu ordinieren und oft die gleiche Zahl Visiten bei den verschiedensten Kranken zu machen hat, den Standpunkt der Noninfektion kaum wahren, also nicht aseptisch bleiben: das ist gewiß ein Ding der Unmöglichkeit. Aseptisch kann der Spezialist, speziell der Gynäkologe und Geburtshelfer am ehesten noch sein, weniger schon der vielbeschäftigte Chirurge.

Ist es auch durch Tatsachen erwiesen, daß ohne diese Schaffung von Septikus und Aseptikus die praktische Medizin ganz gute Resultate erzielte, so handelt es sich doch stets um die Prophylaxe, die bekanntermaßen die beste Medizin ist. Überblicke ich die letzten 30 Jahre, so kenne ich vereinzelte Fälle, wo Wöchnerinnen an Scharlach beispielsweise zugrunde gegangen sind, wo der behandelnde Arzt eben Scharlachfälle in Behandlung hatte, desgleichen sah ich eine Laparatomierte an fudroyanter Sepsis zugrunde gehen, wo ein Assistent bei der Operation an einer akuten Koryza litt, u. a. m.

Wenn ich heute so vielmals auf ärztlichen Tafeln lese: Frauen- und Kinderarzt, so schwebt mir stets der obenzitierte Fall vor Augen. Ich halte diese Praxis einfach für unvereinbar und befinde mich mit meiner Ansicht wohl in bester Gesellschaft. Die geburtshilfliche

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Praxis erfordert ganz apodiktisch den Status auto-noninfectionis von seite des Arztes und es kann wohl niemand sich der Ansicht mehr verschließen, daß — cessante causa cessat effectus — damit die fundamentalste Vorbeugung gegen das Kindbettfieber erreicht wird.

Hätte diese Erkenntnis vor 60 Jahren zu Beginn der Ära Semmelweis bereits Platz gegriffen, so wäre dem furchtbaren „Müttersterben" auch ohne Chlorkalkwaschung der Hände wirksam Einhalt geboten worden.

Wie peinlich wird heute der Leser des Lehrbuches von Scanzoni aus dem Jahre 1855 berührt, wenn er die Ansichten dieses so an Erfahrung reichen Geburtshelfers über das Kindbettfieber liest, wenn er über geburtshilfliche Operationen unterrichtet wird, und mit keinem Ton dabei einer Reinigung der Hände gedacht wird; wie seltsam berührt es den Leser, wenn er das Lehrbuch des großen Schroeder vom Jahre 1886 durchblättert und diesem wichtigsten Faktor in der Verhütung der Infektion noch nicht die ihm zukommende Würdigung erfahren sieht.

Es muß als einen der größten Triumphe unserer Wissenschaft bezeichnet werden, in einer so verhältnismäßig kurzen Spanne Zeit nicht allein zu einer richtigen Erkenntnis der Entstehung des Kindbettfiebers gelangt zu sein, sondern auch, wie dies unsere Kliniken dokumentieren, diese wirksamen Vorbeugungsmaßregeln geschaffen zu haben.

Gegenüber dieser Tatsache muß es in der Tat beschämend für uns Ärzte wirken, in der geburtshilflichen Praxis ein Fortbestehen des Kindbettfiebers konstatiert zu sehen. Das bedeutet einen herben Wermutstropfen in dem Freudenbecher unseres wissenschaftlichen Könnens.

Es kann kein Zweifel bestehen, daß nach wie vor der praktische Arzt Geburtshilfe treiben muß und daß nach wie vor unter solchen Umständen der Status auto-noninfectionis von demselben niemals mit Sicherheit erreicht werden wird.

Zur teilweisen Milderung dieses Mißverhältnisses wird es sich daher empfehlen, daß gerade der praktische Arzt, der in seiner ausgedehnten mannigfachen Berufstätigkeit niemals sicher ist, plötzlich zu einer Entbindung gerufen zu werden, dem Gebrauche der Gummihandschuhe wenigstens in vermehrter Weise Rechnung trägt, um sich, resp. seine Hände und Arme in erster Linie vor Beschmutzung mit infektiösem Material zu schützen, und der Alkoholbewegung bei der Händedesinfektion den größtmöglichsten Vorschub leistet.

Galt es bislang als feststehende Norm, die Gebärende und Wöchnerin als ein „noli me tangere" zu betrachten, so geriet auch die Basis dieser Norm teilweise ins Schwanken.

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Die eminenten Erfolge der Chirurgie auf Basis der Anti- und Asepsis begannen bei den gleichen Erfolgen in der Gynäkologie auch für die Geburtshilfe Schule zu machen und binnen kurzem etablierte sich eine chirurgische Ära vorerst in der klinischen Geburtshilfe, deren Lorbeeren aber auch die geburtshilfliche Praxis nicht schlafen zu lassen scheinen.

Die Kluft zwischen den Verhältnissen der Klinik und denen der Praxis ist aber in jeder Beziehung eine derartig große, daß an eine Überbrückung nur sehr schwer zu denken ist und allfällige Versuche zu großen Enttäuschungen geführt haben.

Der Ruf nach Konservatismus in der Geburtshilfe dürfte daher seine vollständige Berechtigung besitzen, um noch rechtzeitig Mißgeschicke hintanzuhalten, wie sie in der Gynäkologie durch die einstige Rabies operandi heraufbeschworen wurden. Sicher ist es, daß der moderne Geburtshelfer chirurgische Schulung genossen haben muß, um auch lege artis in der konservativen Geburtshilfe auf der Höhe der Leistungsfähigkeit zu stehen.

Darüber detailliert zu sprechen, erachte ich für überflüssig. Auf das nachdrücklichste muß aber vor dem voreiligen, ungeduldigen Verschieben unserer gesunden geburtshilflichen Indikationen gewarnt werden, die sich auf eine Abkürzung der ersten Geburtsperiode beziehen, wofür die Gründe ja hinlänglich allgemein bekannt sind.

Was die Indikationen der zweiten Geburtsperiode, der Expulsionsperiode anbelangt, so stehen dieselben auf solch wissenschaftlich einwandfreier Basis, daß daran in keiner Weise gerüttelt zu werden braucht.

„Du sollst dein Kind in Schmerzen gebären", ist ja für die Gebärenden und deren Umgebung ein harter Schicksalsspruch und fürwahr, die Eröffnungsperiode bewahrheitet denselben mehr denn eigentlich nötig.

Es entspringt deshalb einem natürlichen Bedürfnis einer jeden Gebärenden und dem Wunsch deren Umgebung, diese schmerzensreiche Zeit, wenn schon nicht in der Eröffnungsperiode, so doch wenigstens in der Expulsionsperiode abzukürzen.

Gehört es zu den segensreichsten Erfolgen unserer Wissenschaft, Schmerzen zu bannen, so gehört es zu den schönsten Erfolgen unserer Geburtshilfe, den Arzt in die Lage zu versetzen, die Schmerzensperiode der Geburt ebenfalls abkürzen zu können und zwar ist es die Expulsionsperiode.

Voraussetzung strengster Natur bleibt es natürlich immer, daß alle Indikationen erfüllt sind, dies tun zu können, und alle Möglichkeiten vorhanden sind, dies ohne Schädigung der Gebärenden zu leisten.

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Man vergegenwärtige sich die Expulsionsperiode einer Primipara, wie nach Blasensprung, Verstrichensein des Muttermundes und bei fixiertem Kopf des Kindes die Wehentätigkeit und die Anstrengung des Mitpressens während derselben den ganzen Organismus der Gebärenden in gewaltige Aufregung und Aufruhr versetzt. Und da sollte es als ärztliche Polypragmasie gedeutet werden, bei Vorhandensein aller geforderten Vorbedingungen durch eine vorsichtige Forzepsapplikation diesen Qualen ein Ende zu bereiten? Eine solche Polypragmasie muß als Akt der Menschlichkeit bezeichnet werden, der unter wissenschaftlich erprobten Schutzmaßregeln ausgeführt sogar als segensreich bezeichnet werden kann und diese Abkürzung unter genannten Bedingungen als geradezu indiziert bezeichnet werden. Hier unter allen Umständen auch bei nicht vorhandener Gefahr für Mutter und Kind den Naturkräften zu vertrauen, wäre meines Erachtens ein falsch bewerteter Konservativismus.

Anders verhält es sich selbstredend bei der Behandlung der Nachgeburtsperiode, wo es für eine voreilige und unzeitige Nachgeburtslösung ohne irgendwelche dringende Indikation keine Entschuldigung gibt.

Daß die chirurgische Betätigung in der Geburtshilfe, wie sie an so vielen Kliniken geübt wird, für die Praxis nicht vorbildlich sein kann und darf, bedarf trotz der wenigen gegenteiligen Behauptungen gewiß keiner näheren Erörterung.

Zur Entfernung der Eigenkeime der Scheide werden neuerdings bei Geburten Scheidenspülungen mit verschiedenen Desinfektionslösungen empfohlen, desgleichen zur besseren Involution des Uterus im Wochenbette.

Ich selbst habe die Ära der indikationslosen Scheiden- und Uterusspülungen mitgemacht und ebenfalls die Ära, wo mit der Scheidenspülung bei Geburten und im Wochenbett gründlich gebrochen wurde. Präventive Scheidenspülungen bei infektiösen Prozessen der Scheide hauptsächlich bei Gonorrhöe der Schwangeren und Gebärenden, Uterusspülungen aus demselben Grunde bei Wöchnerinnen blieben ja stets im Gebrauch. Heute wird der Scheidenspülung als Vorbeugungsmittel gegen das Kindbettfieber wieder das Wort gesprochen analog der Entfernung der Blutkoagula aus der Scheide post partum im Sinne Zweifels.

So einwandfrei die diesbezüglichen Erfolge der Klinik anzuerkennen sind, so zweifelhaft muß dies Verfahren in der geburtshilflichen Praxis erscheinen, nachdem doch daselbst in einer großen Zahl der Fälle gerade durch die Scheidenspülung Fremdkeime in die Scheide eingebracht werden können, was insbesonders für die Praxis der

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Hebammen gilt, wo ein Mutterrohr und ein Irrigator für die Besorgung der Gebärenden und Wöchnerinnen verwendet wird. Auch ist die Herstellung der Desinfektionslösung nicht in allen Häusern einwandfrei zu erzielen, so daß vom Standpunkte der breiten geburtshilflichen Praxis dieses Verfahren hier wohl nicht für eine Verallgemeinerung zu empfehlen sein dürfte. Ob durch diese einfachen Scheidenspülungen in der Tat alle Eigenkeime aus der faltenreichen Scheide entfernt werden können, dafür ist der Beweis wohl noch nicht erbracht und scheint auch nicht so einfach zu erbringen zu sein. Eigenkeime werden vor gynäkologischen Operationen durch Ausreiben der Vagina wohl leichter entfernt werden können, nachdem es durch das Vorziehen des Uterus mittels Kugelzange möglich ist, die Scheide zu entfalten und alle Falten zum Verschwinden zu bringen, kurz die Scheide unter Kontrolle der Augen gründlich klar zu machen. All dies ist bei Schwangeren und Gebärenden sowie Wöchnerinnen nur aus ganz zwingenden Gründen möglich.

Eine große Beobachtungsreihe in der Praxis, die aber in einschlägiger Weise kontrolliert wird, ist nicht so leicht zu erbringen, weshalb nach den alten Erfahrungen die obligatorische präventive Scheidenspülung der allgemeinen Praxis für absehbare Zeit vorenthalten bleiben dürfte.

Seit Beginn meiner geburtshilflichen Praxis stehe ich auf dem Standpunkte der obligatorischen Verabreichung von Sekalepräparaten post abortum et partum, aber stets erst nach vollständiger Entleerung des Uterus. Über die Zweckmäßigkeit dieser Medikation zu sprechen, bedarf es keiner Worte, dieselbe liegt vollständig klar zutage. Leider vermißt man in der geburtshilflichen Praxis noch sehr häufig diese die Uterusinvolution so sehr begünstigende Therapie, die gleichzeitig eine prominente Vorbeugung gegen Infektion bildet. In dieser Beziehung wäre eine Verallgemeinerung der Anwendung der Sekalepräparate von seite der Geburtshilfe treibenden Ärzte und Hebammen sehr geboten, selbstredend in streng individualisierender Weise; in erster Reihe nach geburtshilflichen Eingriffen, aber auch nach spontanen Geburten, wenn die Nachwehen eben in nicht genügender Stärke vorhanden sind.

Nach der Darlegung gewisser von v. Herff vom Standpunkte des Praktikers eingehend besprochener Verhältnisse der geburtshilflichen Praxis im allgemeinen müßte es mir in der Folge obliegen, die Mängel unserer Sozialhygiene der Hausgeburten, die eine Minderwertigkeit der Vorbeugung des Kindbettfiebers involvieren, aufzuzählen, Mängel, für deren schädigendes Fortbestehen dem Arzt absolut keine Schuld trifft, die er beim besten Willen eben nicht zu beseitigen vermag,

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wobei seine Hilf- und Machtlosigkeit gegenüber der großen Indolenz des Laienpublikums und dem passiven Verhalten der Staatsbehörden vollständig Entschuldigung finden muß. Ich kann es mir hier füglich versagen, über all diese großen Unterlassungssünden, die konstant begangen werden, zu sprechen, habe ich dies nur allzuoft beinahe durch 6 Jahre, und leider stets ohne entsprechenden Erfolg in einer großen Reihe von Monographien getan. Ich hege zuversichtliche Hoffnung, daß die gesamte geburtshilfliche Neuorganisation, wie sie eben heute immer noch ihrer Lösung harrt, auf rein akademischen Boden gestellt, durch unsere Kliniker und Lehrer der Geburtshilfe in gemeinsamer Arbeit und Einverständnis mit den geburtshilflichen Praktikern und — den Staatsbehörden endlich doch einmal diejenige Würdigung finden wird, um ohne Rücksicht auf materielle Kosten in einwandfreier moderner Weise zur Durchführung zu gelangen.

Speziell in Österreich wurde im Jubiläumsjahre Kaiser Franz Josef I. von Sr. Majestät selbst die Parole für Jubiläumsstiftungen „fürs Kind“ ausgegeben. Millionen werden gesammelt und wahrscheinlich thesauriert werden.

So edel, so ideal und so praktisch notwendig diese ganze Bewegung in der Tat ist, so hat man direkt das Elend des Kindes im Auge gehabt, hat dabei aber ganz der Mutter vergessen, deren Elend mit dem des Kindes leider nur zu oft unzertrennlich verquickt ist. Nicht allein „fürs Kind“, nein, für „Mutter und Kind“ ist zu sorgen und in erster Reihe zu verhüten, daß dem Sterben der Mütter in der höchsten Betätigung ihrer weiblichen Pflicht Einhalt geboten wird, worunter in erster Reihe die Vorbeugung gegen das Kindbettfieber zu verstehen ist.

Die tägliche Erfahrung lehrt, daß eine Entbindung in einer wohlgeleiteten Anstalt unter allen Umständen in bezug auf Infektion sicherer absolviert wird als im Hause. Die tägliche Erfahrung lehrt, daß unsere Bezirkskrankenhäuser nicht nur an einem chronischen Platzmangel leiden, sondern nicht einmal freiwillig, sondern nur der höchsten Not gehorchend, eine Gebärende aufnehmen, geschweige denn eine arme Schwangere, die ihrer schwersten Stunde erst entgegensieht. In echt bureaukratischer Ängstlichkeit wird die Ausgestaltung der Bezirkskrankenhäuser, dem Durchschnitt der Aufnahmen von beispielsweise 5 Jahren entsprechend, hintangehalten, es wird einfach gespart und gespart, um ja nur in bezug auf Ausgaben dem Bezirke keine größere Lasten aufzubürden. Diesen einem Zeitalter der Hygiene und Humanität absolut nicht entsprechenden Zuständen wäre durch diese angesammelten Fonds „fürs Kind“ ein Ende zu bereiten und durch Errichtung von Gebärabteilungen an jedem Bezirksspital für Mutter und Kind in menschenwürdiger Weise zu sorgen. Dazu kommt noch, daß zur Abhilfe und

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endgültigen Beseitigung dieses chronischen Platzmangels mit all den traurigen Konsequenzen jede Stadt von mindestens 10000 Einwohnern zu verhalten wäre, ein Krankenhaus für seine Bewohner zu schaffen, damit das Bezirkskrankenhaus eben für die kleineren Orte des Bezirkes reserviert bliebe. Dies nur nebenbei.

In der Vorbeugung des Kindbettfiebers werden wir wohl nicht eher erfolgreich vorwärts schreiten, insolange nicht durch Gründung eines selbständigen Sanitätsressorts im Ministerium mit gleichzeitiger Errichtung von „sozialen Gesundheitsämtern" die gesundheitliche Volkswohlfahrt der großen Erfolge unserer medizinischen Wissenschaft voll und ganz teilhaftig gemacht werden wird. Einer zu errichtenden Lehrkanzel für Sozialhygiene muß es vorbehalten bleiben, ein wirksames, initiatives Bindeglied zwischen Sanitätsministerium und Gesundheitsämtern zu bilden. Mehr als durch Kriegsschiffe, Luftballons und Kanonen würde so das Volkswohl bewahrt werden. Dann und nur dann wird das von Semmelweis so sehnlich erwartete Zeitalter hereinbrechen, in welchem inn- und außerhalb der Gebärhäuser der ganzen Welt nur Fälle von Selbstinfektion vorkommen.

Zur Besprechung der in den dritten 5 Jahren von mir behandelten geburtshilflichen Fällen übergehend, stehen mir 335 Fälle zur Verfügung. (Siehe nächste Seite.)

Ad I. Die verhältnismäßig geringe Zahl von spontanen Geburten, die hier verzeichnet sind, dokumentiert zur Genüge, wie ungern der Arzt heute noch in der Wochenstube gesehen wird und wie zäh und fest die Frauenwelt an der Hebamme festhält.

Dagegen wäre von vornherein nichts einzuwenden, wenn die Minderwertigkeit des Gros unserer Hebammen nicht mit in Frage käme und gerade in Rücksicht auf das starre Festhalten der Frauen ist die bisherige Minderwertigkeit der Hebammen, das Fehlen der nötigen Intelligenz in diesem Stande nicht genug zu tadeln. Schuld an dieser Unterlassung einer höheren Bewertung des Hebammenstandes und der konsequenten exakten Ausbildung trägt der Staat und in erster Reihe ist der Staat für die Folgen verantwortlich. Es kann gar kein Zweifel bestehen, daß es dem Arzte ganz unmöglich ist, eine Geburt von Anbeginn bis Ende zu leiten, denn dazu fehlt ihm unbedingt die Zeit. Hier liegt die Indikation für den Hebammenstand, für einen Hebammenstand aber, der vermöge seiner Intelligenz und guten Ausbildung den Anforderungen einer lege artis Geburtsleitung gewachsen ist.

Ad II. Die verhältnismäßig große Zahl von Abortusfällen ist ein Zeichen unserer Zeit. Über die Ätiologie der einzelnen Fälle zu sprechen ist unmöglich, der Praktiker gewöhnt es sich ab, bei diesen

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Tabellarische Übersicht.

	Zahl der Fälle	glatter Verlauf	Todesfälle der Mütter	Todesfälle der Kinder
I. Spontane Entbindung.	13	13	—	1 (Lues)
II. Abortus	156	156	—	—
III. Abortus artefic. Nr. VII	14	14	—	10
IV. Forceps.	53	53	—	3
V. Perforation	3	3	—	—
VI. Wendung	12	12	—	10
VII. Bigemini	6	6	—	—
VIII. Retentio placentae p. part.	12	12	—	—
IX. Naht von spontanen Dammrissen . . .	5	5	—	—
X. Atonia uteri	2	2	—	—
XI. Eklampsie	2	2	—	1
XII. Placenta praevia . .	4	4	—	4
XIII. Gravidit. extrauterina	14	13	1	—
XIV. Processus puerperalis	12	12	—	—
XV. Retroversio-flexio uteri gravidi . . .	24	23	1	—
XVI. Sectio caesarea . . .	1	—	1	1
XVII. Apoplexia in graviditate	1	1	—	—
XVIII. Discisio septi vaginae p. part. spontan. .	1	1	—	—
	335	332	3	30

Fällen strenge nachzuforschen; wohin dies führen würde, ist nur zu bekannt. Was die eigentliche Therapie bei Abortus anbelangt, stehe ich nach wie vor auf dem Standpunkte, daß das aktive Verfahren, wie ich dies in meinen Publikationen[1]) detailliert dargestellt habe, das rationellste Verfahren bildet, die Restitutio ad integrum in kürzester Zeit herbeizuführen und dabei den Vorzug der Schmerzlosigkeit besitzt. In bezug auf die Möglichkeit der Infektion ist es bei der Anschauung, die wir über unsere Händedesinfektion besitzen, jedenfalls schwerer möglich mittels Instrumenten zu infizieren als mittels Finger. Was die Uterusperforation anbelangt, so ist dieselbe, wie ich dies in einer meiner letzten Arbeiten[2]) geschildert habe, auszuschließen, wenn eben stets lege artis vorgegangen wird.

Unter den 156 Fällen sind Fälle von Abortus putridus schwerster Natur zu verzeichnen, die mit bestem Erfolge auch zweizeitig behandelt wurden, wenn eben die Zervix nicht fingerdurchgängig war.

Erwähnung möge ein Fall von Monstrosität des Uterus und der

1) Therapie bei Abortus. Stuttgart, Enke, 1901. 2) Gyn. Rundschau 1908.

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Scheide finden. Bei einer 28jährigen Erstgebärenden traten im II. Monate der Gravidität Blutungen ein. Bei der Untersuchung fand sich ein Uterus bicornis duplex cum vagina septa, wovon der linksgelegene Uterus gravid war. Der Befund glich vollkommen dem von Nagel in Veits Handbuch beschriebenen, in der Gebäranstalt der Charité beobachteten Falle. Erwähnt möge noch werden, daß ich die Mehrzahl der behandelten Fälle von Abortus am 3. und 5. Tage besuchte und am 8.—10. Tage zur Nachuntersuchung in meiner Ordination wieder sah. Bei einer gewissen Zahl von Fällen wurde so wegen konstatierter Retroversio oder Retroflexio uteri die Einlegung eines Fritsch-Hodge-Pessares nötig, das nach $^1/_4$—$^1/_2$ Jahre bei zweimonatlichem Wechseln wieder entfernt werden konnte. Gerade der nach Abortus verhältnismäßig oft eintretenden Uterusverlagerungen wegen halte ich eine kräftige Uterusmassage nebst reichlichen Gaben von Sekalepräparaten nach Entleerung des Uterus, ferner die Nachuntersuchung in der Rekonvaleszenz für dringend indiziert. Tägliche Scheidenspülungen mit einer Kal. jodat.-Jodtinkturlösung bis zum Wiedereintritt der Menstruation beeinflussen die Involution des Uterus nach Abortus auf das beste.

Ad III. Getreu dem an der Schule gelehrten Konservativismus in der Geburtshilfe habe ich Gelegenheit genommen, 14mal die künstliche Frühgeburt bei 12 Frauen einzuleiten. Bei 6 Frauen wurde wegen hochgradiger Beckenenge, allgemein verengtem Becken, rachitischem und kyphoskoliotischem Becken die Frühgeburt im VIII. Monat eingeleitet. All diese Frauen hatten schwere Wendungen mit totem Kinde und Perforation bereits überstanden. Bei 1 Frau, die bereits 2mal Perforation durchgemacht hatte, wurde innerhalb 3 Jahre die Frühgeburt im VIII. Monate 2mal eingeleitet. In 1 Falle wurde nach sicherem Nachweise des Abgestorbenseins der Frucht im VII. Monate ebenfalls die Frühgeburt eingeleitet. In allen Fällen habe ich die Frühgeburt mittels Einführung der Knappschen Bougie eingeleitet, wobei die spontane Entbindung in 24—36 Stunden erfolgte. Leider blieben die Kinder nicht am Leben.

Den Abort im I.—IV. Monat wurde eingeleitet in einem Falle von unstillbarem Erbrechen, wo jede Therapie erfolglos blieb, weiters bei einer Zwergin, deren Becken selbst für die Geburt eines Siebenmonatkindes zu klein war, bei einer an Osteomalakie leidenden Frau, bei einer an Morbus Brighti Leidenden und bei einer Frau, die wegen Graviditas extrauterina von mir einige Jahre vorher operiert worden war.

Den Abort im I.—IV. Monate leitete ich ausschließlich durch die intrauterine Tamponade ein. 24 Stunden nach dieser Tamponade

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wurde die instrumentelle Ausräumung des Uterus vorgenommen. Sämtliche Fälle hatten einen glatten Verlauf.

Ad IV. In 51 Fällen wurde der Forceps bei Schädellagen, in 2 Fällen bei Steißlagen angelegt.

Meinen Standpunkt in bezug auf die Indikationen der Zange habe ich bereits klargelegt; der günstige Verlauf aller meiner Fälle rechtfertigt denselben zur Genüge.

Es ist dabei aber auch eine ganz große Zahl von Fällen zu verzeichnen, wo der Forceps bei hochstehendem fixiertem Kopf mit oder ohne vollständige Entfaltung des unteren Uterinsegmentes zur Anwendung kam. Im letzteren Falle habe ich es in 2 Fällen erlebt, daß das untere Uterinsegment während 3—4tägigen Kreißens nicht zur Entfaltung kam, sondern als gerade fingerdurchgängiger Zapfen erhalten blieb. Durch zwei nach rechts und links hinten unten gerichtete Inzisionen wurde dasselbe gespalten und dann die Extraktion mittels Forceps vorgenommen. Nach Ablauf der III. Geburtsperiode wurden die Inzisionen mittels Knopfnähten wieder vereint; die Operation wurde in Narkose vorgenommen, während bei den anderen Entbindungen mittels Forceps die Narkose nicht in Anwendung kam. wie ich dies bereits in meiner diesbezüglichen Arbeit[1]) geschildert habe.

2mal wurde der Forceps zwecks Vornahme des Accouchement forcé zur Anwendung gebracht, in beiden Fällen wegen hochgradiger Eklampsie bei Erstgebärenden, wobei der Muttermund nicht vollständig verstrichen war. Im einen Falle war das Kind lebend geboren, im anderen Falle tot, nachdem die Entwicklung langsam vorgenommen werden mußte, um eben keine schweren Verletzungen der Mutter hervorzurufen. Beide Mütter machten eine glatte Rekonvaleszenz durch.

In 2 Fällen wurde die Kopfzange bei Steißlage zur Anwendung gebracht und zwar mit dem gleichguten Erfolge für Mutter und Kind.

Von Kindern habe ich 3 Todesfälle bei Forzeps zu verzeichnen, einen bei Eklampsie, die beiden anderen bei bestehendem großen Mißverhältnis zwischen Becken und Schädel.

Ad V. Die 3 Perforationen wurden mittels Auvardschen Instrumentes ausgeführt.

Der erste Fall betrifft eine 34jährige IIpara. Die ersten beiden Entbindungen erfolgten mittels Forceps; im Jahre 1899 führte ich die Ventrofixatio uteri bei Retroversio-flexio uteri fixata aus. 4 Jahre später wurde Pat. wieder gravid. Durch das Mißverhältnis zwischen kindlichem Kopf und mütterlichem Becken blieb der Kopf durch

1) Prager med. Wochenschr. 16. Jahrg.

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3 Tage beweglich über dem Beckeneingang. Am 3. Tage erst wurde ich zur Entbindung zugezogen; da war der Kopf bei verstrichenem Muttermund im Beckeneingang fixiert. Ein Zangenversuch mißlang, wobei es unter größter Kraftanstrengung nicht möglich war, den Kopf ins Becken zu ziehen; hierauf führte ich die Perforation aus. Das Kind war in der Tat abnorm stark. Die Ventrifixur des Uterus beeinflußte in keiner Weise den Geburtsverlauf, sondern lediglich das erwähnte Mißverhältnis. Glatter Verlauf mit Erhaltenbleiben der Normallage des Uterus.

Der 2. Fall betrifft eine 24jährige Frau, die 1 mal bereits im II. Monate abortiert hatte. Ich wurde zur Gebärenden gerufen, als die Geburt bereits 4 Tage im Gange war. Ich konnte eine floride Osteomalakie mit typischer Veränderung des Beckens konstatieren. Bei der überaus starken Entwicklung des Kindes und der langen Geburtsdauer kam nur die Perforation in Frage. Dieselbe wurde in Narkose ausgeführt. Trotz der Narkose war es bei vollständig verstrichenem Muttermund nicht möglich, den perforierten Kindesschädel durch das Becken hindurch zu bekommen. Nachdem konstatiert wurde, daß das Mißverhältnis zwischen Kindesschädel und Becken dieses Hindernis bildete, wurde das Auvardsche Instrument abgenommen und die Wendung des perforierten Kindes in der vorsichtigsten Weise vorgenommen. Dieselbe gelang und machte die Entwicklung des nachfolgenden Kindesschädels trotzdem noch große Schwierigkeit.

Ein Dammriß 2. Grades konnte dabei nicht verhindert werden. Trotz dieses großen Eingriffes verlief das Wochenbett fieberfrei.

Die puerperale Osteomalakie heilte nach 2 Jahren durch Phosphorbehandlung insoweit aus, daß bei weiter bestehender Deformität des Rumpfes und Beckens die großen Knochenschmerzen im Bereiche der Arme und Beine zum Schwinden gebracht wurden. Das Kind wog etwas über 5 kg.

Im 3. Falle handelte es sich um eine luetische 26jährige IIpara mit plattem rachitischem Becken. Die Geburt war bereits 4 Tage im Gange als ich zugezogen wurde. Muttermund bequem für 4 Finger durchgängig, Schädel des Kindes im Beckeneingang, nicht fixiert. Die Perforation in Narkose wurde leicht ausgeführt, ein linksseitiger Riß der Zervix, der bei der Extraktion zustande kam, wurde mittels 4 Knopfnähten geschlossen. Fieberloser Verlauf.

In bezug auf die Zuverlässigkeit des Auvardschen Instrumentes, das ich seit dessen Bekanntwerden benütze, kann ich nur berichten, daß dasselbe seinen Zweck stets vollkommen erfüllte, wenn auch die Applikation manchmal und insbesonders dann, wenn der Muttermund nicht vollkommen verstrichen ist, die allergrößte Vorsicht erfordert.

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Ad VI. Was die Ausführung der Wendung anbelangt, so muß ich, wie in meinen beiden vorhergegangenen Berichten, nur immer wieder darüber klagen, daß die Berufung des Arztes mehr minder spät erfolgte, wodurch die hohe Mortalität der Kinder ihre Begründung finden mag.

In 5 Fällen wurde aus Querlage mit oder ohne Nabelschnurvorfall gewendet und dabei 1 Kind lebend zur Welt gebracht.

In 2 Fällen wurde bei hohem Querstand, in 1 Falle bei Gesichtslage gewendet und ebenfalls nur 1 Kind lebend zur Welt gebracht.

In 3 Fällen wurde die Wendung bei Querlage mit Armvorfall ausgeführt, wobei alle 3 Kinder tot zur Welt gebracht wurden.

Sämtliche Fälle hatten ein fieberfreies Wochenbett durchgemacht.

Ad VII. Zwillingsgeburten gestalteten sich folgendermaßen:

1. 28jährige III para. Spontane Entbindung, Knabe, Mädchen. 2 medial adhärente Plazenten.

2. 25jährige I para. Hydramnios, Blasensprengung. 1. Knabe Forceps, 2. Knabe spontan. 1 Plazenta.

3. 34jährige IV para. Doppelte Fußlage, Nabelschnurvorfall, Extraktion, totes Mädchen, Wendung nach Blasensprengung, Extraktion, lebender Knabe. 2 getrennte Plazenten.

4. 32jährige I para. Protrahierter Geburtsverlauf, Forceps, lebender Knabe, Fußlage, Extraktion, lebender Knabe. 1 Plazenta.

5. 25jährige I para. Kam $^1/_2$ Jahr vor der Schwangerschaft wegen Retroversio uteri mobilis in Behandlung. Fritsch-Hodge-Pessar Nr. IV. 6 Monate trägt die Frau das Pessar; als sie wieder zum Wechsel des Ringes kommt, ist sie einen Monat schwanger. Im 3. Schwangerschaftsmonate wird das Pessar entfernt. Am normalen Ende der Schwangerschaft spontane Geburt von 2 Mädchen. 1 Plazenta.

6. 32jährige II para. 1. Schwangerschaft Abortus m. II. Im 7. Monat der bestehenden Schwangerschaft plötzlicher Wasserabfluß, ohne jedwede Wehentätigkeit. Am 2. Tage Forceps, lebendes Mädchen, Wendung, lebendes Mädchen. 1 Plazenta.

Sämtliche Fälle hatten glatten Wochenbettsverlauf.

Ad VIII—XII. ist nichts Wesentliches zu bemerken.

Ad XIII. Von 15 bei 14 Frauen beobachteten Extrauteringraviditäten habe ich in 9 Fällen durch Laparatomie die schwangere Tube entfernt. Darunter ist eine Frau innerhalb 2 Jahre 2mal laparatomiert worden, das erste Mal wegen linksseitiger, das zweite Mal wegen rechtsseitiger Tubargravidität. In 4 Fällen führte die expektative Behandlung bei eingetretener Hämatozelenbildung zur Spontanausheilung. Ein Fall Graviditas tubaria m. III. starb während der Vorbereitung zur Operation. An anderer Stelle wird eingehend über diese Fälle gesprochen werden.

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Ad XIV. Die puerperalen Infektionen, die nach spontanen Geburten und Abortus zur Beobachtung kamen, kamen mit Ausnahme eines Falles unter Leitung von Hebammen zustande.

In 3 Fällen wurde Puerperalfieber als Allgemeininfektion ohne Nachweis lokaler Erscheinungen beobachtet, in 2 Fällen Phlegmasia alba dolens mit parametranen Exsudaten, in 1 Falle Thrombose der rechten Vena femoralis mit rechtsseitigem parametranen Exsudat und in 5 Fällen mehr minder ausgebreitete Pelveoperitonitis.

Die letzteren Fälle hatten einen sehr langsamen Verlauf in bezug auf Resorption der Exsudate. Im akuten Stadium wurde bei allen Fällen Unguentum Credé in ausgiebiger Weise zur Anwendung gebracht und hat es den Anschein, daß diese Therapie, vorschriftsmäßig ausgeführt, auf die Herabsetzung der Temperaturen einen günstigen Einfluß auszuüben vermag. Die Temperaturen fielen nach der Einreibung konstant, was um so auffälliger war, als Antipyretika nicht verabreicht wurden.

Teplitzer Badekuren hatten eine vorzügliche Wirkung auf die Resorption der Exsudate und erlebte ich, daß eine Frau, die nach Zwillingen an einem bis zur Nabelhöhe reichenden linksseitigen Beckenexsudat 2 Jahre nach dieser Erkrankung wieder konzipierte, gebar und ein fieberfreies Wochenbett durchmachte. Teplitzer Thermal- und Thermal-Moorbäder halte ich für ganz vorzügliche Unterstützungsmittel zwecks Resorption von Beckenexsudaten.

Sämtliche 11 Frauen kamen zwar mit dem Leben davon, allein einige von ihnen stehen heute noch in Behandlung.

Das Puerperalfieber, jenes traurige Kapitel in der geburtshilflichen Praxis, richtet derartige Verheerungen im weiblichen Organismus an und führt zu solch schweren Schädigungen desselben, daß es nichts weniger als dringend erscheint, dieses Kapitel zur öffentlichen Diskussion zu stellen, wie dies 1909 durch die Deutsche Gynäkologische Gesellschaft in Straßburg geschehen wird. Es wird sich sehr empfehlen, daß die geburtshilflichen Praktiker sich rege an dieser Diskussion beteiligen, um den Klinikern Material und Vorschläge für eine endliche Bekämpfung desselben auch in der geburtshilflichen Praxis zu liefern.

Ad XV. Eine verhältnismäßig große Zahl von Rückwärtslagerung des schwangeren Uterus gibt Beweis dafür, wie häufig dieselbe vorkommt, insbesonders bei Arbeiterinnen. Zum weitaus größten Teile bildet diese Lageveränderung neben großen Allgemeinbeschwerden die Ursache zum Abortus.

Ich kann in bezug darauf auf die Arbeit R. Chrobaks[1]) verweisen.

1) Volkmannsche Sammlung 377, 1904.

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Bei einer kleinen Zahl der Fälle gelang es durch Pessartherapie den Abortus zu vermeiden. Bestand Blutung und währten die Beschwerden schon längere Zeit, so war auch durch diese Behandlung der Abortus nicht mehr aufzuhalten. In einem Falle war die Retroflexio uteri gravidi 2mal bereits die Ursache des Abortus; es wurde nach erfolgtem Abortus von dem betreffenden Arzte eben nicht nachuntersucht. In einem Falle bei Retroflexio uteri gravidi m. IV. incarcerati, bei einer hochfiebernden, ganz heruntergekommenen Frau mit den hochgradigsten Inkarzerationsbeschwerden wurden durch Wochen von verschiedenster Seite die verschiedensten Medikationen ohne richtige Diagnosenstellung erfolglos angewandt. Es konnte kein Zweifel bestehen, daß auch intrauterin planlos eingegriffen worden war, denn die Frau war eben bereits septisch und starb bei der Aufnahme ins Hospital. Es handelte sich um eine purulente Peritonitis, ausgegangen von dem septischen Uterusinhalt.

In 3 Fällen nur kam es durch die Pessartherapie zum Fortbestehen der Schwangerschaft bis zum normalen Ende; es waren dies Fälle, wo nach Eintritt der Schwangerschaft plötzlich heftige Kreuzschmerzen mit Blasenbeschwerden eingetreten waren, ohne daß eine Blutung auf eine Unterbrechung der Schwangerschaft hatte schließen lassen. Jedesmal war das Heben einer schweren Last die Ursache der Lageveränderung des Uterus.

In 4 anderen Fällen wurde in typischer Weise die Ausräumung des Uterus vorgenommen und gleichzeitig nach Reposition des Uterus ein Pessar eingeführt. Reichliche Ergotingaben nebst Jodjodkalispülungen führten eine kräftige Involution des normal gelagerten Uterus herbei.

Ad XVI. Dieser Fall, in dem es sich um eine Spontanruptur des Uterus am normalen Schwangerschaftsende nach vorheriger Sectio caesarea mit Fritschschem Fundalschnitt handelt, ist ausführlich im Zentralblatt für Gynäkologie 1904 beschrieben worden.

Ad XVI und XVIII ist nichts Bemerkenswertes zu berichten.

Am Ende meines Berichtes angelangt, kann ich nach drei Quinquennien praktisch geburtshilflicher Tätigkeit nur wärmstens empfehlen, den Konservativismus im Sinne von v. Herff in der geburtshilflichen Praxis strengstens zu wahren und zu pflegen. Dabei ist es natürlich unerläßlich, den Status auto-noninfectionis strenge im Auge zu behalten und unaufhörlich auf dem Gebiete der Sozialhygiene das Unmögliche zu verlangen um das Mögliche zu erreichen.

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